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Independent learning program for GPs

Units 490/491 January/February 2013

Depression

The Royal Australian College of General Practitioners

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The five domains of general practice
Communication skills and the patient-doctor relationship
Applied professional knowledge and skills
Population health and the context of general practice
Professional and ethical role
Organisational and legal dimensions

The Royal Australian College of General Practitioners

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Depression is a common condition and the GP is ideally placed to screen for depression as well as diagnose, support and treat patients who present with it. This develops from their relationship of mutual trust, and the GP's knowledge of their patients as individuals and as people in their broader sociocultural context. However, it is important, where appropriate, to involve other health professionals such as psychologists and psychiatrists, who can provide valuable input in a range of situations.

Many of us could reflect back and identify patients who have not responded as intended to treatments we have recommended or support we feel we have given. These patients, in particular, highlight the importance of active listening to patient concerns, understanding their unique context, reconsidering the diagnosis, identifying and managing comorbidities, attending to perpetuating factors and responding to reasons for non-adherence.

This issue of *check* looks at clinical scenarios in relation to diagnosis and management of depression at various stages of the life cycle and aims to explore making a diagnosis of depression, as well as some of the issues involved where a patient does not improve with treatment.

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The learning objectives of this unit are to:

- understand the differential diagnosis in patients who present with symptoms suggestive of depression
- display increased confidence in assessment of risk in patients who present with depression
- display an awareness of the importance of considering a diagnosis of bipolar disorder in patients who present with depression
- obtain a comprehensive history in patients who present with symptoms suggestive of depression and identify comorbidities such as anxiety, substance misuse or features of a possible personality disorder
- understand the role of psycho-education, psychological therapies, antidepressant medication and some common complementary therapies in the management of depression and individualise treatment to the patient involved
- identify reasons for persistence of depressive symptoms despite treatment
- assess when it is appropriate to cease antidepressant medication in patients treated for depression, and manage the process of ceasing antidepressants with confidence.

We hope you enjoy reading the interesting hypothetical scenarios of people who present with depression and that you gain some practical tips to help manage your patients.

Kind regards,

Catherine Dodgshun MBBS, DRANZCOG, FRACGP

Medical Editor, *check* Program

GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

AMI	acute myocardial infarction	IPT	interpersonal therapy	PTSD	post-traumatic stress disorder
CBT	cognitive behavioural therapy	MDD	major depressive disorder	SNRI	serotonin and noradrenalin reuptake inhibitor
DSM IV-TR™	Diagnostic and Statistical Manual of Mental Disorders (fourth edition – text revision)	MDE	major depressive episode	SSRI	selective serotonin reuptake inhibitor
		NSAID	non-steroidal anti-inflammatory drug		
GAD	generalised anxiety disorder	OCD	obsessive compulsive disorder		
GPMHTP	GP mental health treatment plan	PND	postnatal depression		

CASE 1

FRANK HAS SUICIDAL THOUGHTS

Frank, aged 48 years, owns an accounting business. You have known Frank for many years. Over that time, you have found some of his behaviour challenging. He has frequently been irritable if you were running a few minutes late, reminding you of how important he is and that he has little time. He has often told long-winded stories of business successes and appeared to enjoy exploiting competitors. You have gathered from his wife that he has always been a workaholic and has few friends or interests outside work. She also indicated that Frank has always been very sensitive to perceived criticism or insufficient admiration.

Frank consults you for 'something to help me sleep.' You ascertain that his mood has been morose since he lost a large client a month ago and his wife subsequently left him. Initially, he was humiliated and angry because he felt he had worked hard to give her everything she could want and she was ungrateful and 'cold-hearted.' Now he wonders if he was ever good enough for his wife and he thinks she had wanted to leave him for a long time.

For the past few weeks Frank has had difficulty concentrating at work and his business has suffered. He no longer enjoys his work or watching television, has no appetite or energy and has difficulty getting to sleep. Four days ago, Frank received threatening letters from his wife's lawyers. He was indignant and, after half a bottle of whisky, left messages on his wife's telephone complaining that she never appreciated him. He hasn't gone to work since. He feels there is no way out for him and has been thinking about ending his life.

Frank has no past medical illnesses and takes no regular medications. He has no family history of psychiatric illness.

QUESTION 1

What is your working diagnosis? What other diagnoses would you consider in Frank?

QUESTION 2

How would you assess Frank's risk of completing suicide?

FURTHER INFORMATION

With Frank's consent, you obtain further history from his wife, who confirms that Frank's irritable mood and grandiosity is longstanding and stable. You ask Frank about his alcohol use, and he says he drinks two bottles of wine every night to help him get to sleep, and has been doing so for the past month. Frank does not have any symptoms that suggest an underlying physical illness. You perform a physical examination and request investigations.

Frank acknowledges that he doesn't really want to end his life, but feels overwhelmed by hopelessness at times, especially when he is drinking. You are confident that he has no specific suicide plan, but you are concerned that there are limited protective factors. You are also concerned that Frank feels worse when drinking, but has no intention of stopping because it's the only way he can get to sleep.

After discussion, Frank agrees with your diagnosis of depression and wants help, but worries that he will never feel better and his future is doomed. He identifies his brother as someone who he feels close to and could call when overwhelmed.

QUESTION 3

What is your initial management plan?

FURTHER INFORMATION

You review Frank 3 weeks after commencing a selective serotonin reuptake inhibitor (SSRI) and referring him to the local mental health crisis team, who saw him for a week. He found them helpful. Frank says the crisis team recommended he continue treatment with you, but you have received no direct correspondence from them. Frank's mood and sleep have both improved and he is no longer suicidal. However, he remains upset and angry about his financial troubles.

QUESTION 4

What additional management issues should you consider?

CASE 1 ANSWERS

ANSWER 1

Frank describes over 2 weeks of a pervasively low mood, anhedonia, reduced concentration, energy and appetite, insomnia and, more recently, suicidal ideation. These symptoms are consistent with a major depressive episode (MDE).[1] The Diagnostic and Statistical Manual of Mental Disorders fourth edition – text revision (DSM IV-TR™) criteria for a major depressive episode (MDE) are listed on page 5. MDD requires the presence of at least one MDE and is the most likely diagnosis in Frank.

It is important to confirm the diagnosis of MDD and exclude other diagnoses. If Frank consents, collateral history from his wife could assist in confirming the diagnosis of MDD and excluding other diagnoses.

Other diagnoses to consider in Frank include:

- a bipolar disorder (type 1 or 2). Always consider this diagnosis and ascertain if there is any history of manic, mixed affective or hypomanic episodes, which would indicate a bipolar disorder. Distinguishing between unipolar and bipolar depression has important treatment implications (see *Case 4*). While Frank displays some features that can be part of a hypomanic or mixed affective episode such as an irritable mood, grandiosity, a sense of entitlement and a need for admiration, these features appear to be longstanding and stable. Therefore, they are more likely to be part of his personality.
- adjustment disorder with depressed mood. An adjustment disorder is characterised by the development of clinically significant emotional or behavioural symptoms in response to an identifiable stressor. Frank's cluster of symptoms is more consistent with an MDE.
- narcissistic personality disorder. Frank has characteristics such as grandiosity, a sense of entitlement and a need for admiration, which may be considered narcissistic personality traits or part of a hyperthymic temperament. Further longitudinal assessment and careful history are required to determine if these symptoms are of sufficient clinical concern to merit an additional personality disorder diagnosis (see *Page 5*).[1]
- an anxiety disorder. An anxiety disorder such as generalised anxiety disorder (GAD) may present with symptoms that can be confused with irritability, and may co-exist with MDD. Further history regarding excessive worry about a variety of situations should be sought from Frank.
- substance-induced depression, or substance misuse comorbid with depression. Alcohol abuse, dependence and withdrawal are all associated with dysphoria and depressive symptoms. Further history regarding the amount and pattern of alcohol intake and symptoms of dependence or withdrawal is required.
- major depression due to a general medical condition or the effects of medication. Further history, physical examination and investigations can exclude physical illness or medications (*Table 1*)[2] that may cause or exacerbate depressive symptoms. *Table 2* lists screening investigations to consider in order to help exclude physical illness.

Criteria for major depressive episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either 1. depressed mood or 2. loss of interest or pleasure

Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations

1. depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g. feels sad or empty) or observation made by others (e.g. appears tearful). Note: In children and adolescents, can be irritable mood
2. markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)
3. significant weight loss when not dieting or weight gain (e.g. a change of more than 5% of body weight in a month), or a decrease or increase in appetite nearly every day. Note: In children, consider failure to make expected weight gains
4. insomnia or hypersomnia nearly every day
5. psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)
6. fatigue or loss of energy nearly every day
7. feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)
8. diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)
9. recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

B. The symptoms do not meet the criteria for a Mixed Episode*

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning

D. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, or a medication) or a general medical condition (e.g. hypothyroidism)

E. The symptoms are not better accounted for by bereavement, i.e. after the loss of a loved one, the symptoms persist for longer than 2 months or are characterised by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation

*See page 365 of the publication below

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Most **Axis 1 disorders** are syndromes of commonly co-occurring symptoms and include psychiatric conditions such as MDD, bipolar disorders and adjustment disorders as well as mental conditions due to a general medical condition and substance-related disorders.[1] Most of these disorders have symptoms that are associated with significant distress or impairment in functioning. Although these conditions may be chronic, they are thought to have a natural history that does not encompass most of adult life. Treatments to which they may respond include medication and/or short-term psychological interventions, depending on the disorder involved.

A **personality disorder** is diagnosed on the basis of symptoms in at least two of the following: thought, emotion, interpersonal functioning or impulse control, where the symptoms are not due to another psychiatric illness, a general medical condition or a substance. Examples include borderline personality disorder, narcissistic personality disorder and avoidant personality disorder.[1] Symptoms of a personality disorder consistently cause significant distress or impaired functioning in multiple aspects of an individual's life. Symptoms of a personality disorder are present for most of adult life and are pervasive and stable over time. Unlike Axis 1 disorders, in general, personality disorders do not respond to medication and longer-term psychological therapy is the treatment of choice.

While **personality traits** are characteristic patterns of thought, emotion, interpersonal functioning and/or impulse control and they are also usually stable over time, there are some key differences that separate them from personality disorders. Personality traits are more flexible than patterns in a personality disorder and do not consistently cause significant distress or impairment in functioning.[1]

ANSWER 2

A recent long-term cohort study suggests approximately 7% of people with MDD commit suicide, but some groups are at higher long-term risk, particularly males with severe depression.[3] Demographic, social, mental and physical health factors may influence both acute and chronic suicide risks (*Table 3*).[4, 5]

Enquire about suicidal thoughts. Be tactful but direct. Frank gives a history of suicidal thoughts. Enquire about the frequency and persistence of these suicidal thoughts, any specific suicide plan, the means to carry out the plan and any final acts undertaken (such as settling affairs, making a will or writing letters). Take particularly seriously any plans involving pain or lethal means (such as firearms or hanging). Ask about past suicidal attempts and the seriousness of these attempts. Also enquire about protective factors including intellectual functioning, internal coping resources, value systems and available social supports. Ascertain if there is a family history of suicide.

Various clinical features can help differentiate between clinical disorders referred to as 'Axis 1 disorders', personality disorders and personality traits in DSM IV-TR™.

ANSWER 3

Immediate management consists of determining the most appropriate setting for treatment depending on the severity of the MDE, risk of suicide and social supports available.

Frank has a moderately increased risk of suicide, but is willing to accept treatment. Community management is likely to be appropriate, but short-term support, such as that provided by an outreach or crisis team from a local mental health service, is advisable.

Crisis team support allows close monitoring of Frank's mental state and suicide risk as treatment is being implemented, the effectiveness of that treatment and any adverse effects that might emerge. The process of referral to these teams varies according to the Australian state or territory.

Short-term management consists of:

- psycho-education
- treating the presenting symptoms
- determining the plan for future reviews
- making appropriate referrals.

Table 1. Physical conditions and medications or substances that can cause depressive symptoms

Physical condition	Medication or substance
• Malignancy • Hypothyroidism • Congestive cardiac failure • Cerebrovascular disease and stroke • Other intracerebral lesion • Delirium • Diabetes • Anaemia • Post-infective states	• Beta-blockers • Corticosteroids • Benzodiazepines • Alcohol

Adapted with permission from Murtagh JE. General practice. 5th edn. Sydney: McGraw Hill, 2011.

Table 2. Screening investigations to consider to exclude 'organic' causes of depression

- Full blood examination
- Urea, electrolytes and creatinine
- Liver function tests
- Thyroid function tests
- Vitamin D level
- Fasting blood glucose level
- Inflammatory markers
- Urine toxicology
- Cerebral imaging

Adapted from: Curan EM, Loi S. Depression and dementia. MJA Open 1 October, 2012:1Suppl4:40-43.

Table 3. Risk factors for suicide

Social/demographic

- Male
- Increasing age[4]
- Widowed or divorced[4] (especially if recent or if near to anniversary)
- Other recent losses[4]
- Social isolation[5]
- Unemployment[4,5]
- Immigrant
- University student
- Doctor

Psychiatric (most important single cause)

- Depression[4,5]
- Anxiety disorders (especially if comorbid depression)
- Personality disorders
- Schizophrenia
- Alcohol dependence[5]
- Substance abuse[5]
- Prior suicide attempts or deliberate self-harm
- Family history of suicide

Medical

- Chronic medical conditions[4]
- Chronic pain
- Incapacity
- Post-injury

Mental state

- Current acute suicidal thoughts
- Plan, especially if involving pain or lethal means (such as firearms or hanging)
- Acquired means
- Completed final acts (such as making a will)
- Prominent hopelessness
- Agitation
- Tormenting psychotic symptoms (such as command hallucinations)

Goldney R. Suicide prevention. New York: Oxford University Press, 2008.[4] Knox KL, Conwell Y, Caine ED. If suicide is a public health problem, what are we doing to prevent it? American Journal of Public Health, 2004;94(1):37–45.[5]

Antidepressant medication is indicated as Frank's symptoms are of moderate severity. Discuss the different treatments available and the likely side effects of each with Frank and reach a decision together. An SSRI would be appropriate for Frank. Possible side effects, including an increase in anxiety and agitation over the first few days of treatment and sexual side effects, should be discussed. Despite the absence of past manic or hypomanic symptoms, the possibility of an occult bipolar illness remains. The crisis team and Frank himself should be warned about the risk of antidepressant medication provoking a manic 'switch' in his mood (see *Case 4*). Check baseline sodium level because SSRIs may cause or worsen hyponatraemia.

Psycho-education regarding depression, antidepressant medication treatment and the roles of stress and alcohol is imperative to optimise treatment outcome in Frank. Issues to be addressed include the possible effect of depression on his work performance, the importance of adequate sleep and behaviours that form part of sleep hygiene as well as the importance of a balanced diet and regular exercise and the perpetuating effect of alcohol on depressive symptoms and its possible interaction with antidepressant medication. It is also important to discuss the mechanism of action of antidepressant medication and the consequent need to take the tablets every day rather than on an as-required basis and reassure Frank regarding possible 'mild' side effects. Encourage Frank to identify and respond appropriately to mild and more serious side effects. Literature consistently confirms that these interventions, supporting the taking of antidepressant medication, do improve adherence.[6]

In general, the specific techniques used to address alcohol use depend on the patient's motivation to change, the severity of their alcohol use and the associated risks. Inform Frank of the links between alcohol use, depressive symptoms and suicidality. The ongoing use of alcohol may reduce the effect seen from the antidepressant medication, and may also substantially increase the risk of completed suicide.[7] Be aware that depressive symptoms may last for many months following cessation of alcohol consumption.

ANSWER 4

As psychosocial stressors, and Frank's reactions to them, have a central role in his problems, multimodal treatment is optimal. Consider referral for formal psychological therapies. Refer to *Table 4* for a list of some of the Medicare item numbers available for mental health services provided by GPs, clinical psychologists and psychiatrists.[8] See *Table 5* for the steps involved in the preparation of a GP mental health treatment plan (GPMHTP).[8]

It is important to note that suicidality is not a contraindication to psychological therapy and referral should not be delayed until partial recovery. Cognitive behavioural therapy (CBT), interpersonal therapy (IPT) and psychodynamic psychotherapy all have evidence for effectiveness. It is important to note that the benefit of psychological therapy and medication combined may be greater than that from either on their own.[9]

Antidepressant medication may be used long term depending upon the severity of the symptoms experienced, the duration of the episode and the number of previous episodes of depression.

Other factors to consider, and assist Frank with (where possible), include:

- personality and interpersonal relationship problems
- social isolation and accessing available informal supports
- stigma
- the absence of meaningful roles or purposes in life
- employment problems and risks to reputation (medical certificates and return to work plans may help)
- financial problems such as unpaid bills
- accommodation problems
- legal problems.

Table 4. Medicare eligible mental health services by item number and provider

Medicare items available	Purpose
Items 2700, 2701, 2715, 2717 Provided by GP	To establish a GPMHTP (specific item number linked to the amount of time spent with the patient and to the presence or absence of mental health training by the GP). Usually maximum frequency of annual
Item 2712. Provided by GP	To review a GPMHTP or psychiatrist assessment and management plan. Recommended frequency is an initial review between 4 weeks and 6 months after the plan was created, and further review at least 3 months after the first review
Item 2713. Provided by GP	GP extended consultation with the patient where the primary treating problem is related to a mental disorder
Items 2721, 2723, 2725, 2727 Provided by GP	GP provision of focussed psychological strategies derived from evidence-based psychological therapies (where the GP has satisfied associated training requirements)
Items 80000, 80020; provided by clinical psychologist Items 80100 to 80170; provided by occupational therapist or social worker	Psychological therapy services and focused psychological services provided by an allied mental health worker where specific referral requirements are met. Individual therapy sessions (up to ten per calendar year with an additional six sessions in exceptional circumstances) and up to ten group therapy services per calendar year
Items 291, 293 Provided by private psychiatrist	Private psychiatry referral for assessment and establishment of mental health management plan to be implemented and monitored by GP (with annual review)

Adapted from: Australian Government, Department of Health and Ageing. Medicare Benefits Schedule. 2012. Available at www.mbsonline.gov.au [accessed 5 December 2012]

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Table 5. Steps to access Medicare-eligible psychological services

Assessment
• Obtain and record the patient's consent to produce a GPMHTP
• Obtain a history, including presenting complaint, relevant past and present biological, psychological and social factors and comorbidities
• Conduct a mental state examination
• Conduct a risk assessment
• Make a diagnosis and/or formulation
• Administer an outcome measurement tool, if clinically appropriate
Plan
• Discuss the assessment and findings with the patient
• Discuss alternative treatment and referral options with the patient
• Establish collaborative treatment goals
• Establish actions or strategies that the patient will take responsibility for undertaking
• Provide psycho-education
• Develop a plan for crisis intervention and/or relapse prevention
• Make arrangements for required referral, treatment, appropriate support services, review and follow-up
• Ensure documentation of the above and establish a date for formal review
Adapted from Australian Government Department of Health and Ageing. Medicare Benefits Schedule. 2007. Available at www.mbsonline.gov.au [accessed 5 December 2012].
Used with permission of the Australian Government.

CASE 2

MATTHEW ISN'T HIS 'USUAL HAPPY SELF'

Matthew, aged 14 years, attends your practice with his mother Jan. You have not seen Matthew for several years. He is in year 9 at the local high school, and lives at home with his parents and sister, aged 9 years. You remember that Jan's pregnancy with Matthew and his delivery were uncomplicated, and that Jan and Matthew had bonded well. Your records show that Matthew reached all developmental milestones at the expected age. As a child, Matthew experienced mild exercise-induced asthma, but this has now largely resolved. He has attended hospital on several occasions with minor injuries, but has not required surgery or a prolonged hospital stay.

You see Jan and Matthew together, at Matthew's insistence. He tells you that 'Mum's the one that's worried, not me'. Jan agrees that she and her husband are concerned that Matthew has 'changed over the last few months' and that he 'isn't his usual happy self'. She is concerned that Matthew's teachers have also noticed a significant deterioration in his attitude and school performance over the previous 3 months. Jan is concerned that Matthew does not have much energy, despite 'sleeping all the time'. Jan is not aware of any recent conflict or losses.

During your consultation, Matthew appears sullen and makes little eye contact. After some time, Matthew agrees to see you alone. He reluctantly tells you that he often feels like crying and feels angry. He is embarrassed and concerned about his school marks, as he is struggling to concentrate. Matthew has a small group of close friends, but increasingly prefers playing online games alone as he 'can't really be bothered'. He has tried alcohol on one occasion earlier this year, but did not enjoy the taste or 'the way it made everyone act'. He has not tried cigarettes or other drugs and says that he is not interested in doing so. On specific questioning, Matthew says he has no thoughts about harming himself or wanting to die.

QUESTION 1

What is your working diagnosis? What is your differential diagnosis?

FURTHER INFORMATION

You explain the likely diagnosis to Matthew. He is happy for his mother to be involved. You invite Jan back into the room and explain the diagnosis. Jan asks you about treatment options.

QUESTION 2

What advice can you give Matthew and Jan about treatment options?

QUESTION 3

When would you consider referral to a specialist child and adolescent health service?

QUESTION 4

What clinical features might be present if Matthew were being bullied?

CASE 2 ANSWERS

ANSWER 1

Matthew has a history lasting several months of low mood; reduced concentration, motivation and energy; social withdrawal; and hypersomnia. These symptoms are consistent with an MDE. The most likely diagnosis is MDD.

The differential diagnoses that should be considered when adolescents present with depressive symptoms include:

- MDD
- dysthymic disorder. This diagnosis should be considered if the young person has experienced low or irritable mood for most days over at least 1 year. Depression can sometimes complicate dysthymic disorder, but the latter is usually well established first[1]
- an anxiety disorder. GAD and social phobia may present with similar symptoms, causing significant withdrawal and functional impairment. They often co-exist with depression and anxiety can be confused with irritability. However, both GAD and social phobia are usually more chronic than depression. GAD is characterised by excessive anxiety or worry occurring in a variety of settings.[1] Social phobia is a marked fear of scrutiny in social situations[1]
- a bipolar disorder. This diagnosis should always be considered. Seek any history of sustained periods of elated or irritable mood as there are important treatment implications. Depression may be the first presentation of bipolar disorder and there is no way to reliably differentiate these diagnoses at cross-sectional interview. Review and monitoring are essential
- prodromal symptoms of a psychosis. While psychotic illnesses are less common than mood or anxiety disorders, it is imperative to exclude them where depression is suspected. This is because mood and anxiety symptoms, in association with functional decline, may indicate the early 'prodromal' phases of a psychotic illness. Again, ongoing review is paramount.

Other diagnoses to consider include:

- substance misuse. Ask about alcohol and drug use. Substance misuse is commonly comorbid with depression in young people
- a physical condition (*Table 1*). This may contribute to depressive symptoms or impact upon mood
- physical and/or sexual abuse, especially if there is a history of presenting to hospital on multiple occasions with minor injuries. Mandatory reporting requirements of some description apply to doctors in all states and territories of Australia.

ANSWER 2

Treatment for depression in adolescents differs from that in adults. Antidepressants are only recommended in severe depression or where other interventions have been unsuccessful.[10]

A comprehensive management plan includes:

- employing an empathic approach
- psycho-education, including utilising written and internet resources (see *Resources*)
- utilising problem-solving techniques
- advising about lifestyle factors such as diet, exercise and sleep hygiene
- regular monitoring of mental state and risks
- obtaining collateral history
- assessing for and managing parental depression or anxiety, inconsistent parenting, marital discord
- liaising with Matthew's school with his consent
- monitoring Matthew's 9-year-old sister for emotional and behavioural disturbances
- referring to a psychologist or psychiatrist for a structured psychological intervention (see *Tables 4* and *5*). The current best evidence is for CBT and IPT.[10]

ANSWER 3

Referral should be considered where:

- depression is moderate to severe in intensity
- depression is complicated (for example, there is a suicide risk or concurrent substance abuse), or there is diagnostic uncertainty
- prior to commencing antidepressant medication
- a coordinated approach is required involving multiple clinicians and components of the management plan.

ANSWER 4

Bullying is increasingly common in young people of school age and may significantly affect emotional, social and academic functioning.[11] Forms of bullying are diverse, including physical and verbal abuse, and newer forms through the internet and social media. As many young people will not seek help directly, routinely consider bullying for any young person presenting with distress. Refer to *Table 6* for a list of clinical indicators of bullying.[11]

Table 6. Clinical indicators of bullying

- School refusal or refusal to discuss school day
- Being tense, tearful or unhappy before or after school
- Changes to routines around school or wanting to be driven to school
- New talk about hating school or other children
- Physical injuries, including minor ones
- Unexpected/unexplained damage to, or loss of, belongings
- Insomnia, nightmares, secondary enuresis
- Social withdrawal
- Somatic symptoms, especially if used as excuse to avoid school

Adapted from: Carr-Gregg M, Manocha R. Bullying – effects, prevalence and strategies for detection. Aust Fam Physician, 2011;40(3):98–102.

CASE 3

ANGELA HAS CHRONIC BACK PAIN AND DEPRESSION

Angela, aged 44 years, is a divorced childcare assistant with three children who recently commenced attending your practice. She presents requesting treatment for depression and chronic back pain for the first time. She says she has a past history of chronic depression, and has taken several different antidepressants without full symptom resolution.

Angela has been using over-the-counter medications to manage her back pain and describes using 20 ibuprofen 200 mg/codeine phosphate 12.8 mg tablets almost every day for the previous 6 months; she believes these tablets also help her mood.

Angela describes a prolonged history of low mood on most days, longstanding thoughts of deliberate self-harm or suicide, mood swings and periods of intense anxiety. She says she has never attempted suicide and her children have prevented her from acting on her suicidal thoughts in the past. She is not suicidal at present. She is able to work and values her job. She identifies her mother as a support.

Angela is agreeable to ceasing use of ibuprofen/codeine phosphate, but wants you to prescribe something else for the pain, in addition to something to help her mood.

QUESTION 1

What is your differential diagnosis?

QUESTION 2

What further information do you need in order to assess Angela's chronic back pain and depression?

FURTHER INFORMATION

Angela described an incident of injury to her back occurring while lifting 8 years ago with chronic pain ever since. Her pain is the focus of her current thoughts. You determine that Angela has symptoms consistent with a chronic pain disorder, MDD and substance misuse. She says that her mood worsens in association with her back pain.

QUESTION 3

How would you approach assessment of her misuse of ibuprofen/codeine phosphate?

QUESTION 4

What would management of Angela's issues in the longer term involve?

CASE 3 ANSWERS

ANSWER 1

The differential diagnosis includes:

- a mood disorder such as MDD, a bipolar disorder or dysthymic disorder. Angela has features of MDD including low mood and suicidal thoughts. She also describes 'mood swings'. Further history is required to determine their significance
- an anxiety disorder. Angela describes periods of intense anxiety. Anxiety may co-exist with depression
- borderline personality disorder. Caution and longitudinal assessment are urged before any diagnosis of personality disorder is made as symptoms of an Axis I disorder such as MDD may influence reporting of pre-morbid functioning and interpretation of personality features
- a pain disorder associated with psychological factors and/or a general medical condition. Angela describes chronic pain. If her pain is the predominant focus of her clinical presentation, if it is associated with significant distress or impairment in functioning, and if psychological factors play a major role then she may have a chronic pain disorder associated with psychological factors
- somatisation disorder. If Angela had pain in multiple sites or systems and it was associated with gastrointestinal, sexual or pseudo-neurological symptoms, she could have somatisation disorder
- substance-induced depression or substance misuse comorbid with depression.

ANSWER 2

The following information should be obtained:

- current symptoms such as sleep, appetite and concentration disturbance that suggest an MDE
- information in order to assess risk, which should form part of any mental health assessment. Ask about thoughts of deliberate self-harm or suicide (differentiate chronic suicidality from acute increases in intensity), previous suicide attempts and their lethality, previous episodes of deliberate self-harm, family history of suicide and risks to others, especially Angela's children
- details of her past psychiatric history such as previous episodes of depression, including whether there were any psychotic or manic symptoms, precipitants of these episodes and previous antidepressant treatment, including duration, response and side effects
- personality vulnerabilities, including feelings of abandonment, emptiness, isolation, affective instability and poor frustration tolerance that suggest borderline personality disorder. It is associated with increased tendency to depressive symptoms and MDEs
- details of Angela's back condition and pain, given the close relationship between chronic pain and depression. Ask about her past history of injury, results of investigations and the current pattern of pain, its precipitants and previous treatments including both medications and non-pharmacological treatment, previous referrals to health practitioners and perpetuating factors such as psychosocial stressors
- current substance use including current use of ibuprofen/codeine phosphate and over-the-counter medications, alcohol, illicit drugs, reasons for use and motivation to cease ibuprofen/codeine phosphate, physical complications of non-steroidal anti-inflammatory drug (NSAID) use, such as gastritis and renal dysfunction, and side effects of opiates, such as constipation and fatigue
- past history of substance abuse or dependence
- past medical and surgical history
- family history of psychiatric illness.

Collateral history, with Angela's consent, would be helpful.

Include assessment of her current mental state: in particular, assessment of mood and thinking. Thoughts often change with disturbances of mood. Depressed mood may be associated with thoughts that one is worthless, guilty and remorseful thoughts about relatively minor past indiscretions and pessimistic or hopeless thoughts about the future. Anxious thoughts and particular worries about health may be present. These thoughts can be unrealistic or even delusional in intensity (psychotic depression). The organisation, flow and production of thought, termed 'form' of thought, is also frequently disturbed in depression: there may be fewer thoughts overall and they may be slow.

ANSWER 3

Adopt an empathic and collaborative approach to a comprehensive drug and alcohol assessment. Use principles of motivational interviewing.[12] Provide education regarding dependence (including tolerance and withdrawal) and other long-term issues associated with NSAID use and codeine use.

Principles of motivational interviewing include: expressing empathy, exploring discrepancy, avoiding argument, rolling with resistance and supporting self-efficacy.[12]

A warm and genuine approach facilitates engagement. As discomfort generates change, tactfully highlight inconsistency between how the patient sees their current situation and how they would like it to be. In the process it is important to avoid argument, as confrontation increases resistance and does not help change. When confronted with resistance such as arguing, interrupting, negating and ignoring, explore the reasons for such resistance. Alongside all these techniques in promoting change, it is crucial to build the patient's confidence in their capacity to change.[12]

ANSWER 4

Longer-term management of Angela's issues could involve:

- explaining the relationship between a chronic pain disorder and depression. Emphasise the need to adequately manage her mood in order to improve her pain
- managing her depression and anxiety. Antidepressant medication may be appropriate. If so, consider a serotonin and noradrenalin reuptake inhibitor (SNRI) such as venlafaxine or duloxetine, as these also have pain modulating properties.[11] Side effects should be discussed and monitored for. Benzodiazepines for treatment of anxiety should be avoided as Angela may be at high risk of developing dependence
- reassessing Angela's pain once she has ceased use of ibuprofen/codeine phospate and consider:
  - referral for physical therapy
  - referral to a pain management service
  - referral to drug and alcohol services for discussion of longer-term management options. These services may also be of assistance in providing initial advice regarding cessation of codeine-containing medications
- exploring personality vulnerabilities, as they may affect prognosis and treatment. Comorbid borderline personality disorder may limit the effectiveness of antidepressant treatment. Consider non-pharmacological measures to manage anxiety and mood swings. Examples include breathing exercises and scheduling pleasurable activities. Similarly, practical measures to address current stressors, such as childcare concerns or financial issues, are helpful and important. A GPMHTP and referral to a psychologist to utilise psychological therapies may be helpful in this regard (*Tables 4* and *5*)
- exploring Angela's social supports and encouraging opportunities for social interaction.

CASE 4

BERNADITA IS TEARFUL AND WITHDRAWN

Bernadita, aged 24 years, lives alone and works as an article clerk in a large corporate law firm. You have been her GP since she was a young child, but have seen her infrequently over the years. She has no known psychiatric history.

Bernadita says she has been struggling with low mood recently. She feels heavy and tired, and she has been frequently tearful. She has been sleeping excessively, resulting in frequent lateness to work, and comfort eating, with 4–5 kgs of weight gain. She feels unable to cope with work, lamenting that she has wasted the opportunities her parents worked so hard to give her.

Bernadita was socially active throughout her university years, but has become increasingly withdrawn and stayed at home over the previous 2 months. She has started drinking at least a bottle of wine each night 'just to forget about how I've stuffed everything up and about how pathetic I am'.

QUESTION 1

What further information do you need in order to make a diagnosis?

FURTHER INFORMATION

Bernadita reports that she was 'pretty down' during her final year of high school and a couple of times in university, but says that she had not suffered from any significant depression in the past. She is physically well. Her childhood was relatively uneventful. The only family psychiatric history is a maternal aunt who had 'manic depression.'

QUESTION 2

What is your working diagnosis?

FURTHER INFORMATION

You diagnose an MDE and assess risk. Your management includes commencing venlafaxine 75 mg daily. You arrange to review Bernadita again in 2 weeks.

However, in the meantime, you are called by a psychiatry registrar at the hospital nearby, where Bernadita has been admitted with a manic episode. She had not been sleeping and was giving friends expensive gifts, dressing in flamboyant and provocative clothes at work and acting strangely. Her parents called the crisis team when she contacted them late one night to excitedly say 'goodbye' as she believed she had been asked to report to duty by the secret service to 'save the world.' They reported that Bernadita experienced a similar episode when she was in her first year at university. However, Bernadita's behaviour at that time had not been quite as erratic and had settled after 4 or 5 days.

In hospital, venlafaxine was ceased. Lithium and olanzapine were commenced, effectively treating the manic and psychotic symptoms. You receive a discharge summary cautioning against further use of antidepressant medication and requesting you check Bernadita's serum lithium levels, but little other follow-up advice. Bernadita sees you 2 weeks after discharge and appears to be doing well.

QUESTION 3

What is your revised diagnosis?

FURTHER INFORMATION

You arrange to see Bernadita monthly. One month later she has returned to work, but still experiences a low mood at times. She is worried about what a diagnosis of bipolar disorder means for her future and often thinks about her behaviour when she was an inpatient, feeling very embarrassed. She is also having trouble sleeping. She ceased olanzapine as she believed it hampered her ability to think properly at work. You are concerned that Bernadita has some residual depressive symptoms and has not returned to the mental state and level of functioning she had prior to her most recent episode of illness.

QUESTION 4

How would you manage Bernadita?

CASE 4 ANSWERS

ANSWER 1

Bernadita's presenting symptoms require clarification. Enquire about:

- the duration, severity and pervasiveness of her current symptoms
- the presence of melancholic symptoms, such as worsening of mood in the morning, and psychotic symptoms, such as thought insertion or auditory hallucinations
- concurrent symptoms of an elevated mood suggesting that her current presentation is part of a 'mixed affective episode'
- suicidal ideation – and perform a risk assessment.

Subsequently, clarify Bernadita's past psychiatric history, and:

- exclude unrecognised manic or hypomanic symptoms
- consider whether functional decline has been in conjunction with acute symptoms or has been more insidious.

Also ask about:

- past medical history
- medications
- substance use
- family history of psychiatric illness.

ANSWER 2

The most likely diagnosis is a mood disorder, such as:

- MDD (see *Page 5*) or
- dysthymic disorder.

Other diagnoses to consider would be:

- major depression due to a general medical condition
- substance-induced depression or substance misuse comorbid with depression
- bipolar disorder–depressive episode or mixed affective episode
- prodromal symptoms of a psychosis
- alcohol abuse or dependence comorbid with depression.

Some of Bernadita's symptoms, including hypersomnolence and increased appetite, are termed 'atypical' features of depression. There is some evidence that depression with atypical features is more common in people who have a bipolar mood disorder than a unipolar mood disorder. This is not conclusive and should not be used as a basis for diagnosis.[14] Comorbid anxiety disorders, substance use disorders and personality disorders may also be more common in bipolar disorder and should be screened for.[14]

ANSWER 3

Bipolar disorder type 2 is now apparent, as Bernadita's earlier symptoms occuring while she was at university are consistent with an episode of hypomania.[1] Although her current symptoms meet the criteria for a full-blown manic episode, they do appear to have been due to the effects of antidepressant medication so should not 'count' towards a diagnosis of bipolar disorder type 1.

Bipolar disorder commonly presents with depressive symptoms prior to the onset of any manic or hypomanic symptoms.[15] Although there may be some indicators, bipolar and unipolar depression cannot be reliably distinguished cross-sectionally.

Treatment of bipolar depression with antidepressant monotherapy can induce a manic or mixed affective episode (termed 'switching'). Antidepressant monotherapy may also be ineffective for depressive symptoms or may worsen the cycling nature of the patient's mood, without inducing frank mania.[15]

ANSWER 4

Management of bipolar depression can be difficult and medications often result in incomplete symptom resolution. Bipolar disorder is thought to have a 'polyvalent' aetiology, involving biological, psychological and social factors. Thus, multi-modal treatment is most likely to optimise outcomes.[15]

In general, regarding medications for treatment of bipolar depression:

- first line monotherapy for bipolar depression could involve lithium, lamotrigine, quetiapine or olanzapine[15]
- avoid using an antidepressant medication first line, and certainly not without mood stabiliser 'cover'

- ensure a therapeutic dose, adequate patient adherence and sufficient duration of treatment. Where lithium is prescribed, check serum lithium level; generally, a serum level between 0.4–0.8 mmol/L is considered therapeutic, but check with your local pathology provider
- all medications used to treat bipolar depression have some limitations regarding effectiveness and/or tolerability.[16] See *Table 7* for an outline of medications used in bipolar disorder
- if considering adding or changing medications, specialist psychiatric support is often helpful.

For Bernadita, it would be advisable to:

- check adherence to lithium treatment and check her serum lithium level
- explore further her reasons for ceasing olanzapine
- discuss the treatment options available to her (including adding quetiapine, lamotrigine or sodium valproate to her lithium) and involve her in the decision
- assess her for comorbidities such as alcohol abuse or dependence.

Bernadita would be eligible for a GPMHTP and referral for assessment by a psychiatrist (*Tables 4* and *5*).

Psychological therapy may aid recovery from depressive symptoms, improve social functioning and reduce relapse:[17]

- psycho-education, including mood mapping (charting mood on a daily basis) and discussion about recognition of early warning signs of relapse
- CBT: addressing negative thinking
- IPT and social rhythm therapy: to help Bernadita come to terms with the losses associated with her illness and learn to regulate her activity and sleep.

Lifestyle factors to address include:

- substance abuse or dependence
- exercise
- social connectedness.

Table 7. Medications used for bipolar depression

Medication	Effectiveness	Problems
Quetiapine (300–600 mg/day)	Effective as treatment for bipolar depression Effective as prophylaxis for mania and depression	Significant side effects, especially sedation and weight gain/metabolic effects
Lithium (serum level 0.4–0.8 mmol/L)	Likely to be effective as treatment for bipolar depression, but concerns exist with methods in research to date Effective in reducing suicidality May be effective as prophylaxis for bipolar depression	Slow onset of action Monitoring requirements Significant side effects
Lamotrigine (50–200 mg/day)	Possibly effective as treatment and prophylaxis for bipolar depression Uncertain effectiveness as prophylaxis for mania May be best when used in combination with lithium	Slow titration required Optimal dose unclear Small risk of rash (reduced with slow titration)
Sodium valproate	Little evidence for use in treatment of bipolar depression as a monotherapy May be effective as prophylaxis for bipolar depression Probably effective as treatment and prophylaxis for mania May be best as an adjunct to lithium	Side effects, especially weight gain and sedation Caution in women of childbearing age required (teratogenic)
Antidepressants	Not likely to be effective, either as monotherapy or in combination with a mood stabliser May have a role in a very select group of patients	Should not be used first line or without concurrent mood stabiliser Risk of manic switch or rapid cycling

CASE 5

MARY WANTS TO STOP HER ANTIDEPRESSANT MEDICATION

Mary, aged 32 years, is a married accountant and mother of two children who recently moved to your practice. She has been treated for moderate post-natal depression (PND) with 150 mg of sertraline for the past 12 months. She now feels that she has fully recovered, and has gone back to work part time. Her youngest child is aged 16 months and attending childcare. She wishes to discuss cessation of her antidepressant treatment and management of her depression with complementary therapies and dietary and lifestyle factors.

Mary also recently started seeing a naturopath to treat her mild eczema. She has been otherwise well in the past with no past history of medical illnesses.

QUESTION 1

What factors would you consider in the decision to stop Mary's antidepressant?

QUESTION 2

If Mary had a past history of recurrent MDD, how long should she consider staying on antidepressant medication for?

FURTHER INFORMATION

Following further discussion, you ascertain that Mary has no current symptoms of depression. She has experienced no other episodes of depression other than the PND described, which responded well to treatment. She says that her husband, mother and two close friends are supportive and she has regular contact with other mothers with small children. You decide that it is appropriate to cease Mary's antidepressant.

QUESTION 3

In general, what is your approach to the cessation of SSRIs?

QUESTION 4

What would you say to Mary about the role of complementary therapies and dietary and lifestyle factors in the management of depression?

CASE 5 ANSWERS

ANSWER 1

PND is a common condition, affecting one in seven women.[18] Risk factors include antenatal depression, a traumatic delivery, a past history of MDD, a family history of depression, and limited social support.[19] Although Mary states that her depression has resolved completely, deciding to cease effective treatment also depends on the risk of relapse and the risks associated with relapse. Risks to children from PND are manifold, including disturbed attachment (with significant implications for subsequent interpersonal functioning and mental health in adulthood), neglect, abuse and infanticide. These risks should be carefully explored, including with collateral sources of history.

You should consider the following factors in deciding whether it is appropriate to stop Mary's antidepressant medication:

- Mary's current mental state:
  - assess Mary's current mental state, particularly focusing on her mood, anxiety, energy and the presence of suicidality
- features relating to the episode of PND:
  - its precipitating factors
  - whether it commenced as antenatal or perinatal depression
  - the severity of symptoms experienced during the episode
  - whether suicidality was present or not
  - its response to antidepressant medication including number of trials of different medications necessary before response to treatment
  - the presence of psychotic or manic symptoms during the episode
  - the risks to Mary or others (especially her children) during the episode
  - Mary's insight in relation to the episode
- features relating to Mary's past psychiatric history:
  - previous depressive or manic episodes
  - functioning in between episodes of depression
  - previous treatment with antidepressant medications
- whether Mary has a family history of depression or not
- features relating to her current social circumstances:
  - social supports
  - social stressors. In the presence of current psychosocial stressors, for example, financial strain or relationship problems, it would be advisable to consider deferring withdrawal from her antidepressant until this stress resolves, given the potential for deterioration of her mood in this context.
- Mary's family planning preferences:
  - ascertain whether Mary is planning to have more children. In general, if a woman has experienced PND with one pregnancy, it is more common to experience PND with subsequent pregnancies. If Mary was planning a pregnancy in the future then a discussion involving a risk–benefit analysis about whether to remain on antidepressant medication during pregnancy is warranted (*Table 8*).

ANSWER 2

If Mary had a past history of recurrent MDD, this would indicate a significant potential for relapse. Current evidence suggests a minimum treatment period of 2–5 years of pharmacological treatment to prevent further recurrence.[16]

ANSWER 3

The general principles for antidepressant withdrawal are:

- explaining to the patient the possible effects including anorexia, nausea and anxiety, and the emergence of symptoms suggestive of any underlying depression
- slow weaning over at least 4 weeks (for example, for sertraline 150 mg – a dose reduction of 50 mg every 1–2 weeks is suggested)
- more frequent review during tapering – for example every 2–4 weeks
- monitoring for depressive symptoms and particularly enquiring about worsening sleep, energy levels, reduced concentration, low mood, increasing anxiety and the presence of suicidal features.

ANSWER 4

Complementary therapy use is increasingly widespread.[20] Modifying lifestyle factors potentially contributing to depression is part of an integrative approach to its management. Exercise has been shown to have a small effect size in the treatment of mild to moderate depression.[21] Where possible, use evidence-based complementary therapies alongside 'mainstream' approaches (e.g. medication and psychological therapy).

The role of dietary factors in depression is a focus of investigation: a 'junk food'– style diet may correlate with depression in women.[22] A diet including lean red meat and a high intake of fruit and vegetables may be protective. There is also preliminary evidence for protective effects from a Mediterranean diet,[23] and possible antidepressant effects from seafood and omega-3 fatty acids.[24] Eating regular meals to maintain blood sugar will prevent hypoglycaemia-associated anxiety. Ceasing or reducing caffeine intake may reduce anxiety and improve sleep.

Research suggests there is a link between low vitamin D levels and mild to moderate depression.[25] Evaluating vitamin D levels is particularly important in women with PND who are breastfeeding as their infants may also be at risk of vitamin D deficiency.[25]

Hypericum perforatum (also known as St John's Wort) is the most researched herbal medication for treatment of depression. This agent acts via a similar mechanism to SSRI antidepressant medications and St John's Wort has evidence of effectiveness in treating mild to moderate depression.[26] Issues with purity of the compound and difficulties in estimating the delivered dose of the drug can make its use problematic. St John's Wort also has multiple drug interactions and must not be prescribed with antidepressant medication, given the risk of serotonin syndrome. As it is freely available without a prescription, its use should be routinely enquired about.

Table 8: Risks to consider in prescribing antidepressant medication during pregnancy

	Infant	**Mother**
Risks of prescribing	• Possible teratogenic effects • Short-term toxicity • Possible long-term neuro-developmental effects	• Overdose • Adverse effects • Possible negative impact on therapeutic alliance
Risks of not prescribing	• Infant abuse/neglect • Adverse impact of maternal mental state on the mother–infant relationship	• Relapse of psychiatric illness • Suicide/self-harm/infanticide • Family/relationship deterioration • Use of harmful substitutes

CASE 6

REKIK IS FEELING 'RUN DOWN' AND HAS BEEN LYING AWAKE AT NIGHT WORRYING

Rekik, aged 45 years, is a single teacher who is originally from Bosnia. He has been your patient for 10 years. He presents with 2 months of difficulty sleeping and feeling 'run down'. He has been lying awake at night worrying about his job, his relationships and other aspects of his life. Rekik has missed a few days at work recently because he says 'I just can't get out of bed'.

Rekik has previously sought treatment for asthma and various minor illnesses, but has not sought your help for this problem before. He does not take any medications. He usually does not drink alcohol and has not taken any illicit drugs.

QUESTION 1

What is your differential diagnosis?

QUESTION 2

What further information do you need in order to make a diagnosis?

FURTHER INFORMATION

Rekik describes 3 weeks of low mood, which is worse in the evening. He finds initiating sleep difficult and wakes frequently throughout the night. He has been having a glass or two of wine 'to get to sleep', but no more. Rekik also describes poor appetite and a few kilograms of weight loss. Over the last few weeks, Rekik has lost interest in his usual hobbies of reading and bird-watching, and feels that his concentration is diminished to the point where he has lost track of the lesson while teaching. He feels guilty about this and worries that he is a poor teacher. He has not had any suicidal thoughts.

Rekik has always appeared to be a shy, nervous man and describes himself as being a 'worrier' all his adult life. There is no particular focus to his worry. It includes his work, finances and relationships. His family left Bosnia when he was a young child for his father's work and he did not experience any trauma as a result of the conflict there. You exclude post-traumatic stress disorder (PTSD). Rekik agrees that his fears are excessive and can be reassured. Worsening anxiety pre-dated his low mood. Symptom review reveals no other symptoms. Physical examination is normal. You request some investigations to help exclude physical illness. You diagnose Rekik with GAD and a MDE.

QUESTION 3

What is your initial management plan?

FURTHER INFORMATION

Rekik is initially not keen on CBT as he feels his energy and motivation are too poor to commit to this. You commence him on escitalopram 10 mg. You increase this to 20 mg at 4 weeks after he does not experience any symptomatic relief. Rekik describes only minor improvement after a further 4 weeks at 20 mg, but he agrees to commence CBT. He does find the sleep hygiene techniques you outlined helpful and is able to return to work.

Rekik misses the next appointment with you. After a phone call, he tells you he is going well and will make an appointment following a holiday. He also tells you that he has completed his CBT course and found it helpful.

Three months later, Rekik returns saying that his antidepressant is not working any more. He is experiencing persistent low mood and has lost interest in things he previously enjoyed. He is worried that he will never get back to 'normal'.

QUESTION 4

What are your next steps in managing Rekik and his condition?

CASE 6 ANSWERS

ANSWER 1

The differential diagnosis includes:

- a mood disorder such as MDD
- an anxiety disorder such as GAD, social phobia or PTSD. Rekik may have experienced significant trauma in the past, relating to conflict and war in his country of origin and/or may face ongoing stress or trauma. Depending on the nature and extent of his symptoms, he could have PTSD.

Consider issues particular to Rekik's cultural background as some of his symptoms may be normal within his culture.

ANSWER 2

Obtaining further information should aim to:

- explore Rekik's own sense of his cultural identity and identify culturally bound expressions of distress that can be normal within his culture
- identify or exclude organic factors (*Tables 1* and *2*)
- exclude differential diagnoses,
- assess risk.

To distinguish between anxiety and depression:

- ask about low mood and other symptoms of depression (see *Page 5*)
- ask about anxiety. You need to determine if this is the primary issue.

Table 9 outlines symptoms that can help differentiate between primary anxiety and primary depression, but it important to note that these two disorders often co-exist.

Table 9. Distinguishing between depression and anxiety

Symptoms indicating primary depression	Distinct depressed quality to mood Diurnal variation of mood Psychomotor retardation Negative cognitions (guilt, worthlessness) Suicidal thoughts Change in appetite or weight
Symptoms indicating primary anxiety	Irrational, excessive worry Feeling tense/wound up
Symptoms shared between both diagnoses	Anxiety Irritability Restlessness Poor concentration Insomnia Fatigue

If anxiety is the primary issue, you need to determine what type of anxiety Rekik experiences (*Table 10*). Note that different anxiety disorders commonly co-exist. PTSD should be screened for by tactfully asking about specific traumatic experiences, his response to them at the time, re-experiencing the trauma, emotional numbing and hypervigilance.

ANSWER 3

Given that you have diagnosed comorbid depression and anxiety and Rekik's depression is of moderate severity, his illness warrants both biological and psychological management. Medication, most likely an SSRI, should be commenced. Rekik should also be referred for a course of CBT. Both of these treatments are effective for GAD and depression.[9]

Psychological management includes psycho-education. This includes discussion about depression; collaborative exploration of factors such as vulnerability, triggering and perpetuating factors; and information regarding treatment, including medication, psychological and social strategies, relapse prevention and early warning signs. Rekik's insomnia should be specifically addressed. Education about sleep hygiene techniques should be first line (see *Page 22*).[27] Close follow-up in the initial stages to monitor response to the medication, side effects, and possible risk to self is imperative.

Basic **sleep hygiene** recommendations include maintaining a sleep routine and comfortable pre-bedtime routine; avoiding naps; reserving bed for sleep and sex and avoiding watching television, using a computer or reading in bed; avoiding inappropriate ingestion of caffeine or other substances; exercising regularly; and ensuring the bedroom is quiet and comfortable.[27] If confronted with waking during the night, it is advisable to avoid staying in bed for more than 5 to 10 minutes and instead get out of bed and rest in a chair in the dark (and avoid watching television or using a computer) until feeling sleepy.

More specifically, in order to improve sleep quality, Rekik should be advised to go to bed at the same time and wake up at the same time. His bedroom should be quiet and dark with a slightly cool temperature and clocks should be obscured from view.[27] As coffee, tea and some sodas contain caffeine, which can significantly disrupt sleep,[27] these substances should only be consumed before noon. Cigarettes and alcohol can also interfere with sleep and should be avoided before bedtime.[27] A warm bath, shower, meditation or quiet time may help prepare the mind and body for sleep. Inform Rekik that exercise can help sleep be more continuous. Rekik should aim to exercise before 2 pm as exercise close to bedtime can stimulate the production of endorphins, which interfere with sleep initiation.

ANSWER 4

Management could include:

- reviewing the history to confirm the diagnosis
- checking for and addressing any comorbid substance use disorder
- revisiting and addressing psychosocial stressors. If Rekik has significant problems in interpersonal relationships, managing at work or with grief, he may benefit from a course of IPT. This views depression as intimately linked with interpersonal circumstances and aims to ameliorate depressive symptoms by identifying and working through the particular interpersonal difficulties at play
- ensuring medication is prescribed at an adequate dose, for an adequate duration; is not interacting with other medications; and is being correctly taken
- maximising other lifestyle interventions such as diet, exercise and social opportunities.

Once these issues have been addressed, consider changing medication or adding augmenting medication. In a situation such as Rekik's, switching to an antidepressant from a different class, such as an SNRI, which also has proven efficacy in anxiety disorders, is usually indicated. Alternatively, when considering augmentation of antidepressant medication, lithium has the most evidence, but care must be taken regarding toxicity and side effects.[16] Keep in mind that specific discussions regarding risks and benefits must be undertaken for women where pregnancy may be a possibility or may be considered in the future.

Table 10. Distinguishing between different anxiety (and related) disorders

Anxiety disorder	The patient is anxious about:	Helpful questions
GAD	numerous everyday life events or conflicts	Do you find that you're worried more often than not?
Panic disorder	having a panic attack	A panic attack is a sudden rush of intense fear or discomfort that comes from out of the blue. Have you experienced something like this?
Specific phobia	a specific object/situation	Do you have any particular fears or phobias?
Social phobia	social or performance situations	Do you have fear in social situations or when you have to perform in some way?
PTSD	re-experiencing a traumatic event	Have you ever had a terrible experience where your life was in danger that still bothers you now?
Obsessive compulsive disorder (OCD)	obsessions and compulsions	Do you have any annoying, repetitive thoughts that you can't stop? Is there anything you need to do repeatedly, such as washing or checking?
Hypochondriasis	having a serious illness	Do you spend a lot of time worrying that you have a serious illness and find it hard to be reassured by others?
Somatisation disorder	various physical symptoms	Do you have lots of physical worries that getting in the way of you enjoying life?
Body dysmorphic disorder	a perceived physical flaw	Do you think that you spend more time than others worrying about a particular aspect of your appearance?
Anorexia nervosa	perceived fatness	Do you think that you spend more time than others worrying about your body shape or size?

CASE 7

THOMAS IS TIRED FOLLOWING HIS HEART ATTACK

Thomas, aged 78 years, is a widower who has been your patient for several decades. He lives alone and has always been socially active. His three children live interstate. He has no known psychiatric history. His medical history includes hypertension and hypercholesterolaemia. He has been mildly overweight for many years and does little physical exercise. He gave up smoking 18 years ago.

Thomas consults you for his first appointment following an acute myocardial infarction (AMI) 1 month previously. His recovery was complicated by pneumonia and delirium, with significant physical deconditioning. He had a brief period of inpatient rehabilitation and was discharged 2 weeks prior to you seeing him. His discharge medications included aspirin, atenolol, perindopril, atorvastatin and esomeprazole.

After discharge, Thomas was very anxious. He feared that he would have another heart attack, but would be unable to obtain the help he required in time. He increasingly thought about how close he had come to death and that he might never get his 'life' back. He has struggled with his medication regime and feels irritable. He has little energy and is too tired to bother leaving the house. He mainly eats canned soups and toast. He has lost 8 kg since you last saw him. His children want him to go and stay with them, which he says makes him feel like a child.

QUESTION 1

What is your working diagnosis? What is your differential diagnosis?

QUESTION 2

What investigations would you request to exclude differential diagnoses?

QUESTION 3

What is your initial management plan?

FURTHER INFORMATION

You diagnose an MDE and commence escitalopram. You request relevant investigations to exclude medical causes of his symptoms. You also refer Thomas to an inpatient cardiac rehabilitation program. When you review him 1 month later his mental and physical health are both much improved.

QUESTION 4

What medication side effects should you monitor Thomas for?

CASE 7 ANSWERS

ANSWER 1

Thomas is most likely experiencing an MDE (see *Page 4*). Symptoms consistent with MDE include: low and irritable mood, reduced energy and motivation, hopelessness and loss of weight.

Thomas' anxiety after discharge from hospital may have been a normal response to his experiences. However, it may also be part of depression and can occur in delirium or dementia as a result of diminished capacity to comprehend experiences and environmental stimuli.

MDD is more common in patients with cardiovascular disease and there is a particular connection between acute cardiac events and MDD.[28] Depressive symptoms increase an individual's risk for a first cardiac event and patients with sub-syndromal depressive symptoms prior to a cardiac event are at particular risk of subsequently developing a full MDE.[29]

Identifying depression in patients post-AMI or cardiac intervention is vital as depression significantly influences physical outcomes, including mortality and recurrence of cardiac events, slower recovery, more functional impairment and greater healthcare utilisation.[28, 30]

Commonly, depressive symptoms in patients who are elderly or who have significant medical issues are considered 'normal' or 'expected'. These symptoms are frequently attributed to the effects of the physical illness, especially in the case of symptoms such as fatigue or insomnia. However, unless there is a clearly established and direct physical reason for the symptoms they should 'count' towards a diagnosis of depression.

'Sub-syndromal depression' or milder symptoms may also be particularly common in elderly patients, including in up to 20% of all elderly people.[31] While they may not meet the specific DSM IV-TR™ criteria, there is evidence that at least some of these people may benefit from treatment.

Important diagnoses to exclude in the elderly include:

- depression due to a general medical condition or medication (*Table 1*). Prescribed medications that may be responsible for depressive symptoms should always be considered and enquired about
- delirium. This is common in elderly people who have physical illnesses and the hypoactive form may present like depression. The main differentiating factor is the presence, in delirium, of fluctuating impairment in consciousness and impairment in attention and (usually) orientation[1]
- dementia. The relationship between early dementia and depression is complex and many symptoms are common to both. Cognitive impairment may be the primary presentation of depression and vice versa. Bedside cognitive testing, such as the mini-mental-status examination, may be useful but formal neuropsychological testing is sometimes required to assist in differentiating between these diagnoses.

ANSWER 2

Investigate for medical conditions that may be causing or exacerbating the depressive symptoms (*Table 1*).

Mid-stream urine for microscopy and culture, chest X-ray, blood tests and electrocardiograph (ECG) may help determine a physiological 'cause' of delirium such as a urinary tract infection, pneumonia, electrolyte disturbance or arrhythmia. A diagnosis of dementia is made on the basis of the history, cognitive testing and, usually, cerebral imaging. Less common causes of dementia such as vitamin B12 deficiency may require specific investigations.

ANSWER 3

An initial management plan could include:

- assessment of risk

  Risks of MDD in the elderly may include suicide, physical neglect and falls. Risk assessment helps determine the most appropriate setting for management, and may help to establish the need for use of legal measures to compel treatment. The specific legislation regarding this issue differs across states and territories in Australia

  The history suggests that Thomas' depressive symptoms are impairing his capacity to appropriately care for himself. An inpatient admission may be appropriate. Alternatively, additional services and support may be provided to allow Thomas to receive treatment in his own home. Aged psychiatry teams usually form part of community mental health services and provide outreach for further assessment as well as implementation and modification of treatment

- psycho-education

  Regular, sensitive educational discussions regarding depression, its causes and consequences, including physical health and management strategies the patient may be able to institute, are important.

- medication

  SSRIs are usually better tolerated than other antidepressants in older people and are recommended first line.[9, 32] The SNRIs, such as venlafaxine or duloxetine, are possibly more effective than SSRIs but may also be less tolerated.[29]

  Tricyclic antidepressants have numerous, well-studied adverse cardiovascular effects and are relatively contraindicated in the elderly. Problematic pharmacodynamic and pharmacokinetic interactions with other prescribed medications may also occur. Overall, sertraline is recommended first line in post-AMI patients,[33] but other SSRIs and mirtazapine may be suitable alternatives.[16]

- Increase of psychosocial support
  The relationship between depression and cardiac mortality diminishes as perceived psychosocial support increases.[33] Cardiac rehabilitation programs often deliver psychosocial management and physical rehabilitation, possibly improving physical and psychological parameters, including depression.[35,36] Both inpatient and outpatient programs are described, but availability varies according to geographical location.

  Other psychological and social interventions that should be considered include:

  – psychological therapy. CBT has been specifically studied following AMI and found effective for achieving remission from depression (12–16 sessions).[28] Combining psychological therapy with medication is recommended for moderate or severe depression and may be more effective than either treatment alone.[28]

  – respite care. Where there are significant concerns about elderly patients' safety at home due to physical or psychiatric symptoms or significant carer 'burnout', a short-term period of residential respite may be appropriate to provide a safe environment for implementation of effective treatment and to increase perceived psychosocial support. These services may be accessed through government aged-care assessment services/teams.

  – other community supports. These are available on a temporary or permanent basis, usually through local governments or non-governmental organisations. They include Meals on Wheels, Home Help, shopping assistance, volunteer community visitors, and transport assistance.

ANSWER 4

Differences in the side-effect profiles between the individual antidepressants should be considered. Elderly patients and those with physical comorbidities are more susceptible to medication side effects and/or interactions. Initial side effects on commencement of SSRIs include anorexia, nausea, diarrhoea and dizziness. Other possible side effects include hyponatraemia, postural hypotension and falls, reduced bone density and fractures, bleeding (especially gastrointestinal), and, with some SSRIs (e.g. paroxetine), ECG changes can occur and should be monitored for.[32]

Risk factors for hyponatraemia include: old age, female gender, low body weight, low baseline sodium, concurrent use of medications that may affect sodium levels/renal function (such as diuretics, NSAIDs and carbamazepine), impared renal function, other medical comorbitities (such as hypothyroidism, diabetes, chronic obstructive pulmonary disease, hypertension, cerebrovascular disease or cerebral injury) and numerous cancers.

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th edn. Text revision. Virginia: American Psychiatric Association Ltd, 2000
2. Murtagh JE. General practice. 3rd edn. Sydney: McGraw Hill, 2011
3. Bradvik L, Mattisson C, Bogren M, et al. Long-term suicide risk of depression in the Lundby cohort 1947–1997 –severity and gender. Acta Psychiatr Scand 2008;117(3):185–91
4. Goldney R. Suicide prevention. New York: Oxford University Press, 2008
5. Knox KL, Conwell Y, Caine ED. If suicide is a public health problem, what are we doing to prevent it? Am J Public Health 2004;94(1):37–45
6. Osterberg L, Blaschke T. Adherence to medication. N Engl J Med 2005;353(5):487–97
7. Schuckit MA. Comorbidity between substance use disorders and psychiatric conditions. Addiction 2006;101(Suppl 1):76–8
8. Australian Government Department of Health and Ageing. Medicare benefits schedule. Canberra: AGDHA, 2007 [accessed 5 December 2012]. Available at www.mbsonline.gov.au
9. National Institute for Health and Clinical Excellence. Depression: the treatment and management of depression in adults (update). National Institute for Health and Clinical Excellence, 2009
10. Thapar A, Collishaw S, Pine DS, et al. Depression in adolescence. Lancet 2012;Mar 17;379(9820):1056–67
11. Carr-Gregg M, Manocha R. Bullying – effects, prevalence and strategies for detection. Aust Fam Physician 2011;40(3):98–102
12. Miller WR. Motivational interviewing with problem drinkers. Behavioural Psychotherapy 1983;11:147–72
13. Ivanova JI, Birnbaum HG, Kantor E, et al. Duloxetine use in chronic low back pain: treatment patterns and costs. Pharmacoeconomics 2012;1;30(7):595–609
14. Pae CH, Tharwani H, Marks DM, et al. Atypical depression: a comprehensive review. CNS Drugs 2009;23(12):1023–37
15. Berk M, Berk L, Davey CG, et al. Treatment of bipolar depression. Med J Aust Open 2012;(1 suppl. 4):S32–5
16. Taylor DP, C. Kapur, S. The South London and Maudsley NHS Foundation Trust Prescribing Guidelines. 11th edn. London: Wiley Blackwell, 2012
17. Lauder SD, Berk M, Castle DJ, et al. The role of psychotherapy in bipolar disorder. Med J Aust 2010;193(suppl. 4):S31–5
18. Bener A, Gerber LM, Sheikh J. Prevalence of psychiatric disorders and associated risk factors in women during their postpartum period: a major public health problem and global comparison. Int J Womens Health 2012;4:191–200
19. Leigh B, Milgrom J. Risk factors for antenatal depression, postnatal depression and parenting stress. BMC Psychiatry 2008;8:24
20. MacLennan A, Wilson D, Taylor A. The escalating cost and prevalence of alternative medicine. Prev Med 2002;35:166–73
21. Rimer J, Dwan K, Lawlor DA, et al. Exercise for depression. Cochrane Database Syst Rev 2012;7:CD004366
22. Jacka FN, Pasco JA, Mykletun A, et al. Association of Western and traditional diets with depression and anxiety in women. Am J Psychiatry 2010;167(3):305–11
23. Sanchez-Villegas A, Delgado-Rodriguez M, Alonso A, et al. Association of the Mediterranean dietary pattern with the incidence of depression: the Seguimiento Universidad de Navarra/University of Navarra follow-up (SUN) cohort. Arch Gen Psychiatry 2009;66(10):1090–8
24. Parker G, Gibson NA, Brotchie H, et al. Omega-3 fatty acids and mood disorders. Am J Psychiatry 2006;163(6):969–78
25. Hoang MT, Defina LF, Willis BL, et al. Association between low serum 25-hydroxyvitamin D and depression in a large sample of healthy adults: the Cooper Center longitudinal study. Mayo Clin Proc 2011;86(11):1050–5
26. Linde K, Berner MM, Kriston L. St John's Wort for major depression. Cochrane Database Syst Rev 2008;4:CD000448
27. Morin CM, Espie CA. Insomnia: a clinician's guide to assessment and treatment. New York: Kluwer Academic/Plenum, 2003
28. Lichtman JH, Bigger JT, Blumenthal JA, et al. Depression and coronary heart disease. Recommendations for screening, referral and treatment. Circulation 2008;118:1768–75
29. Rudish B NC. The epidemiology of comorbid coronary artery disease and depression. Biol Psychiatry 2003;54:227–40
30. Frasure-Smith NL, Gravel F, Masson G, et al. Depression and health-care costs during the first year following myocardial infarction. J Psychosom Res 2000;48(4–5):471–8
31. Curran, EM, Loi,S. Depression and dementia. Med J Aust Open 2012(1 suppl. 4):S40–3
32. Coupland C, Dhiman P, Morriss R, et al. Antidepressant use and risk of adverse outcomes in older people: population based cohort study. BMJ 2011;343:d4551
33. Roose S. Treatment of depression in patients with heart disease. Biol Psychiatry 2003;54:262–8
34. Frasure-Smith NL, Gravel F, Masson F, et al. Social support, depression and mortality during the first year after myocardial infarction. Circulation 2000;101:1919–24
35. Enache D, Winblad B, Aarsland D. Depression in dementia: epidemiology, mechanisms, and treatment. Curr Opin Psychiatry 2011;24(6):461–72
36. Ades P. Cardiac rehabilitation and secondary prevention of coronary artery disease. New Engl J Med 2001;345:892–902

RESOURCES FOR DOCTORS

GP Psych Support provides GPs throughout Australia with access to patient management advice from a psychiatrist within 24 hours. It is available by calling telephone number (03) 8699 0414 or at www.psychsupport.com.au

Twenty-four hour mobile on-call psychiatric services are available in most but not all parts of Australia. Contact details vary depending on the state or territory of Australia or location within that state or territory

beyondblue aims to improve community awareness of depression in order to prevent and respond effectively to depression. It is available at www.beyondblue.org.au and provides information for health professionals on depression as well as links to relevant journal articles and resources

Mental Health Professionals Network is available at www.mhpn.org.au. It provides clinical information and allows networking between health professionals who provide mental healthcare to their patients

The Australian Society of Psychological Medicine aims to provide a peer support network for GPs to increase their competency and confidence in managing mental health problems. It is available at www.aspm.org.au

MBS online provides information on Medicare item numbers in relation to the provision of mental healthcare. It is available at www.health.gov.au/internet/mbsonline/publishing.nsf/Content/Medicare-Benefits-Schedule-MBS-1

Information on psychological strategies and motivational interviewing techniques is available in the September 2012 issue of *Australian Family Physician*, which can be found at www.racgp.org.au/afp/2012/september

RESOURCES FOR PATIENTS

beyondblue is available at www.beyondblue.org.au. It provides a symptom checklist for depression, information on predisposing factors and treatment of depression, managing stress and information for carers of people with depression

Sane Australia is available at www.sane.org.au and provides factsheets and podcasts on a variety of mental health issues

Lifeline is available at www.lifeline.org.au. It provides information on depression, a 'coping kit', a search facility to access services according to location and a 24-hour crisis telephone counselling crisis service by calling telephone number 13 11 14

The Post and Antenatal Depression Association is available at www.panda.org.au and provides information about postnatal depression and fact sheets on various issues in relation to parenthood

Mens Line Australia is available at www.menslineaus.org.au. It provides information on common issues affecting mental health as well as 24-hour access to counselling. Counselling is available online, via Skype or telephone 1300 78 99 78

Kids Help Line is available to children aged 5–25 years and is available at www.kidshelp.com.au or telephone 1800 55 1800. It provides 24-hour access to counselling

Headspace is available at www.headspace.org.au and is the National Youth Health Foundation. It provides information on general health, mental health, education, employment and other services, and alcohol and other drug services for young people aged 12–25 years

Australian Indigenous HealthinfoNet is available at www.healthinfonet.ecu.edu.au. It provides culturally appropriate information on a range of medical conditions, including depression, for Indigenous Australians

Mental Health in Multicultural Australia is available at www.mhima.org.au. It has links to articles and reports on mental health as well as links to transcultural services available in some states or territories of Australia

The Australian Psychological Society provides a search facility to help patients find a psychologist based on location and area of specialisation. It is available at www.psychology.org.au

The Royal Australian and New Zealand College of Psychiatry provides a search facility to help patients find a psychiatrist based on location, area of specialisation and primary clinical problem. It is available at www.ranzcp.org

The Better Health Channel is available at www.betterhealth.vic.gov.au and provides general information for patients on a range of medical conditions including depression

The following online programs are available to consumers, but are not a substitute for seeking advice from a health professional where appropriate

- MoodGYM is an interactive web program designed to prevent depression, which utilises CBT skills. It is available at www.moodgym.anu.edu.au
- E-couch is a self-help interactive program with modules for depression, generalised anxiety, social anxiety, relationship breakdown as well as grief and loss. It is available at ecouch.anu.edu.au
- Anxiety online is available at www.anxietyonline.org.au/ and provides information, assessment and treatment for a range of anxiety disorders

Depression

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and
- log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.

FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

QUESTION 1

Murali, aged 33 years, presents with symptoms suggestive of both depression and anxiety. In distinguishing depression from anxiety, which of the following features is MOST likely to indicate depression as the primary, underlying problem?

A. Poor concentration

B. Insomnia

C. Irrational, excessive worry

D. Feeling tense and 'wound up'

E. Diurnal variation in mood.

QUESTION 2

Nick, aged 35 years, presents with low mood, insomnia, appetite loss, fatigue and diminished concentration. You diagnose an MDE and consider the most appropriate treatment to recommend. Regarding treatment of an MDE, which of the following is true?

A. Psychological therapy should not be used where suicidality is present.

B. There is evidence for the effectiveness of CBT in the treatment of depression.

C. IPT is commonly used, but evidence for its effectiveness is lacking.

D. Psychodynamic psychotherapy is commonly used, but evidence for its effectiveness is lacking.

E. Psychological therapy, when used in combination with antidepressant medication, rarely confers benefit over the use of antidepressant medication alone.

QUESTION 3

Paolo, aged 49 years, has a history of recurrent MDD with two major depressive episodes, both treated with antidepressants. He presents with a further episode; you prescribe antidepressants and he achieves remission. In a patient such as Paolo with recurrent MDD, current evidence suggests treatment with antidepressants for which of the following minimum periods to prevent further recurrence?

A. 6–9 months

B. 1 year

C. 2–5 years

D. 10 years

E. Lifelong.

QUESTION 4

Caitlin, aged 17 years, says she has not been enjoying school or her extracurricular activities. She has insomnia, is fatigued and has fallen behind in her homework – about which she has felt very guilty. She has lost 5 kg in weight, but is otherwise physically well. She has been withdrawn, tearful after school and says she hates school. Her mother says Caitlin has been asking to be driven to school. These symptoms have been present for the past month. Based on these initial historical features, what is Caitlin most likely to be experiencing?

A. Bipolar depression, anxiety

B. Personality disorder, MDE

C. MDE, psychosis

D. MDE, bullying

E. Dysthymic disorder, bullying.

QUESTION 5

Stephanie, aged 18 years, presents with mood swings, poor frustration tolerance and feelings of emptiness and isolation. You consider whether she has a personality disorder, but are reluctant to label her with a diagnosis of a personality disorder at this stage. In general, which of the following is true with respect to personality disorder?

A. Symptoms usually arise for the first time in middle age.

B. The criteria for diagnosis of a personality disorder can be met without necessarily causing significant distress or impaired functioning in multiple aspects of a person's life.

C. In the presence of a personality disorder, an MDE cannot be diagnosed.

D. The presence of an MDD may influence interpretation of personality features and contribute to difficulty in coming to a definitive diagnosis of a personality disorder.

E. Medication is the most effective treatment for a personality disorder.

QUESTION 6

Jairo, aged 26 years, is new to your practice and presents with depression. Given that his father suffers from bipolar disorder, you consider if Jairo's depression might be due to bipolar disorder. When considering bipolar disorder and comparing bipolar depression with unipolar depression, which of the following is true?

A. When presenting for the first time, bipolar depression and unipolar depression can be reliably distinguished from one another at the one interview.

B. Atypical features such as hypersomnia appear to be more common in bipolar depression.

C. Bipolar depression appears less likely to be associated with comorbid anxiety disorders.

D. Bipolar depression appears less likely to be associated with substance use disorders.

E. Bipolar disorder usually presents with hypomanic or manic symptoms prior to the onset of depression.

QUESTION 7

Samuel, aged 25 years, has a past history of bipolar disorder. He was on prophylactic medication, but ceased it because he felt he didn't need it any more. He now presents with depression and wants to know about his medication options for treatment of depression. Which of the following should NOT be considered a first line medication option when used alone without other medication in the treatment of Samuel's depression?

A. Sertraline

B. Lithium

C. Quetiapine

D. Olanzapine

E. Lamotrigine.

QUESTION 8

Shakra, aged 28 years, presents with an MDE. He has read about the use of St John's Wort in depression and would like to know more. Which of the following is true regarding the use of St John's Wort in depression?

A. There is very little research regarding the use of St John's Wort in depression.

B. St John's Wort has been shown to be more effective than placebo in treating mild to moderate depression.

C. St John's Wort has more side effects than SSRIs.

D. St John's Wort can be used in conjunction with SSRIs.

E. St John's Wort acts in a similar way to TCAs.

QUESTION 9

Tara, aged 34 years, consulted your colleague 3 months ago, who diagnosed GAD and an MDE. Your colleague excluded medical conditions and a comorbid substance abuse disorder, recommended lifestyle interventions, discussed psychosocial factors, referred Tara to a psychologist for a course of CBT and IPT and commenced escitalopram 20 mg. He subsequently increased it to 40 mg. Tara presents with persistent anxiety and depression. You seek further history to confirm the diagnosis, explore psychosocial factors once again, ask about her engagement with the psychologist, check adherence to medication and decide that further pharmacological intervention is warranted. Which of the following would be the next most appropriate step in relation to management of her medication?

A. Increase the dose of escitalopram to 60 mg.

B. Cease escitalopram and start an SNRI.

C. Cease escitalopram and start a TCA.

D. Continue escitalopram and add quetiapine.

E. Continue escitalopram and add lithium.

QUESTION 10

Maude, aged 78 years, presents to you after being discharged from hospital following an AMI. She takes atorvastatin, metoprolol, ramipril and aspirin. You diagnose an MDE, exclude depression due to a medical condition or medication, assess risk, discuss the diagnosis and explore her social supports. You decide that medication is appropriate and consider your options for treatment of her depression. Which of the following groups of medications or groups of medications would be the LEAST suitable option in Maude?

A. SSRIs

B. SNRIs

C. TCAs

D. Noradrenalin reuptake inhibitors

E. Tetracyclic antidepressants.

check

Independent learning program for GPs

Unit 492 March 2013

Chronic viral hepatitis

RACGP

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Independent learning program for GPs

Chronic viral hepatitis

Unit 492 March 2013

The five domains of general practice
Communication skills and the patient-doctor relationship
Applied professional knowledge and skills
Population health and the context of general practice
Professional and ethical role
Organisational and legal dimensions

RACGP

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This unit of *check* explores the topic of chronic viral hepatitis. It focuses mainly on diagnosis and management of hepatitis B and C given their propensity for chronicity. The importance of recognising individuals at risk cannot be over-emphasised given the morbidity that is often associated with these viruses.

The authors of and contributors to this unit bring a wealth of clinical, research and teaching experience.

The authors of and contributors to this unit are:

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Sonja Hill BSc, a senior project officer at ASHM working primarily on the hepatitis C education and prescriber program. Her interests include the development of resources and online modules in the area of hepatitis C.

The learning objectives of this unit are to:

- develop increased confidence in the diagnosis of hepatitis B and C, including a knowledge of potential presentations, priority populations for testing, what investigations to request and an awareness of the importance of informed consent and accurate interpretation of hepatitis serology
- develop increased confidence in requesting appropriate investigations in patients who present with abnormal liver function tests (LFT) or whose preliminary serological tests for hepatitis B or C virus are positive
- advise patients with chronic hepatitis B or active hepatitis C on lifestyle issues to prevent deterioration of liver function, and advise patients on prevention of transmission
- understand the indications for referral to a specialist 'liver clinic' in patients infected with hepatitis B or hepatitis C virus, and list the factors that affect when to treat with therapy
- describe the standard of care for neonates born to mothers with chronic hepatitis B
- understand the importance of managing a patient in their wider sociocultural context and in light of their other medical problems.

This issue concludes my role as medical editor. I would like to thank the publications team, *gplearning,* and all the authors and reviewers of *check* and extend a warm welcome to the new medical editors, Dr Trisha Boetto and Dr Jill Pope.

Kind regards,

Catherine Dodgshun MBBS, DRANZCOG, FRACGP
Medical Editor, *check* Program

GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

Ab	antibody	BMI	body mass index	HCV	hepatitis C virus
AFP	alpha-fetoprotein	BP	blood pressure	HCV PCR	hepatitis C virus polymerase chain reaction
ALP	alkaline phosphatase	COPD	chronic obstructive pulmonary disease	HDV	hepatitis D virus
ALT	alanine transaminase			HIV	human immunodeficiency virus
anti-HBc	hepatitis B core antibody	CRP	C-reactive protein	IgG	immunoglobulin G
anti-HBe	antibody to the e antigen of hepatitis B	ESR	erythrocyte sedimentation rate	INR	international normalised ratio
		FBE	full blood examination	LFT	liver function test
anti-HBs	hepatitis B surface antibody	GGT	gamma-glutamyl transferase	MSU	midstream urine
anti-HCV	hepatitis C antibody	HAV	hepatitis A virus	NAFLD	non-alcoholic fatty liver disease
APTT	activated partial thromboplastin time	HBeAg	hepatitis B e antigen	PBS	Pharmaceutical Benefits Scheme
		HBsAg	hepatitis B surface antigen	UEC	urea, electrolytes, creatinine
AST	aspartate transaminase	HBV	hepatitis B virus		
BGL	blood glucose level	HCC	hepatocellular carcinoma		

CASE 1

JACK'S PARTNER THINKS HE DRINKS TOO MUCH

Jack, aged 42 years, is new to your practice. He tells you that his partner has sent him to see you, as she thinks he drinks too much. Jack says he likes 'to party' on the weekend when he drinks more than 10 standard drinks (100 g) of alcohol on both Friday and Saturday nights. Otherwise he says he has always been well. On specific questioning, he also reports occasional intravenous use of 'ice' (amphetamines).

Jack reports that he has been well recently and hasn't had any abdominal pain. He has a history of obesity, but has no other relevant past history. He takes no regular medications and has not been prescribed or taken any over-the-counter medications for some time. He has no relevant family history.

On examination Jack is overweight, with a body mass index (BMI) of 31 kg/m² and central abdominal obesity with a waist circumference of 102 cm. Jack's blood pressure is 133/86 mmHg. Abdominal examination reveals no hepatomegaly and he has no peripheral stigmata of chronic liver disease.

Table 1. Jack's LFT results

	Result	Normal reference interval
Bilirubin	17 µmol/L	3–20 µmol/L
Alkaline phosphatase (ALP)	52 U/L	30–110 U/L
Gamma-glutamyl transferase (GGT)	125 U/L	10–50 U/L
Alanine transaminase (ALT)	168 U/L	5–40 U/L
Aspartate transaminase (AST)	173 U/L	5–40 U/L
Total protein	76 g/L	64–79 g/L
Albumin	38 g/L	35–48 g/L

FURTHER INFORMATION

After obtaining informed consent for the appropriate tests, you request blood tests. The results of Jack's LFTs are shown in *Table 1*.

The results of Jack's hepatitis B and C serology are:

- hepatitis B surface antigen (HBsAg) negative
- hepatitis B surface antibody (anti-HBs) 56 mIU/mL
- hepatitis C antibody (anti-HCV) positive

Jack's human immunodeficiency antibody (HIV Ab) is negative. His fasting blood glucose level (BGL) is elevated and there is evidence of dyslipidaemia with hypertriglyceridaemia.

Jack returns to discuss the results, having booked a long consultation at your suggestion.

QUESTION 1

What investigations would you request for Jack?

QUESTION 2

How would you interpret and explain Jack's results to him?

QUESTION 3

What are possible causes for Jack's abnormal LFTs? What is/are the most likely cause/s?

QUESTION 4

What further investigations would you request to determine the cause of Jack's abnormal LFTs?

FURTHER INFORMATION

Hepatitis C virus ribonucleic acid by polymerase chain reaction test (HCV PCR) is negative. Fasting iron studies are normal. Upper abdominal ultrasound reveals changes consistent with fatty liver disease and no evidence of focal pathology.

You determine that Jack has fatty liver disease with alcoholic liver disease.

QUESTION 5

What further management and follow-up would you suggest?

CASE 1 ANSWERS

ANSWER 1

Investigations should include:

- Full blood examination (FBE) as Jack's alcohol intake may be associated with FBE abnormalities
- urea, electrolytes, creatinine (UEC)
- LFTs as Jack's alcohol intake and obesity place him at risk of fatty liver disease, and his drug use places him at risk of hepatitis B and C
- fasting lipids, as Jack's alcohol intake may be associated with hypertriglyceridaemia. In addition, obesity may be associated with dyslipidaemia as part of the metabolic syndrome
- fasting BGL, as obesity may be associated with an elevated fasting BGL as part of the metabolic syndrome
- hepatitis A IgG - to identify whether Jack has immunity and assess the need for hepatitis A immunisation[1]
- HBsAg, anti-HBs and hepatitis B core antibody (anti-HBc)[1]
- anti-HCV[1]
- human immunodeficiency virus antibody (HIV Ab).[1]

Informed consent should be obtained before testing for hepatitis B, C and HIV. This consent should include an assessment of risk, discussion about the reason for the test, the meaning of a positive or negative test result, the window period and ways to prevent infection or reduce the risk of transmission. It should also involve discussion about confidentiality, the availability of treatment, assessment of social supports and arrangement of follow-up to discuss the results.

Sexual health screening should also be performed depending on Jack's sexual history.

ANSWER 2

Jack may require a long appointment to explain his results. You should inform him that:

- he has been infected with hepatitis C, but that further investigation is needed to determine if the infection is active or has cleared. About 25% of people will clear hepatitis C virus (HCV) spontaneously.[2]
- he is immune to hepatitis B
- his HIV Ab test is negative, but that it can take up to 3 months following exposure for a positive result to occur, so retesting is necessary
- his LFTs are abnormal and further investigation is necessary to determine the cause
- he has a metabolic disturbance that can predispose him to conditions such as diabetes. Note that Jack has central obesity (defined as waist circumference ≥94 cm) in combination with raised BP (defined as systolic BP ≥130 mmHg or diastolic BP ≥85 mmHg), raised fasting BGL (≥5.6 mmol/L) and hypertriglyceridaemia (TG ≥1.7 mmol/L) consistent with metabolic syndrome.

This consultation also provides an opportunity to discuss Jack's injecting drug use and alcohol use.

ANSWER 3

There are a number of possible causes for Jack's abnormal LFTs. *Table 2* summarises the causes of abnormal LFTs,[3] which can be divided into causes of cholestasis or hepatocellular damage. Jack's pattern of abnormal LFTs is consistent with hepatocellular damage. The most likely cause of Jack's abnormal LFTs is a combination of obesity-related fatty liver disease and alcoholic liver disease. Hepatitis C might also be causing Jack's abnormal LFTs. Further investigation is necessary.

Jack's LFTs also reveal an isolated elevation of GGT, which could be consistent with recent consumption of excessive alcohol.

Table 2. Classification of LFT abnormalities

Pattern	Laboratory features	Common causes
Cholestasis	ALP >200 U/L ALP more than three times ALT	• Biliary obstruction • Pregnancy (needs further assessment) • Drugs (e.g. erythromycin, oestrogen) • Infiltration (e.g. malignancy)
Hepatocellular damage	ALT >200 U/L ALT >three times ALP	• Infection (e.g. hepatitis B, C, A; Epstein–Barr virus; cytomegalovirus) • Alcohol (AST often >twice ALT) • Fatty liver disease • Drugs (e.g. paracetamol) • Metal overload (e.g.hereditary haemochromatosis, copper overload) • Hypoxia (lactate dehydrogenase usually >1.5 times AST) • Autoimmune conditions

Reproduced with permission from Coates, P. Liver function tests. Aust Fam Physician 2011 Mar;40(3). [3]

ANSWER 4

HCV PCR should be requested to determine if HCV is active or cleared. Fasting iron studies should be requested to help exclude haemochromatosis. Abdominal ultrasound may be helpful in showing signs of fatty liver disease or other pathology. An oral glucose tolerance test also may be indicated.

Other investigations for rarer diseases (such as an autoimmune screen and test for copper overload) should be performed where appropriate.

ANSWER 5

Issues that require further management or follow-up in Jack are listed below.

- Jack's problem drinking and injecting drug use. With respects to Jack's problem drinking, he may benefit from brief intervention in general practice or referral for more in-depth counselling.[4] *Figure 1* outlines an approach to the management of problem drinking in general practice.[4] His drug use should also be addressed. In the longer term, Jack will need regular review of his alcohol and drug intake.
- Jack's HCV status. The fact that HCV PCR is negative is highly likely to mean that HCV has cleared. However, HCV PCR should be repeated in 6 months to confirm HCV clearance. Explain to Jack that previous HCV infection does not provide any protection against future infections of a different genotype (strain) and Jack should have ongoing monitoring if he continues to be at risk from injecting drug use.
- Discussion regarding prevention of transmission and testing of others.
- Vaccination with hepatitis A vaccine if not immune.
- Repeat HIV Ab testing in 3 months.
- Jack's risk for other health problems as part of the metabolic syndrome, including diabetes.[5] It is important to inform Jack about the importance of a healthy diet, regular exercise and maintaining an ideal weight, and to support him in his efforts.
- Jack will need regular review to monitor his weight, LFTs, BGL and serum lipids.

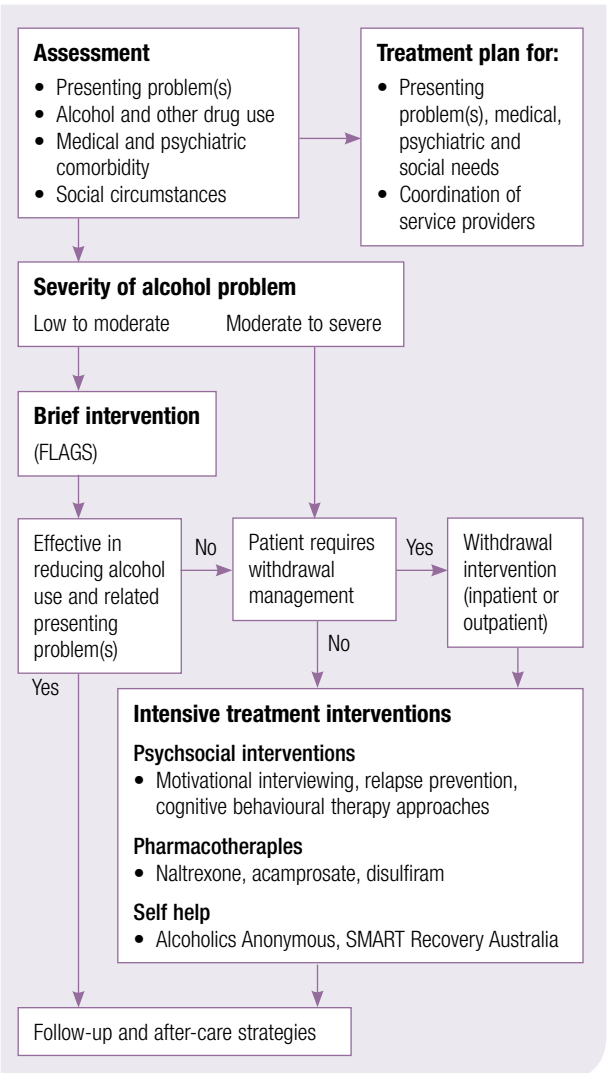


Figure 1. An approach to managing problem drinking in general practice.
Key: FLAGS is an acronym for feedback, listening, advice, goals and strategies.
Reproduced with permission from Demirkol A, Conigrave K, Haber P. Problem drinking management in general practice. Aust Fam Physician 2011 Aug;40(8)576–82.[4]

CASE 2

MICHELLE HAS TESTED POSITIVE TO HEPATITIS C IN THE PAST AND IS THINKING ABOUT HAVING A BABY

Michelle, aged 35 years, has been attending your practice for the past 5 years on an occasional basis. She used heroin in her early 20s, but stopped using after a year of methadone treatment. She previously tested positive for HCV, but wasn't interested in treatment at that time because she had heard that 'interferon can make you crazy'. In the past few years Michelle says she has 'turned her life around' – she is working full-time and is in a stable relationship. Currently, she and her partner are using condoms for contraception. Michelle is thinking about having a child, but is worried about passing on hepatitis C to her partner or baby.

Michelle has no gastrointestinal symptoms, has no other past history and takes no medications. On examination, her BP is 121/76 mmHg, her abdominal examination reveals no hepatomegaly and she has no peripheral stigmata of chronic liver disease.

QUESTION 1

What investigations would you request to assess Michelle in relation to her history of hepatitis C?

FURTHER INFORMATION

You request HCV PCR and LFTs. Michelle's HCV PCR is positive. The results of her LFTs are shown in *Table 3.*

Table 3. Michelle's LFT results

	Result	Normal reference interval
Bilirubin	15 µmol/L	3–20 µmol/L
ALP	72 U/L	30–110 U/L
GGT	43 U/L	10–50 U/L
ALT	224 U/L	5–40 U/L
AST	256 U/L	5–40 U/L
Total protein	73 g/L	64–79 g/L
Albumin	42 g/L	35–48 g/L

QUESTION 2

What advice would you give Michelle about transmission of HCV to her partner or potential unborn child?

QUESTION 3

Michelle asks about the timing of hepatitis C treatment. She wonders if she should try to have a baby now, or have hepatitis C treatment first and then try to conceive. What advice would you give Michelle in relation to the timing of hepatitis C treatment?

QUESTION 4

Are there any other investigations that you could request prior to referral to a 'liver clinic'?

FURTHER INFORMATION

Michelle has genotype 3 hepatitis C with a viral load of 300 000 IU/mL.

QUESTION 5

What information would you give Michelle about her chances of clearing HCV?

QUESTION 6

Michelle asks about common side effects from treatment. What information would you provide?

QUESTION 7

Where could you obtain patient information for Michelle and where would you refer her for peer support?

CASE 2 ANSWERS

ANSWER 1

The main tests to perform are HCV PCR and LFTs to confirm that she has chronic active HCV infection. Additional investigations that you can perform are listed below in *Table 4*.[2] A pregnancy test should also be performed, especially if she is considering treatment.

Investigations that form part of recommended screening prior to planning a pregnancy (such as FBE and rubella IgG) should also be performed and other issues, such as commencement of folic acid, should be discussed.

Table 4. Baseline screening and further investigations in HCV infection

Baseline screening to exclude other conditions that may influence treatment:
• FBE • UEC • international normalised ratio (INR) and activated partial thromboplastin time (APTT) • fasting BGL and lipids • thyroid function tests • liver ultrasound
Investigations to detect other causes of liver disease such as:
• hepatitis A – check hepatitis A IgG, vaccinate if negative • hepatitis B – check anti-HBs, anti-HBc, HBsAg, vaccinate if all negative • HIV – check HIV Ab • haemochromatosis – check iron studies • autoimmune conditions – autoimmune screen • alpha-1 antitrypsin deficiency – alpha-1 antitrypsin level • Wilson disease – serum copper, caeruloplasmin
Determine HCV genotype and quantitative PCR (viral load) as this influences the length of treatment and response to treatment

Reproduced with permission from Australasian Society for HIV Medicine. General practitioners and hepatitis C. Darlinghurst, [Australia]. ASHM 2012.[2]

ANSWER 2

Sexual transmission of HCV is very rare via heterosexual contact, but it can occur occasionally via unprotected anal sex in men who have sex with men. Mother-to-child transmission occurs in about 5% of pregnancies on average.[6]

The possibility of transmission via sharing razors and toothbrushes, although rare, still exists so Michelle should be advised to avoid sharing these items.

ANSWER 3

This is a complex decision for Michelle to make. Commencing treatment now may result in her clearing HCV, but Michelle would then need to delay conception until 6 months after completing

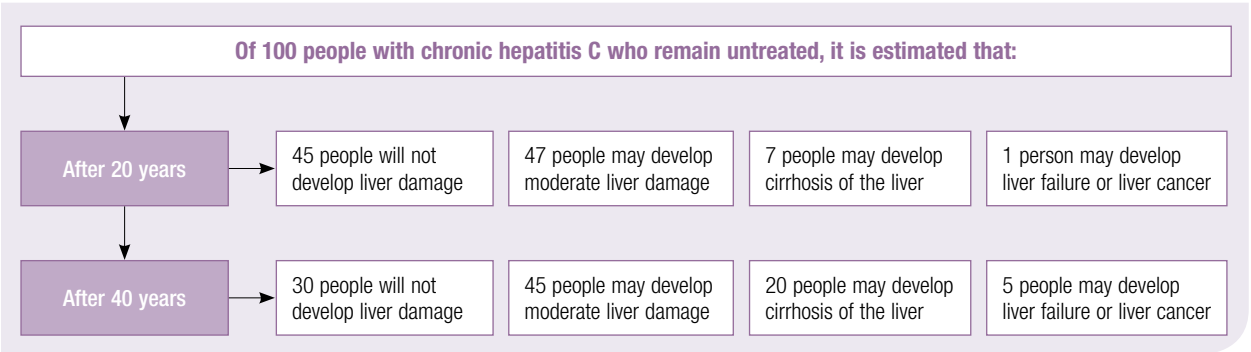


Figure 2. Outcomes for chronic hepatitis C. This figure shows the different potential outcomes for untreated chronic hepatitis C. It does not show the outcome for individual people. Factors such as alcohol intake, age when hepatitis C was acquired, and current level of inflammation may all influence a person's individual outcome. Adapted with permission from Dore G. Hepatitis NSW. 2HepCQuestions. Sydney, NSW: Hepatitis NSW, 2012:9.[7]

antiviral therapy (12–18 months in total), as one component of current treatment – ribavirin – is a teratogen. Alternatively, she may decide it is best to try to start a family now, given her age, and accept the small risk of HCV transmission to her baby. It is important to inform Michelle of the possible outcomes for chronic hepatitis C (see *Figure 2*). [7]

ANSWER 4

Prior to treatment, the HCV genotype and the HCV quantitative PCR (also called the viral load) should be determined.

There are six HCV genotypes, with the three more common types in Australia being 1, 2 and 3.[2] HCV genotype is the most predictive factor associated with the effectiveness of antiviral treatment.[2] HCV genotype testing is funded by Medicare for those patients considering hepatitis C treatment.

ANSWER 5

Michelle has genotype 3, with a HCV viral load <400 000 IU/mL, so she has a good chance of clearing HCV with 24 weeks of pegylated interferon/ribavirin therapy. She should be referred to a specialist clinic. At the clinic, her degree of liver fibrosis is likely to be assessed with a non-invasive test called a FibroScan®. If she has early liver disease, she could reasonably wait for improved treatment to become available in the next 4–5 years.[8] If she has more advanced liver fibrosis, or if she wants to clear HCV, she could have standard pegylated interferon/ribavirin treatment now. If fibrosis is advanced, she will require 48 weeks of treatment. This may influence her decision.

ANSWER 6

There are multiple potential side effects from, as well as contraindications to, the use of interferon/ribavirin.[6] *Table 5* outlines the side effects and contraindications of interferon/ribavirin therapy.[9] Patients require careful assessment prior to treatment, as well as considerable support and monitoring throughout their treatment. This support is provided by multidisciplinary teams, which increasingly involves GPs. Good social/family support networks can also help patients manage side effects. Newer therapies, currently in clinical trials, promise to be more effective and safer to use.

ANSWER 7

Various patient resources are available, such as *Pregnancy, birth and beyond: a resource for women about hepatitis C*, which is available online from the Hepatitis Resource Centre's website (*see Resources*).

For peer support, Michelle can be referred to the Hepatitis Australia National Information line (see *Resources*).

Table 5. Current therapy for hepatitis C: adverse effects and contraindications

Interferon		
Common adverse effects	**Rare adverse effects**	**Contraindications**
• Malaise, fatigue, low-grade fever • Diarrhoea, anorexia, weight loss • Irritability, forgetfulness • Depression, anxiety • Insomnia • Neutropenia • Thrombocytopaenia • Thyroid dysfunction • Decreased sexual libido • Injection-site erythema • Hair thinning/loss • Worsening of psoriasis	• Interstitial lung disease • Cardiomyopathy • Retinopathy	• Decompensated liver disease • Severe depression, psychosis • Uncontrolled diabetes • Cardiac failure • Autoimmune disease • Organ transplantation (other than liver) • Pregnancy/ breastfeeding
Ribavirin		
Common adverse effects		**Contraindications**
• Rash/pruritus • Upper respiratory tract congestion • Haemolytic anaemia (dose dependent) • Teratogenicity		• Renal failure • Pregnancy/ breastfeeding • Inability/ unwillingness to use adequate contraception

Reproduced with permission from Dore G, Temple-Smith M, Lloyd A. Hepitits C: an expanding perspective. Melbourne: IP Communications 2009.[13]

CASE 3

MUSTAFA PRESENTS WITH FATIGUE

Mustafa, aged 58 years, has been attending your practice for the past few years. He was born in Egypt and migrated to Australia when he was 24 years of age with his wife and children. He manages an import business and works about 40 hours a week. Mustafa presents today requesting a 'blood test' as he has been very tired over the past 6 months. He describes feeling physically 'worn out' and has to 'push himself' through the day before collapsing at home exhausted on the couch. On further questioning, he describes vague upper abdominal discomfort and some nausea after meals. Symptom review reveals no other symptoms and he says his mood is normal, he sleeps well from 11 pm to 7am on most nights, eats a balanced diet and his weight has been stable.

Mustafa has a history of schistosomiasis, for which he was treated in Egypt many years ago. He rarely drinks alcohol.

On examination, Mustafa is overweight with a BMI of 27 kg/m^2. He has a few spider naevi and you can just feel a firm liver edge below the costal margin. There is no splenomegaly. The rest of his physical examination is normal.

QUESTION 1

What investigations would you request?

FURTHER INFORMATION

You request blood tests. Mustafa's FBE reveals an anaemia with a haemoglobin of 112 g/L (normal 130–180 g/L) and reduced platelet count of 110 000 × 10^9/L (normal 150–400 × 10^9/L). His fasting BGL is 6.5 mmol/L (normal 3.8–6.0 mmol/L); the results of Mustafa's LFTs are shown in *Table 6*.

Table 6. Mustafa's LFT results

	Result	Normal reference interval
Bilirubin	20 µmol/L	3–20 µmol/L
ALP	56 U/L	30–110 U/L
GGT	88 U/L	10–50 U/L
ALT	180 U/L	5–40 U/L
AST	232 U/L	5–40 U/L
Total protein	62 g/L	64–79 g/L
Albumin	28 g/L	35–48 g/L

QUESTION 2

What further investigations you would request?

FURTHER INFORMATION

You request further blood tests including HBV and HCV serology, iron studies and an oral glucose tolerance test. HBsAg, anti-HBs and anti-HBc are not detected. Anti-HCV is detected and further testing reveals that HCV PCR is positive. An oral glucose tolerance test shows impaired fasting glucose and normal 1-hour and 2-hour readings.

Abdominal ultrasound shows probable cirrhosis.

QUESTION 3

What is the likely diagnosis?

QUESTION 4

Are there any other investigations that you could perform prior to referral to a 'liver clinic'?

FURTHER INFORMATION

Mustafa has genotype 4 HCV, with a viral load of 850 000 IU/mL. His alpha fetoprotein (AFP) is 5 µg/L (normal <20 µg/L).

QUESTION 5

What information would you give Mustafa about treatment with interferon/ribavirin?

QUESTION 6

What would management and follow-up of Mustafa's condition involve?

QUESTION 7

What symptoms and signs might Mustafa have if his liver disease decompensated? What would you do then?

CASE 3 ANSWERS

ANSWER 1

Investigations for lethargy could include:

- FBE
- iron studies
- UEC
- LFTs
- fasting BGL
- thyroid stimulating hormone and, if abnormal, the laboratory will usually perform free thyroxine and free triiodothyronine
- C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR).

ANSWER 2

As Mustafa has signs suggestive of cirrhosis, investigations should include:

- hepatitis A IgG – to identify whether Mustafa has immunity and assess the need for hepatitis A immunisation
- HBsAg, anti-HBs and anti-HBc – as hepatitis B can cause cirrhosis
- anti-HCV – as hepatitis C can cause cirrhosis
- INR – to assess synthetic function of the liver
- fasting iron studies to check for haemochromatosis.[1]

Informed consent for these investigations should be obtained.

Note that Egypt has a high prevalence of hepatitis C, thought to be due to poor infection control standards during the past practise of parenteral therapy for schistosomiasis.[10]

A positive hepatitis C antibody test should be followed by an HCV PCR test to confirm active infection. Given the initial examination and results suggest cirrhosis, Mustafa should be referred for an abdominal ultrasound.

Given Mustafa's raised fasting BGL, an oral glucose tolerance test should be requested.

ANSWER 3

Mustafa is likely to have chronic active HCV infection that has progressed to cirrhosis. Cirrhosis occurs as a consequence of chronic liver disease, but is actually a pathological diagnosis that describes fibrosis and nodular regeneration in the liver.

ANSWER 4

It would be reasonable to check HCV genotype and HCV quantitative PCR. Genotype 4 hepatitis C is common in Egypt, but less common in Australia. In general, genotype 4 is less responsive to treatment than types 2 or 3, but more responsive than genotype 1.

AFP (and abdominal ultrasound) could also be requested to screen for hepatocellular carcinoma (HCC).

ANSWER 5

Cirrhosis without complications is termed 'compensated'.[11] Cirrhosis associated with complications (such as ascites, coagulopathy, variceal bleeding, hepatorenal syndrome or hepatic encephalopathy) is termed 'decompensated'. Treatment with interferon/ribavirin is a treatment option if Mustafa's cirrhosis is compensated. However, in general, decompensated cirrhosis cannot be treated with interferon/ribavirin.[12]

Table 7 reveals a list of some of the markers of cirrhosis.[2]

Table 7. Markers of cirrhosis include:

- Increased INR and prolonged activated partial thromboplastin time (APTT)
- Hypoalbuminaemia
- Thrombocytopaenia
- AST/ALT ratio >1

Reproduced with permission from the Australian Society for HIV medicine. General practitioners and hepatitis C. Darlinghurst, NSW: ASHM, 2012.[2]

ANSWER 6

Mustafa should be referred to a gastroenterologist for assessment, which should also include a discussion about interferon/ribavirin and an upper gastrointestinal endoscopy to exclude oesophageal varices, as well as his long-term management. He will need regular and lifelong follow-up as he has cirrhosis.

Important lifestyle issues should be discussed with Mustafa, including minimising alcohol use and avoiding hepatotoxic medication. HCV infection and cirrhosis are associated with insulin resistance, so weight management is important. Prevention of transmission of HCV should also be discussed.

Mustafa should be monitored for deteriorating liver function and for the development of hepatocellular carcinoma. Screening involves abdominal ultrasound and AFP levels every 6 months.[13] Mustafa's abnormal fasting BGL also needs monitoring, especially during HCV treatment, as interferon may lead to a worsening of glycaemic control.

ANSWER 7

Decompensated cirrhosis may present with jaundice or complications such as ascites, coagulopathy, variceal bleeding, hepatorenal syndrome or hepatic encephalopathy.[13] It may be associated with hypoalbuminaemia or a prolonged INR.

Patients with decompensated cirrhosis should be referred to a specialist centre for liver transplant assessment.

CASE 4

CHENG BRUISES EASILY AND HAS OCCASIONAL MILD RIGHT UPPER QUADRANT DISCOMFORT

Cheng, aged 50 years, migrated to Australia from mainland China with his family 20 years ago. He works as a cleaner in an industrial/commercial cleaning company. He is married and has two adult children who live at home. Cheng is an ex-smoker and has chronic obstructive pulmonary disease (COPD). He does not drink alcohol and has not consumed alcohol in the past. He has no other relevant history and uses a tiotropium inhaler daily.

He presents with a history of 'easy bruising' but has had no gastrointestinal bleeding, weight loss or appetite loss. He reports occasional mild right upper abdominal discomfort. Cheng experiences frequent skin trauma to his hands due to his work with chemical cleaners and frequent hand washing. In addition, the repetitive use of cleaning equipment means that he has constant blisters and often open sores on his hands.

On examination, Cheng's BMI is 22 kg/m^2. Cheng has more than six separate spider naevi on his abdomen and face, and mild palmar erythema. There is evidence of hepatosplenomegaly with a mildly enlarged liver and tippable spleen. Cheng is not jaundiced and has no ascites.

Cheng reports a family history of liver cancer and when asked, is not sure whether his family has been tested or vaccinated for hepatitis B.

QUESTION 1

What is your differential diagnosis? What is the likely diagnosis?

QUESTION 2

What investigations would you request to confirm your diagnosis and assess for complications?

FURTHER INFORMATION

The results of Cheng's LFTs are shown in *Table 8.* Cheng's hepatitis serology reveals:

- HBsAg positive
- anti-HBs negative
- anti-HBc positive.

Table 8. Cheng's LFT results

	Result	Normal reference interval
Bilirubin	19 µmol/L	3–20 µmol/L
ALP	52 U/L	30–110 U/L
GGT	125 U/L	10–50 U/L
ALT	159 U/L	5–40 U/L
AST	209 U/L	5–40 U/L
Total protein	55 g/L	64–79 g/L
Albumin	20 g/L	35–48 g/L

Cheng's FBE reveals an anaemia with a haemoglobin of 101 g/L (normal 130–180 g/L) and a reduced platelet count of 120 000 × 10^9/L (normal 150–450 × 10^9/L).

Cheng's INR is 0.8 and his UEC are within normal limits.

Abdominal ultrasound reveals that Cheng's liver is mildly enlarged with a coarsened echotexture and an irregular contour. No focal lesion is seen within the liver. The spleen is slightly enlarged at 11.5 cm. Portal flow is towards the liver.

QUESTION 3

What further investigations would you request to clarify the phase of hepatitis B?

FURTHER INFORMATION

You request further tests, and the results show that Cheng is HBeAg negative, anti-HBe positive and his serum hepatitis B virus deoxyribonucleic acid (HBV DNA) level (also called the HBV DNA viral load) is 1.65×10^4 IU/mL.

QUESTION 4

What phase of chronic hepatitis B is Cheng currently in?

QUESTION 5

Given Cheng's diagnosis of chronic hepatitis B and family history of liver cancer, what tests would you request to screen for HCC? How frequently would you perform surveillance for HCC?

QUESTION 6

When would you refer Cheng to a specialist for treatment?

QUESTION 7

Given that Cheng has chronic hepatitis B, what would you advise Cheng's family?

CASE 4 ANSWERS

ANSWER 1

Cheng has clinical manifestations (spider naevi, palmar erythema, splenomegaly and 'easy bruising') of cirrhosis. His cirrhosis appears to be compensated, as there is no evidence of jaundice or complications such as ascites, variceal bleeding, hepatorenal syndrome or hepatic encephalopathy. This would need to be confirmed with further investigations and imaging.

The differential diagnosis includes causes of chronic liver disease such as infection with hepatitis B or C, malignancy, alcoholic liver disease, fatty liver disease, autoimmune disease or conditions such as haemochromatosis. Given Cheng's country of origin, absence of alcohol intake and normal BMI, hepatitis B is the most likely cause of his chronic liver disease.

ANSWER 2

Cheng is in one of the priority populations for chronic hepatitis B testing. In order to confirm whether Cheng has chronic HBV infection, exclude other causes of chronic liver disease, ascertain his hepatic function and check for complications, blood tests should include:

- HBsAg, anti-HBs and anti-HBc
- anti-HCV
- LFTs
- FBE
- INR
- UEC
- fasting BGL
- fasting iron studies
- AFP.

An abdominal ultrasound should be requested to confirm the likely presence of cirrhosis and help exclude malignancy.

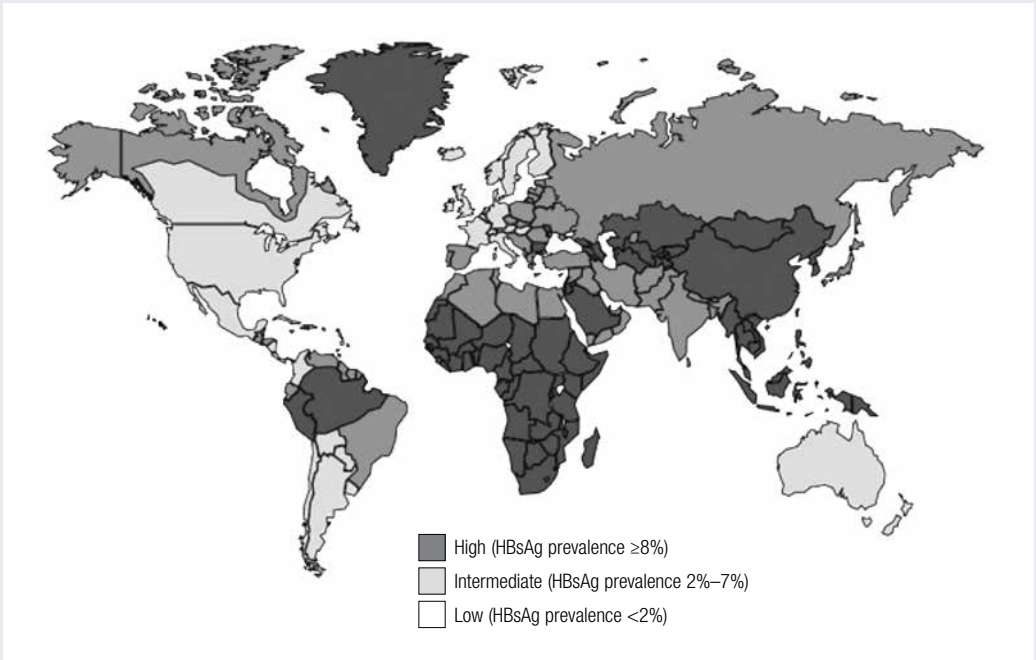


Figure 3. Geographic distribution of hepatitis B.

Reproduced with permission from Centres for Disease Control and Prevention. Geographical distribution of hepatitus B. Atlanta, Georgia: United States Department of Health and Human Services, Centres for Disease Control and Prevention, 2013. Available at www.cdc.gov/immigrantrefugeehealth/guidelines/domestic/viral-hepatitis-figure3.html [accessed 13 February 2013].

FEEDBACK

The priority populations for chronic hepatitis B testing include:

- being born in Asia, Africa, the Middle East, Pacific Islands, Eastern and Southern Europe
- Aboriginal and Torres Strait Islander peoples
- men who have sex with men
- people who inject drugs
- sex workers
- household contacts of individuals diagnosed with chronic hepatitis B.[14,15]

Figure 3 shows the geographic distribution of hepatitis B virus infection.[16]

HBV is one of the world's most common infectious diseases. In 2008, the World Health Organization estimated that 2 billion people had been infected with HBV globally and 350–400 million were living with chronic hepatitis B worldwide.[14] People with chronic hepatitis B experience a high burden of chronic disease and premature death.[17] In countries where hepatitis B infection is endemic, most HBV is acquired at birth or in early childhood and leads to chronic infection. This explains the high proportion of people living with chronic hepatitis B being migrants from high prevalence countries. All patients with chronic hepatitis B require regular monitoring and consideration of treatment.

ANSWER 3

Cheng's AST/ALT ratio is consistent with established cirrhosis. His platelet count is low, which can occur in cirrhosis. Hepatosplenomegaly was evident on examination, which is also consistent with cirrhosis.

Further investigations for HBV to clarify the phase of his chronic hepatitis B include HBeAg and serum HBV DNA level.

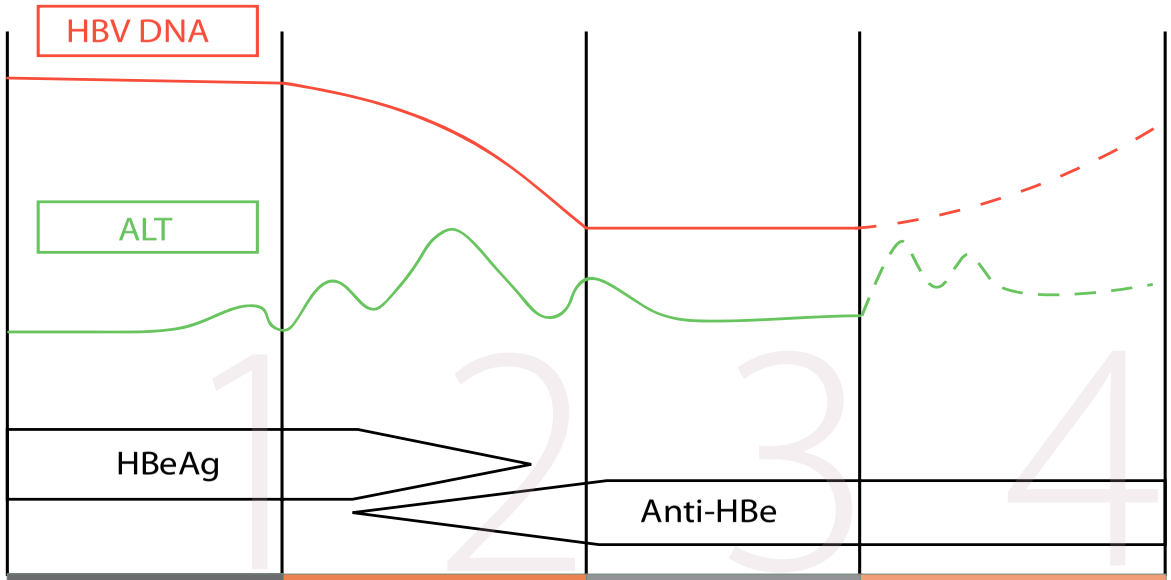
ANSWER 4

Given Cheng's serology, he is in phase IV, also called the 'Immune escape phase'. He has abnormal LFTs, an elevation of his serum HBV DNA level, he is HBeAg negative and anti-HBe positive. He remains infectious and HBV transmission to close household contacts via blood or body fluids is a high possibility. Even though Cheng is HBeAg negative, his HBV DNA is raised, and this in addition to the open sores on his hands would make him somewhat infectious to household contacts. Cheng will need to be advised regarding the risks to his household contacts and work colleagues given the sores on his hands. However, he should be advised that HBV is not spread through kissing, hugging or sharing of food.

Refer to *Figure 4* for a diagrammatic representation of the natural history and phases of chronic hepatitis B.[18] Refer to *Figure 5* for a summary of interpretation of hepatitis B serology.[19]

Decision Making in HBV

Natural History of Chronic HBV
The 4 Phases and Relevance to Treatment Decisions



Immune Tolerance	Immune Clearance	Immune Control	Immune Escape
High HBV DNA, Normal LFTs, HBeAg positive	High HBV DNA, Abnormal LFTs, HBeAg positive At risk of progression to cirrhosis and HCC ***therefore should be referred for consideration of treatment***	Low HBV DNA, Normal LFTs, HBeAg neg; anti-HBe pos	High HBV DNA, Abnormal LFTs, HBeAg neg; anti-HBe pos At risk of progression to cirrhosis and HCC ***therefore should be referred for consideration of treatment***

For more information see: *B Positive – All you wanted to know about Hepatitis B: a guide for primary care providers.* Additional copies and electronic version available at: http://www.ashm.org.au/publications

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Produced April 2010

Funded by the Australian Government, Department of Health and Ageing.

ashm Australasian Society for HIV Medicine · Cancer Council · NHBA National Hepatitis B Alliance · hepatitis australia · GESA · ala · ASID

Figure 4. Australasian Society for HIV Medicine. Decision making in HBV. Natural history of chronic HBV. The 4 phases and relevance to treatment decisions. Reproduced with permission from www.ashm.org.au/images/publications/.patientfactsheets/hbv/decision_making_hbv.pdf [accessed 30 December 2012].

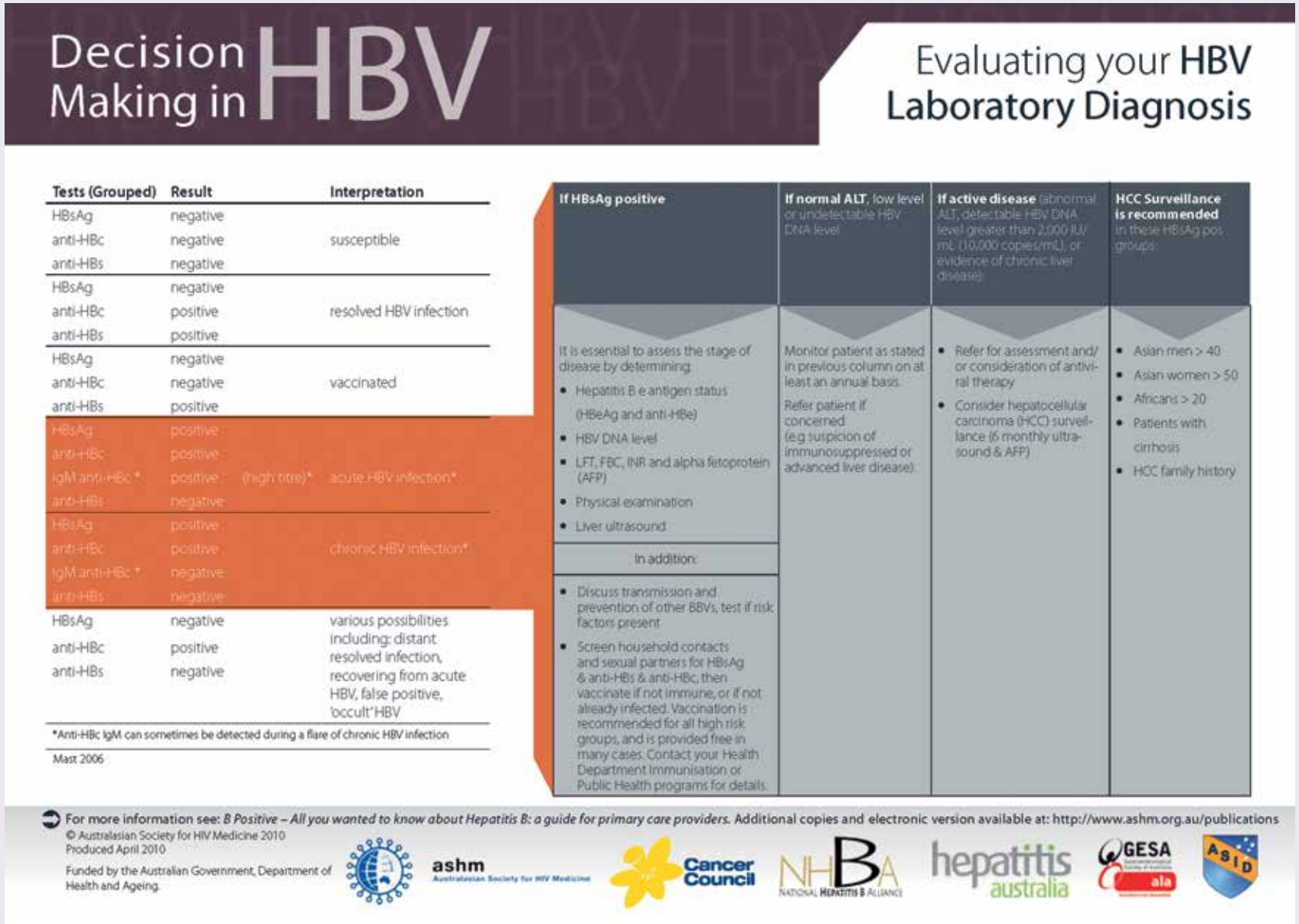

Figure 5. Australasian Society for HIV Medicine. Decision making in HBV. Evaluating your HBV: laboratory diagnosis. Reproduced with permission from www.ashm.org.au/images/publications/patientfactsheets/hbv/decision_making_hbv.pdf. [accessed 30 December 2012].[19]

ANSWER 5

Cheng should be monitored because he is an Asian man aged ≥40 years irrespective of having a family history of liver cancer.

It is recommended that all patients living with chronic hepatitis B be monitored with regular LFTs and serum HBV DNA level testing.[20] The following patients should undergo surveillance for HCC with 6-monthly AFP and liver ultrasound:

- Asian men aged ≥40 years
- Asian women aged ≥50 years
- Africans aged ≥20 years
- people with cirrhosis (see *Case 3*)
- those with a family history of liver cancer (HCC).[20]

FEEDBACK

HCC is an important cancer to screen for. The following facts relate to HCC:

- HCC is the fifth most common cancer in the world and third leading cause of cancer-related death.[21]
- China alone accounts for >50% of newly diagnosed HCC.[22]
- Unlike HCV, HBV-associated HCC may occur in the absence of cirrhosis, although the incidence is higher in people living with cirrhosis.
- Up to one in four adults who become chronically infected with hepatitis B during childhood die from liver cancer or cirrhosis caused by the chronic infection.[23]
- Aboriginal and Torres Strait Islander peoples are also disproportionately affected.

ANSWER 6

Cheng's serum HBV DNA level exceeds 2000 IU/mL and his ALT is elevated >1–2 times the upper limit of normal so he should be referred to a specialist for consideration for treatment. However, due to the evidence of his cirrhosis, he is eligible for treatment at any detectable serum HBV DNA level and irrespective of his ALT result.[15,24]

Since November 2011, the mandatory requirement for liver biopsy to access HBV treatment funded by the Pharmaceutical Benefits Scheme (PBS) has been removed. If required, the patient with chronic hepatitis B can undergo a FibroScan® to assess the extent of his liver fibrosis prior to consideration of treatment.[14,15]

ANSWER 7

Cheng is likely to have been infected with hepatitis B for many years – probably since birth or early childhood. His wife and children should be tested for hepatitis B and, if negative, given vaccination (which is funded for household and sexual contacts of people living with chronic hepatitis B in many jurisdictions) as soon as practicable. It is imperative that if any of Cheng's family have been infected with HBV they should be monitored and referred for treatment as required.

Cheng should also be advised to contact his family in China to urge them to be tested, vaccinated and treated, if appropriate.

CASE 5

SHANE RETURNS FROM ASIA UNWELL

Shane, aged 21 years, is an Australian-born single man, employed in a local electronics store as a salesman. He has recently returned from a trip through Asia where he had heterosexual contact with several partners. He says that only some of these encounters involved using condoms. He ate at many roadside stalls, but did not develop any gastrointestinal symptoms while on holiday. He says that he planned his trip quickly and did not receive any vaccinations before travelling.

Shane lives at home with his parents and two younger siblings, who are all well.

He says he has tried ecstasy tablets, amphetamines (by injection) with clean equipment and heroin on one occasion using clean equipment. He drinks 8–10 standard drinks (80–100 g) of alcohol at parties every 2–3 weeks. Shane takes no medications and has no other relevant past medical history or family history.

Shane presents feeling very unwell, with joint aches and pains but no swelling. In the last 12 hours, he has had anorexia, nausea and some vomiting and pain in his right upper quadrant.

On examination Shane looks unwell and is jaundiced. His weight is 70 kg with a BMI of 21 kg/m^2. He is afebrile and his other vital signs are within normal limits. Abdominal examination reveals tenderness in the right upper quadrant and hepatomegaly with a span of 15 cm. He also has a tippable spleen. There are no masses and ascites is not present. He has no peripheral stigmata of chronic liver disease. His peripheral joints are tender, but are not swollen and there is no overlying erythema. The rest of his physical examination is normal and there are no track marks.

QUESTION 1

What is your differential diagnosis?

QUESTION 2

What investigations would you request to confirm the diagnosis? What problem might you encounter with Medicare funding in requesting these tests?

FURTHER INFORMATION

You obtain informed consent and request various tests in Shane. Shane's LFT results are shown in *Table 9*. His hepatitis serology reveals:

- HBsAg positive
- anti-HBc positive
- anti-HBs negative
- anti-HCV negative
- hepatitis A antibody (HAV Ab) negative.

Table 9. Shane's LFT results

	Result	Normal reference interval
Bilirubin	80 µmol/L	3–20 µmol/L
ALP	130 U/L	30–110 U/L
GGT	120 U/L	10–50 U/L
ALT	6508 U/L	5–40 U/L
AST	3014 U/L	5–40 U/L
Total protein	77 g/L	64–79 g/L
Albumin	38 g/L	35–48 g/L

Shane's human immunodeficiency antibody test (HIV Ab) test is negative.

Shane's FBE reveals an elevated white cell count and his haemoglobin and platelet count are within normal limits. His INR and UEC are within normal limits.

QUESTION 3

Given Shane's preliminary test results, what investigations would you request next?

FURTHER INFORMATION

You request further tests. HBeAg is positive, anti-HBe is negative and hepatitis D virus antibody (HDV Ab) is negative. His serum HBV DNA level is 6.2×10^9 IU/L.

QUESTION 4

What is your clinical diagnosis?

QUESTION 5

What management would you advise for Shane?

QUESTION 6

How is hepatitis B transmitted and what is the likely source in Shane's case? Who of Shane's contacts need to be contacted and managed in this situation?

QUESTION 7

Is HCV excluded by the negative HCV result?

QUESTION 8

What is the prognosis for Shane?

CASE 5 ANSWERS

ANSWER 1

Given Shane's history of travel, his drug use, clinical presentation and unremarkable family history, the most likely diagnosis is acute viral hepatitis. Hepatitis A, B, C, D and E are all possibilities. Other possible causes of Shane's hepatitis are cytomegalovirus or Epstein–Barr virus, but rarely are these quite so acute in their presentation.

While hepatitis A virus (HAV) has become much less common in Australia over the past 30 years, the occasional outbreak does still occur when food supply is contaminated with sewage. It should always be considered in an adult presenting with acute hepatitis, especially if they have been overseas and have not been vaccinated against HAV. Note that in children, acute infection with each of the hepatitis viruses is associated with milder liver disease than is the case with adults.

Drug-induced liver injury and alcohol-related liver inflammation do not usually cause such systemic symptoms and are therefore less likely diagnoses in Shane.

ANSWER 2

It is important to determine the presence or absence of hepatitis, define the cause of that hepatitis and assess the impact of this on Shane's liver. LFTs, FBE, INR and UEC are all appropriate.

For some time now it has not been possible to request a full viral hepatitis screen and receive Medicare rebates on an initial screen for viral hepatitis. If all of the serology for HBV is requested (HBsAg, anti-HBc, anti-HBs), the laboratory may not perform HAV and HCV serology or, if these tests were performed, the patient might receive an account for the latter two. For more detailed information on Medicare funding of hepatitis B tests see *Resources*.

In a situation like Shane's, where the patient is experiencing an acute illness, it may be most practical to request HBsAg, HAV antibody and HCV antibody on the first round of investigations, and once the nature of the infection has been determined, further serology or viral studies can then be requested.

ANSWER 3

The results are consistent with active hepatitis B, which may be acute and the cause of the current symptoms, or it could be chronic and the current symptoms may reflect a flare of chronic hepatitis B. HDV super-infection is excluded by the negative HDV Ab test. It is important to check for HDV co- or super-infection in Shane, as co-infection with any other virus (HCV, HDV, HIV) does alter the clinical course of a viral hepatitis and it may complicate management strategies. It is still possible that Shane has acquired HCV on top of a chronic hepatitis B virus (HBV) infection. Further information is required.

ANSWER 4

The most likely diagnosis is acute hepatitis B infection and in this situation, a serum HBV DNA level is not going to alter management. In adults, acute hepatitis B will result in viral clearance from serum in 95% of cases in a period of 2–6 months.[14] The acute hepatitis reflects an active immune response to the infection and it would be expected that in 6 months, the patient is likely to have normal LFTs and hepatitis serology that reveals:

- HBsAg negative
- anti-HBc positive
- anti-HBs positive
- anti-HeAg negative
- anti-HBe positive.

While seroconversion means that an individual is unlikely to transmit HBV at this stage it must be remembered that the HBV is never completely eradicated from the liver. In older age or if the patient is immune suppressed, the disease may flare again.

ANSWER 5

Currently, while Shane is acutely unwell, management consists of:

- advising him that active treatment of his HBV is not required at present as he has a very high likelihood of clearing the virus himself
- advising him that his liver should settle back to its normal function in the next 2–3 months
- encouraging him to communicate with his sexual contacts to advise them of the need to be tested to confirm their HBV status, if they have not been fully vaccinated previously.

ANSWER 6

In adults, sexual transmission is the most likely means of acquiring HBV infection. In children, mother-to-child transmission (also known as vertical transmission) or transmission from a peer (also known as horizontal transmission) are the common means of acquisition.

ANSWER 7

Shane should be advised that it would be wise to repeat HIV and HCV Ab testing in 3 months as HCV has not been excluded because of the 'window period' for seroconversion. Testing for HCV Ab would also be very helpful if Shane did clear HBV, but his LFTs failed to normalise. In that case HCV infection would be a possible explanation for his persisting abnormal LFTs. Shane should be advised on the appropriate means to prevent transmission in the meantime.

ANSWER 8

At this stage Shane's prognosis should be regarded as good, with the hope that he will recover fully, become immune to HBV and have normal liver function for life.

He should be provided with information about alcohol and drug use and will need regular monitoring in that regard.

CASE 6

HAZEL HAS ABNORMAL LFTs

Hazel, aged 45 years, is a married Indigenous Australian living in Tennant Creek. She has three children aged 20, 18 and 14 years. She presents to see you today for the results of tests that you requested recently in the setting of her diabetes and renewal of her simvastatin prescription for hyperlipidaemia. You note that her medical record states that she is a 'healthy HBV carrier'.
On specific questioning, Hazel says that she has been well lately, with no symptoms such as abdominal pain, vomiting or change in weight.

Hazel has a history of type 2 diabetes, for which she takes metformin. She also has hyperlipidaemia and obesity. On examination her BMI is 30 kg/m^2 and her BP is 125/85 mmHg.

The results of Hazel's LFTs are shown in *Table 10*.

Table 10. Hazel's LFT results

	Result	Normal reference interval
Bilirubin	15 µmol/L	3–20 µmol/L
ALP	130 U/L	30–110 U/L
GGT	253 U/L	10–50 U/L
ALT	234 U/L	5–40 U/L
AST	120 U/L	5–40 U/L
Total protein	77 g/L	64–79 g/L
Albumin	37 g/L	35–48 g/L

Other results reveal the following:

- Hazel's FBE is within normal limits
- her UEC reveal electrolytes that are within normal limits
- her creatinine is 90 µmol/L (normal 60–110 µmol/L)
- her estimated glomerular filtration rate is 79 mL/min/1.73m^2 (normal >90 mL/min/1.73 m^2)
- her HbA1c is 9.5% (normal <6.0%), which is essentially unchanged from her last HbA1c performed 6 months ago
- Hazel's serum lipid studies are within normal limits.

QUESTION 1

What does the term 'healthy carrier' of hepatitis B really mean?

QUESTION 2

What factors in Hazel's history could be contributing to her abnormal LFTs? What further information do you need?

FURTHER INFORMATION

Hazel consumes up to 15 standard drinks (150 g) of alcohol in a drinking session at least once a week. She says she would like to stop, but feels pressured by her family and peers. She is concerned by the abnormal LFTs and asks you for help.

You request hepatitis serology, the results of which reveal that she is:

- HBsAg positive
- anti-HBc positive
- anti-HBs negative.

QUESTION 3

What possibilities now exist to explain Hazel's abnormal LFTs? What further investigations might you request in order to plan further management?

FURTHER INFORMATION

You request further tests and they reveal that Hazel is HBeAg negative, anti-HBe positive and HCV Ab negative. Her serum HBV DNA level is 4×10^5 IU/mL. Her AFP is 7 µg/L (normal <20 µg/L).

Abdominal ultrasound suggests fatty liver and indicates there is no evidence of chronic liver disease, cirrhosis or portal hypertension.

You conclude that Hazel has active hepatitis B infection.

QUESTION 4

What phase of HBV infection is Hazel currently in? Is HBV the only liver problem she is facing?

QUESTION 5

Does Hazel need treatment for her HBV infection? What does treatment achieve?

QUESTION 6

Outline your management plan for Hazel and her relevant contacts.

CASE 6 ANSWERS

ANSWER 1

Hazel's medical record states that she is a 'healthy carrier' of HBV. There is no such thing as a 'healthy carrier'. It is imperative to reassess all chronic HBV-infected patients on a regular basis, as the phase of the disease changes over time. The term 'health carrier' was coined 30 years ago, before the natural history of the disease was well defined. It referred to patients with serological evidence of HBV infection (i.e. HBsAg positivity) but normal LFTs. It then became applied to those who were HBsAg positive and anti-HBe positive with normal LFTs. It is now very well recognised that both of these states are not stable endpoints and patients may progress to active liver disease again.[2]

ANSWER 2

Hazel has abnormal LFTs consistent with a mixed hepatic-cholestatic picture. Possibilities include:

- non-alcoholic fatty liver disease (NAFLD)
- alcohol
- medication
- biliary disease
- HCC
- liver metastases.

Hazel's abnormal liver tests may be the result of various factors, either alone or in concert. Hazel is overweight and this, in its own right, can cause NAFLD. She also has diabetes, which is not well controlled. Diabetes can cause NAFLD in the presence or absence of overt obesity. However, further information, including an alcohol history, is required before making a diagnosis of NAFLD. It is most confidently diagnosed in those who abstain from alcohol. Diagnosis of NAFLD in those who are drinking at recommended 'safe' levels is perilous, as some patients appear to be more sensitive to the toxic effects of alcohol than others[1] and alcohol histories are notoriously unreliable. Medication could also be causing Hazel's abnormal LFTs.

The raised ALP and GGT raise the possibility of some obstructive component to her liver condition and also the less likely presence of a lesion within the liver (such as an HCC or liver metastases).

Further information such as an alcohol history and hepatitis serology, AFP and abdominal ultrasound are required.

ANSWER 3

The tests do not provide a definitive diagnosis and possibilities include:

- active HBV in either the immune clearance or immune escape phase
- fatty liver, which is contributed to by alcohol, diabetes and/or obesity
- biliary disease, HCC or liver metastases.

Further investigations that should be requested include HBeAg and

anti-HBe, serum HBV DNA level, as well as AFP. Hazel should also be referred for an abdominal ultrasound. Refer to *Table 11* for a list of tests that should be performed in hepatitis B positive individuals.[25]

Table 11. Investigations to perform in hepatitis B

Test	Reason why the result is important
HBeAg/anti-HBe serology HBV DNA viral load	Quantify replication, identify phase of infection, identify who to treat and assess prognosis
HAV, HCV, HDV and HIV serology	Identify co-infection and assess the need for vaccination for HAV
LFTs	Assess inflammatory activity and synthetic function
FBE	Identify thrombocytopaenia, which could suggest cirrhosis
INR and APTT	Help establish synthetic function
AFP	Screen for hepatocellular carcinoma
Abdominal ultrasound including portal venous doppler	Assess for the presence of cirrhosis, portal hypertension and hepatocellular carcinoma
All these investigations are rebatable by Medicare for a patient diagnosed with hepatitis B, although there are restrictions on how often HBV DNA viral load can be performed.	
Hepatitis B Program, Epidemiology Unit, Victorian Infectious Diseases Reference Laboratory (2011). New HBV Diagnosis – What now? Reproduced with permission from www.hepBhelp.org.au [accessed 30 December 2012].[25]	

ANSWER 4

Hazel has chronic HBV infection in the immune escape phase with a high HBV DNA viral load (see *Figure 4*). This is likely to be contributing to her abnormal LFTs. She should be considered for HBV treatment, in addition to receiving a management plan for her diabetes, obesity and alcohol intake. It would be helpful to involve an Aboriginal health worker and utilise a multidisciplinary approach. A GP management plan and team care arrangement could be useful in this situation.

In a patient who is HBV positive, the risk of HCV infection needs to be considered. In Australians who have no history of injecting drug use, prevalence of HCV is higher in Indigenous Australians.[26] In general, screening for HCV, HDV and HIV is recommended in HBV infected people as co-infection has a significant impact on their management. To download a Hepatitis B GP Management Plan template see *Resources*.

Hazel also has fatty liver. Reduction in alcohol intake, weight loss and control of diabetes are important. The cause of the cholestatic picture of her abnormal LFTs remains unclear.

People with HBV have an increased risk of HCC. Screening for HCC is indicated for many patients, even in the presence of normal LFTs. The risk for HCC only becomes clinically relevant after a number of years of HBV infection and the Gastroenterological Society of Australia guidelines recommend the appropriate starting age and frequency of screening for populations with chronic HBV infection.[15] In Hazel's case, the absence of cirrhosis and the lack of any family history of HCC place her in a low-risk group and screening is recommended from 50 years of age, with 6-monthly AFP and abdominal ultrasound.

ANSWER 5

Treatment options should be discussed with Hazel and she should be advised of the need to be referred to a specialist unit to discuss treatment further.

Managing her other medical and social issues is imperative if treatment is to be effective in the long-term.

Active treatment of HBV with antiviral agents such as entecavir and tenofovir has been shown to decrease the risk of:

- disease progression
- HCC development
- HBV transmission to other family and personal contacts.

The fact that treatment is not available in Hazel's local town should not stop discussion of referral and treatment, as many services now offer shared care protocols and telemedicine connections to allow treatment without the need for extensive travel.

ANSWER 6

A management plan for Hazel could include:

- addressing alcohol intake – Hazel should be referred to an Aboriginal Health Service for support to reduce alcohol intake and empowerment to be able to avoid alcohol, as her liver disease requires her preferably to abstain in order to avoid progression to cirrhosis. The issue of HBV plus alcohol is important as the two factors together speed the progress of disease complications. Consider pharmacotherapy for alcohol use disorder, but be aware that this is often not acceptable to patients
- attending to her diabetes – check her adherence to metformin (and monitor her renal function), consider adding gliclazide, and if this combination does not control her diabetes adequately, consider other oral agents or insulin therapy
- assessing her further for renal disease
- addressing Hazel's weight issue, and supporting her with an appropriate diet and exercise plan
- discussing referral to a liver or infectious diseases specialist for advice on treatment for her HBV. It is likely that entecavir or tenofovir would be recommended for her HBV infection, and treatment is usually taken life-long. Adherence to treatment is important
- checking the HBV status of her children and partner (test for HBsAg, anti-HBs and anti-HBc) to assess whether they are susceptible, immune or chronically infected. A number of issues are relevant to consider here including whether Hazel's children were vaccinated, and whether vaccination courses were completed. If testing shows that they are susceptible, they should be vaccinated.

CASE 7

KIM-LY IS PREGNANT

Kim-Ly, aged 30 years, is new to your practice and presents for her first antenatal visit with her husband. She recently migrated to Australia from rural Vietnam with her husband and two of her siblings. Kim-Ly has one child aged 2 years and she is now 12 weeks pregnant by certain dates. Kim-Ly's parents are still in Vietnam, as are three of her siblings. She has one sister and one brother in Australia, living in different cities.

You seek the services of a telephone interpreter with her consent and obtain further history. With her first child, Kim-Ly says she had a normal vaginal delivery at term with no antenatal or postnatal complications. Kim-Ly takes no medications and is not aware of any past medical problems. She has never smoked and does not drink alcohol.

Examination reveals a healthy woman with a BMI of 21 kg/m^2, BP of 103/56 mmHg with a uterus consistent with dates, no hepatomegaly, no peripheral stigmata of chronic liver disease and normal cardiovascular, respiratory and thyroid examinations.

QUESTION 1

What are some of the issues that need to be considered in Kim-Ly's management?

QUESTION 2

What investigations would you request?

FURTHER INFORMATION

You obtain informed consent and request blood tests consistent with the antenatal screening recommendations of your local tertiary hospital, but also with an awareness of the conditions Kim-Ly is at greater risk of than the Australian population in general.

Her FBE reveals a haemoglobin of 105 g/L (normal 115–165 g/L) with a mean corpuscular volume of 70 fL (normal 80–100 fL), normal white cell count and platelet count.

Kim-Ly's iron studies are consistent with iron deficiency and her DNA analysis for thalassemia is negative. You ascertain that her iron deficiency is likely to be related to her low intake of dietary iron in the setting of the increased demands of pregnancy. You suggest increasing her intake of iron and commencing iron supplements.

Her hepatitis serology reveals:

- HBsAg positive
- anti-HBs negative
- anti-HBc positive.

Her LFTs are within normal limits, her blood group is A positive, she is protected against rubella and has negative syphilis serology. Her vitamin D level is within the normal range and a midstream urine (MSU) for microscopy and culture is negative.

Given the above results, you request further tests and the results of further hepatitis serology are as follows:

- HCV Ab negative
- HAV total Ab positive; HAV IgM negative
- HBe Ag positive
- anti-HBe negative.

QUESTION 3

How would you interpret Kim-Ly's hepatitis B serology? How would you manage Kim-Ly?

QUESTION 4

What is the most likely source of Kim-Ly's HBV infection?

FURTHER INFORMATION

When you explain to Kim-Ly that she has HBV infection, she appears very anxious and asks if she might have HIV as well. You are unsure why she is concerned about HIV.

QUESTION 5

What is the most appropriate way to approach and address Kim-Ly's concern about HIV?

FURTHER INFORMATION

With interpreter assistance, you assess that:

- Kim-Ly is very anxious about the effect of HBV on her pregnancy
- she requests treatment for her HBV
- she has had no recent risk exposure for HIV
- her family in Vietnam has not been tested for HBV
- her sister in Melbourne has HBV
- her brother is immune to HBV, having been vaccinated

You explain to Kim-Ly about the importance of testing and vaccination for HBV and advise that her family members consult their doctors. You obtain informed consent and request an HIV test.

FURTHER INFORMATION

You refer Kim-Ly to a specialist antental clinic with a link to a specialist HBV treatment service. They measure her serum HBV DNA level and it is 6 x 10^8 IU/L. They determine that antiviral treatment is not indicated at this stage of pregnancy.

QUESTION 6

What is the effect of pregnancy on HBV infection? What is the standard approach to preventing HBV spread from mother to child at delivery?

CASE 7 ANSWERS

ANSWER 1

The following issues need to be considered in Kim-Ly's management:

- effective communication, the use of an interpreter and the effect of culture on health and the patient–doctor interaction, with the need for the doctor to be sensitive and culturally safe
- general health and health in this pregnancy to date
- vaccination history – especially against rubella, pertussis and influenza, and past history of chickenpox
- family history of Kim-Ly and her husband – including of diabetes, multiple births, chromosomal conditions or genetic conditions such as thalassaemia
- social supports and connectedness
- provision of advice regarding the effects of medications, the importance of a healthy diet and exercise, and prevention of infections such as listeriosis and toxoplasmosis
- supplementation with folic acid and iodine, and consideration of supplementation with vitamin D, calcium and iron if deficiency exists
- investigations that should be requested as part of antenatal screening including blood tests
- investigations that should be offered as part of screening for Down syndrome
- discussion about the models of antenatal care.

In particular, two conditions that could have a significant impact on the pregnancy need to be considered in an individual with Kim-Ly's ethnicity. They are thalassaemia and HBV.

Kim-Ly is from a high prevalence country for HBV (>8%)[27] and she is pregnant. HBV is more prevalent than HCV and HIV in Vietnam. HIV has an estimated prevalence of 0.4%, but there are provinces throughout Vietnam with a higher prevalence.[28] HCV is mainly found if injecting drug use has been part of the patient's life. It would be important to gently make enquiries about injecting use, but probably not at the initial appointment, if this is the first time you have met the patient.

ANSWER 2

Suggested screening tests (depending on the guidelines of your local tertiary hospital) include: FBE, blood group and antibodies, HBsAg, rubella IgG and syphilis serology, vitamin D level and MSU for microscopy and culture. Given that Kim-Ly is from a country with a high prevalence of HBV, it would be reasonable to request HBsAg, anti-HBs and anti-HBc and LFTs. HIV may also form part of the recommended screening guidelines, and informed consent for testing is essential.

Screening for Down syndrome should be discussed and offered.

ANSWER 3

Kim-Ly is infected with HBV (she most likely has chronic HBV) and is immune to HAV.

LFTs do change significantly in pregnancy and most obviously in the third trimester. The increase in blood volume leads to a fall in albumin levels, placental production of ALP increases and oestrogens cause a fall in GGT levels. The altered immune status of pregnancy results in less liver inflammation in active viral hepatitis in many patients, but for some with HBV, the opposite occurs and the disease flares during pregnancy.

Kim-Ly's hepatitis B is in the immune tolerance phase (see *Figure 4*), in that she is eAg positive and has normal LFTs. Treatment for her HBV infection is not indicated in the immune tolerance phase but it would be important to:

- ensure she is referred to a specialist antenatal clinic with a link to a specialist HBV treatment service. They will measure her serum HBV DNA level, as she may warrant treatment in the third trimester to minimise transmission of HBV to her baby at delivery.[29] If her HBV viral load is $>10^7$ IU/mL, antiviral therapy needs to be considered. This treatment is in addition to the standard of administration of HBV vaccine and hepatitis B immunoglobulin (HBIg) to the baby at delivery.
- advise Kim-Ly that during and particularly following pregnancy HBV may flare and thus it is important to monitor her liver tests 3 monthly during and for 6 months after the pregnancy
- explain that there is a need to test Kim-Ly's relevant contacts, and offer vaccination to those who are at risk of HBV through current lack of immunity.

This information needs to be discussed with Kim-Ly in the presence of an interpreter to ensure she understands the complexity of this infection and its implication both on her life and that of her baby.[30] Contact the Australian Government's Department of Immigration and Citizenship's Translating and Interpreting Service to engage the services of a telephone interpreter (see *Resources*).

ANSWER 4

In high prevalence countries, the most common means of transmission of HBV infection is from mother to child around the time of birth. This makes it important to try to determine her mother's HBV status and that of her siblings. Refer to the ASHM testing portal (see *Resources*) for more information.

Kim-Ly is in the immune tolerance phase of the disease and this further supports the fact that she has been infected from childhood, as adult exposure to the disease leads to viral clearance from the serum in >95% of cases.[14] Finding HBV in any of her siblings increases the likelihood of her mother being the source of her infection. This must be conveyed in a way that does not lay blame on the mother and instead heightens the need for correct management of this pregnancy to prevent the transmission of HBV to Kim-Ly's baby.

ANSWER 5

It is important to recognise that while many patients will nod assent to their understanding of complex medical issues, there is often a reluctance to acknowledge that they do not understand what is being said.[31] This often applies to people whose first language is not English, be they Australian-born, Aboriginal or Torres Strait Islander peoples or people recently arrived from other countries. Every effort should be made to use interpreter services (either telephone or face-to-face) to facilitate a meaningful discussion.

When testing for blood-borne viruses (HIV, HCV, HBV), informed consent should be obtained. Informed consent for testing means that the person being tested agrees to be tested on the basis of understanding the testing procedures, and the reasons for testing and is able to assess the personal implications of testing. Obtaining informed consent may take more than one consultation. The importance of using an interpreter in this process cannot be over-emphasised. It would be imperative to obtain the results of HIV testing and explain those to Kim-Ly before she sees the specialist.

ANSWER 6

Pregnancy may be associated with both a reduction of HBV activity and occasional flares of activity. Rarely, a patient may experience a severe flare of activity that can be life threatening. After delivery, disease activity can again change, so it is critical that LFTs are monitored 3 monthly through pregnancy and in the early post-partum period.

As indicated in *Answer 3*, standard care to prevent HBV transmission from mother to child is to:

- administer HBV vaccine as soon as possible (preferably within 24 hours) after delivery to all babies born to mothers who are HBsAg positive.[14] Universal vaccination programs mean this is now offered to all children in many countries.
- Give HBIg to the baby (in a different injection site to HBV vaccine) when the mother is HBeAg positive, to minimise risk of transmission.[14]

Note that mode of delivery has no effect on mother-to-child transmission when prophylaxis is given.

1. Australasian Society for HIV Medicine. ASHM testing portal. Darlinghurst [Australia]: ASHM. Available at www.testingportal.ashm.org.au/about/policy-process [accessed 8 February 2013].
2. Australasian Society for HIV Medicine. General practitioners and hepatitis C. Darlinghurst [Australia]: ASHM, 2012.
3. Coates, P. Liver function tests. Aust Fam Physician 2011 Mar;40(3):113–16.
4. Demirkol A, Conigrave K, Haber P. Problem drinking: management in general practice. Aust Fam Physician 2011 Aug; 40(8):576–82.
5. Wittert G, Farrell G, Macdonald G, George J, Weltman M, Storman M. Fatty liver disease. 1st edn. Mulgrave [Australia]: Gastroenterological Society of Australia, 2007.
6. Australasian Society for HIV Medicine. Nurses and hepatitis C. Darlinghurst [Australia]: ASHM, 2006.
7. Dore G. Hepatitis. NSW 2012. 2HepCquestions. Sydney, NSW: Hepatitis NSW, 2012.
8. Dore G. The changing therapeutic landscape for hepatitis C. Med J Aust 2012; 196(10):629–32.
9. Australasian Society for HIV Medicine, The Pharmacy Guild of Australia. Pharmacy and hepatitis C. Darlinghurst [Australia]: ASHM, 2010:4.
10. World Health Organization. Global alert and response: hepatitis C. Available at www.who.int/csr/disease/hepatitis [accessed 8 February 2013].
11. Pavendranathan G, Strasser S. Cirrhosis: a management guide. Medicine Today 2007;Sept.
12. European Association for the Study of the Liver. EASL clinical practice guidelines: management of hepatitis C virus infection. J Hepatol 2011; 55(2):245–64.
13. Dore G, Temple Smith M, Lloyd A. Hepatitis C: an expanding perspective. Melbourne: IP Communications 2009.
14. Matthews G, Robotin M (eds). B Positive: all you wanted to know about hepatitis B: a guide for primary care providers. Darlinghurst, [Australia]: ASHM, 2012.
15. Chronic Hepatitis B recommendations – Australia and New Zealand. 2nd edn. Digestive Health Foundation, Gastroenterology Society of Australia, 2010. Available at www.gesa.org.au/files/editor_upload/File/Professional/CHB.pdf [accessed 8 February 2013].
16. Centers for Disease Control and Prevention. Geographic distribution of chronic hepatitis B virus infection. Atlanta [US]: CDCP, 2008. Available at www.cdc.gov/immigrantrefugeehealth/guidelines/domestic/viral-hepatitis-figure3.html [accessed 8 February 2013].
17. Matthews G, Robotin M (eds). B Positive: all you wanted to know about hepatitis B: a guide for primary care providers. Darlinghurst [Australia]: ASHM, 2012.
18. Australasian Society for HIV Medicine. Decision making in HBV. Natural history of chronic HBV. The 4 phases and relevance to treatment decisions. Darlinghurst [Australia]: ASHM, 2010. Available at www.ashm.org.au/images/publications/patientfactsheets/hbv/decision_making_hbv.pdf [accessed 8 February 2013].
19. Australasian Society for HIV Medicine. Decision making in HBV. Evaluating your HBV: laboratory diagnosis. Darlinghurst [Australia]: ASHM, 2010. Available at www.ashm.org.au/images/publications/patientfactsheets/hbv/decision_making_hbv.pdf [accessed 8 February 2013].
20. Australasian Society for HIV Medicine. Hepatitis B and primary care providers. Darlinghurst [Australia]: ASHM, 2012.
21. McClune AC, Tong MJ. Chronic hepatitis B and hepatocellular carcinoma. Clin Liver Dis 2010 Aug;14(3):461–76.
22. Yuen MF, Hou AL, Chutaputti A. Hepatocellular carcinoma in the Asia Pacific region. J Gastroenterology Hepatol 2009. Mar;24(3):346–53.
23. Lavanchy D. Hepatitis B virus epidemiology, disease burden, treatment, and current and emerging prevention and control measures. J Viral Hepat 2004;11:97–107.
24. Robotin MC, Kansil M, Howard K, George J, Tipper S, Dore GJ et al. Antiviral therapy for hepatitis B-related liver cancer prevention is more cost-effective than cancer screening. Journal of Hepatology, 2009; 990–98.
25. Victorian Infectious Diseases Reference Library. HepB Help. Tests to request in hepatitis B positive individuals. Available at www.hepBhelp.org.au [accessed 8 February 2013].
26. Kirby Institute UNSW. Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander people: surveillance and evaluation report 2011. Sydney: Kirby Institute, The University of New South Wales, 2011.
27. Nguyen DH, Le Tran N, Nguyen DC, Vu Thi Tuong V, Nguyen TH, Nguyen TL, et al. International epidemiological collaborative surveillance, epidemiology and prevention of HBV, HCV, HIV and rabies in the greater Mekong sub-region. Hanoi: Hanoi Medical University, 2010. Available at http://bit.ly/14EYKDN [accessed 7 February 2013].
28. World Health Organization, UNAIDS, UNICEF. UN AIDS report on the global AIDS Epidemic. Joint United Nations Program on HIV/AIDS. WHO: November; 2010.
29. Wiseman E, Fraser MA, Holden S, Glass A, Kidson BL, Heron LG, et al. Perinatal transmission of hepatitis B virus: an Australian experience. Med J Aust 2009;190(9):489–92.
30. Australian Bureau of Statistics. 4228.0. Adult Literacy and Life Skills Survey, summary results, Australia, 2006. Canberra: ABS, 2008. Available at http://bit.ly/XY6dYp [accessed 8 February 2013].
31. Phillips C. Using interpreters. A guide for GPs. Aust Fam Physician 2010; 39(4);188–95.

RESOURCES FOR DOCTORS

- ASHM (Australasian Society for HIV Medicine) is an Australasian organisation supporting the HIV, viral hepatitis and sexual health workforce. Its website is www.ashm.org.au and it provides information on HIV and hepatitis as well as training courses and online modules for doctors. Resources available on its website include:
  - decision making in HBV
  - decision making in HCV
  - hepatitis B and primary care providers
  - general practitioners and hepatitis C
  - nurses and hepatitis C
  - HIV, viral hepatitis and STIs: a guide for primary care
  - B Positive: all you wanted to know about hepatitis B – a guide for primary care
  - co-infection: HIV & viral hepatitis – a guide for clinical management
  - hepatitis C: clinical management in opiate pharmacotherapy settings
  - HIV and viral hepatitis C: policy, discrimination, legal and ethical issues
  - hepatitis B GP management plan template – available at www.ashm.org. images au/ /hbv/hbv_gpmp.rtf [accessed 14 February 2013].
- National hepatitis C and hepatitis B testing policies as well as information on requesting, interpreting and conveying the results of hepatitis B and C serology are available at www.testingportal.ashm.org.au [accessed 14 February 2013].
- HepBHelp is an independent website that aims to assist GPs in the further investigation and management of patients diagnosed with chronic hepatitis B infection. It also provides information on access to the nearest hepatitis B clinic. It is available at www.hepbhelp.org.au [accessed 14 February 2013].
- The Gastroenterology Society of Australia provides recommendations on the management of chronic hepatitis B in its publication *Australia and New Zealand chronic hepatitis B recommendations*. It is available at www.gesa.org.au/files/editor_upload/File/Professional/Chronic%20Hep%20B%20Summary%20Algorithm.pdf [accessed 14 February 2013].
- The Australian Government's Department of Immigration and Citizenship's Translating and Interpreting Service provides access to telephone interpreters via 1300 131 450 (doctors priority line). Further information and registration forms for medical practitioners are available at www.immi.gov.au/living-in-australia/help-with-english/help_with_translating/ [accessed 14 February 2013].
- Information on the delivery of sexual health services with respect and culturally appropriate communication is available in *Cultural respect and communication guide. A resource to assist sexual health service delivery to Aboriginal communities*. It is available at www.tvgpn.org.au/Welcome_files/Programs/ATSI_Health/Sexual_Health_guide.pdf [accessed 14 February 2013].
- The *Australian Immunisation Handbook* provides information on immunisation for hepatitis B and C and is available at www.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook-hepatitisb [accessed 14 February 2013].
- The Medicare Benefits Schedule provides information on Medicare funding of blood tests and is available at www.mbsonline.gov.au/ [accessed 14 February 2013].
- *AUDIT: The alcohol use disorders identification test. Guidelines for use in primary health care* by Babor TF, De la Fuente JR, Saunders J, Babor M (1989), is published by the World Health Organization
- The CAGE questionnaire is an acronym based on four questions used to screen for alcohol abuse. It is available in the *Journal of the American Medical Association*. 252(14);1905–1907, 1984.

RESOURCES FOR PATIENTS

- ASHM is available at www.ashm.org.au and provides hepatitis B fact sheets in a range of languages for people with newly diagnosed hepatitis B. It also provides access to the DVD *C me, hear me. Hepatitis C in our own words*.
- The Australian Capital Territory Hepatitis Resource Centre provides fact sheets and access to a range of publications containing information on hepatitis B and C for patients and is available at www.hepatitisresourcecentre.com.au/resource.html [accessed 14 February 2013].
- Hepatitis Australia provides information for patients on hepatitis B and C. Its website is available at www.hepatitisaustralia.com. It also services a national hepatitis helpline, which are available by telephoning 1300 437 222.
- The Gastroenterological Society of Australia is available at www.gesa.org.au and provides information for patients on a range of gastroenterological conditions including fatty liver disease.

Chronic viral hepatitis

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- **read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and**
- **log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and**
- **complete the online evaluation.**

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.

FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

QUESTION 1

Mahali, aged 64 years, presents to you with symptoms and signs of cirrhosis. You request blood tests, which reveal that he has a hepatitic picture of abnormal LFTs, is HBsAg positive, HBeAg negative, anti-HBe positive and has a high HBV DNA level. Which of the following phases of HBV is Mahali most likely to be currently in?

A. Immune tolerance phase
B. Immune activation phase
C. Immune clearance phase
D. Immune control phase
E. Immune escape phase.

QUESTION 2

Which of the following describes the most appropriate situation with respect to access to treatment for Mahali?

A. He is not currently eligible for treatment and should be monitored with 6-monthly LFTs and annual HBV DNA.
B. An ALT of twice the upper limit of normal is required for him to be eligible for PBS-funded HBV treatment.
C. He requires a liver biopsy to be eligible for PBS-funded HBV treatment.
D. He requires a CT scan confirming the clinical diagnosis of cirrhosis to be eligible for PBS-funded treatment.
E. He is currently eligible for PBS-funded HBV treatment.

QUESTION 3

Tai, aged 53 years, was born in Thailand and has chronic hepatitis B. He does not have cirrhosis and has no family history of HCC. Which of the following best describes the monitoring Tai should undergo to screen for HCC?

A. Three-monthly AFP and abdominal ultrasound
B. Six-monthly AFP and abdominal ultrasound
C. Six-monthly AFP and annual abdominal ultrasound
D. Annual AFP and annual abdominal ultrasound
E. AFP annually and abdominal ultrasound every 2 years.

QUESTION 4

Kartia, aged 28 years, is 38 weeks pregnant and is HBsAg positive, HBeAg positive with normal LFTs and a high HBV DNA. Which of the following best describes the standard of care that is recommended at delivery for Kartia's baby to prevent transmission of HBV?

A. HBIg alone
B. HBV vaccination alone
C. HBIg and HBV vaccination
D. HBIg, HBV vaccination and antiviral therapy for baby
E. HBIg, HBV vaccination, antiviral therapy for baby and avoidance of breastfeeding.

QUESTION 5

Swahali, aged 45 years, presents to you for the first time and says that she has chronic hepatitis B. She would like to know more about infection with HBV. Which of the following is true of HBV or patients infected with chronic hepatitis B?

A. Most HBV is acquired at birth or in early childhood in countries with a low prevalence of HBV.
B. In countries where HBV is endemic, most HBV leads to infection that spontaneously clears.
C. Regions with a high prevalence of HBV include the United Kingdom and Northern Europe.
D. All patients with chronic hepatitis B require regular monitoring.
E. Patients in all phases of hepatitis B infection require antiviral treatment.

QUESTION 6

Matthew, aged 32 years, presented to you with acute hepatitis B 6 months ago. His LFTs are now normal and his serology reveals that he is HBsAg negative, anti-HBc positive, anti-HBs positive, anti-HeAg negative and anti-HBe positive. On the basis of these results, you could inform him that:

A. he has eradicated HBV from the liver
B. he is currently highly infectious
C. he should be considered for treatment
D. the disease could flare in the future if he were immune supressed
E. he needs no further follow-up at any stage.

QUESTION 7

Aiden, aged 35 years, is new to your practice and presents with abdominal pain and nausea. In the course of investigation you discover a hepatitic picture of abnormal LFTs. Subsequent hepatitis serology reveals that he is HBsAg negative, anti-HBc negative and anti-HBs positive. This serology is most consistent with:

A. resolved HBV infection
B. 'healthy carrier' status
C. vaccination
D. a false positive
E. low grade chronic HBV infection.

QUESTION 8

You are giving a presentation on HCV to colleagues in your practice. Which of the following is true of HCV?

A. It is commonly transmitted via sexual means.
B. Mother-to-child transmission occurs in about 5% of pregnancies on average.
C. About 75% of people infected with HCV will clear the infection spontaneously.
D. The presence of anti-HCV on serological testing is consistent with clearance of the infection.
E. A negative antibody test reliably excludes exposure to the infection.

QUESTION 9

Kalem, aged 38 years, has risk factors for acquiring HCV. You request blood tests and note that his results include that he is anti-HCV positive and HBsAg negative and has abnormal LFTs. Which of the following tests would be most appropriate to confirm that he has active infection with HCV?

A. HCV antigen
B. HCV RNA by PCR
C. HCV DNA by PCR
D. Abdominal ultrasound
E. FibroScan®.

QUESTION 10

Which of the following factors best predicts the effectiveness of antiviral treatment for HCV?

A. Age
B. Gender
C. Duration of infection
D. HCV genotype
E. Viral load.

NOTES

check

Independent learning program for GPs

Unit 493 April 2013

Paediatrics

RACGP

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Independent learning program for GPs

Paediatrics

Unit 493 April 2013

The five domains of general practice
Communication skills and the patient–doctor relationship
Applied professional knowledge and skills
Population health and the context of general practice
Professional and ethical role
Organisational and legal dimensions

RACGP

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The publisher, editors and staff of the RACGP would like to farewell Dr Catherine Dodgshun as Medical Editor for *check* Program over the past 2 years and thank her for her outstanding contribution to the College's publishing program. Dr Dodgshun has been an important part of *check* and she leaves a legacy that will enrich the professional lives of medical professionals for years to come.

This unit of *check* explores several issues common in young children. It covers a broad spectrum, from a 7-month-old baby with a urinary tract infection (UTI) to a 5-year-old child with behavioural issues.

Managing a child in general practice can be difficult, in particular because they are unable to give a history themselves and the communication and history-taking must be done with their parents or guardians. In addition, examining an uncooperative child can be frustrating, regardless of whether the non-cooperation is due to illness or behavioural issues. What investigations are performed also need to take into account the age of the child and the invasiveness of the procedure.

The authors of this unit provide a wealth of clinical research and teaching experience.

The authors of this unit are:

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The learning objectives of this unit are to:

- better understand how to manage an infant with a suspected UTI
- develop increased confidence in managing toddlers with a limp
- understand the complexities of investigating a child with a rash
- improve knowledge of how to deal with young children with behavioural problems at home and learning problems at school
- realise the number of illnesses that can present as 'gastro'
- source appropriate support for parents of children with behavioural problems.

We hope that this unit of *check* will assist you to more confidently assess and manage young children.

Kind regards

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GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

ADHD	attention deficit hyperactivity disorder	IBD	inflammatory bowel disease	PEDS–DM	Parents' Evaluation of Developmental Status: Developmental Milestones
CPK	creatinine phosphokinase	LFT	liver function test	RAST	radioallergosorbent test
CRP	C-reactive protein	MAG3 scan	technetium-99m mercaptoacetyltriglycine scan	SUFE	slipped upper femoral epiphysis
CT	computerised tomography	M-CHAT	Modified Checklist for Autism in Toddlers	TSH	thyroid stimulating hormone
DMSA scan	dimercaptosuccinic acid scan	MRI	magnetic resonance imaging	UEC	urea, creatinine and electrolytes
DTPA scan	diethylene triamine pentaacetic acid scan	NICE	National Institute for Health and Clinical Excellence	URTI	upper respiratory tract infection
EEG	electroencephalogram	NSAIDS	non-steroidal anti-inflammatory drugs	UTI	urinary tract infection
ENT	ear, nose, throat	ORS	oral rehydrating solution	WCC	white cell count
ESR	erythrocyte sedimentation rate				
FBE	full blood count				

CASE 1

BILLY IS STRUGGLING AT SCHOOL

Billy, aged 5 years, commenced school 6 months ago. His mother, Sarah, brings him to your surgery because he has been having problems at school. She tells you that his teacher has noticed Billy is inattentive in the classroom and appears to be unable to sit still. He is easily distracted and on occasions, the teacher said, appears to be daydreaming.

According to Sarah, Billy has not made any progress with his learning. During recess he either plays on his own or stands around watching other children at play. He has not made any friends of his own.

You review Billy's file. You check his head circumference, weight and height and compare these with previous data on his growth chart. Billy has been tracking normally since birth. He is up-to-date with his immunisations, and you have seen him several times at your surgery with mild respiratory tract infections as well as an episode of croup at the age of 2 years.

QUESTION 1

What could be the causes for Billy's behaviour at school and his slow learning?

QUESTION 2

How would you assess Billy?

QUESTION 3

What type of dysmorphic features would alert you to a possible genetic problem?

QUESTION 4

What investigations would you request?

QUESTION 5

Who would you refer Billy to?

CASE 1 ANSWERS

ANSWER 1

Billy could have problems that affect one or more of the following areas.[1]

Physical health

The physical fitness of children affects their ability to pay attention, to learn and to play well with other children. Children with visual or hearing impairment or delayed motor or oromotor skills may struggle in school. Children with genetic disorders, chronic illness (e.g. asthma, food allergies and cardiac, neurological and renal disorders) and disturbed sleep may also have difficulty coping at school.

Social and emotional development

The social and emotional development of a child is the best predictor of academic success in early school years. Children who are ready for school have the capacity to self-regulate, express themselves in an appropriate manner, understand the perspectives of others and have pro-social behaviours such as sharing, turn-taking, and empathy. A child with delayed or disturbed social and emotional development can demonstrate separation anxiety or challenging and disruptive behaviours.

Language development

The development of language includes not only speech and listening, but also literacy skills such as reading and writing. Language is required for attaining knowledge. Receptive language is more important than expressive language in predicting school readiness.

Cognition

A child's intellectual ability (reasoning, learning and problem-solving) and adaptive skills (everyday practical and social skills) affect school function. Most children with an intellectual disability are identified in preschool due to developmental delay. Some children with mild intellectual disability may not be identified until they attend school.

ANSWER 2

Take a thorough history from Billy's mother Sarah, examine him and observe his behaviour.[2, 3, 4, 5, 6] Validated behavioural tools can also be used.

The history should include:

- antenatal history
- birth and neonatal history
- current symptoms and past medical history
- family history
- developmental history – enquire about Billy's developmental milestones, and whether concerns were raised by his maternal and child health nurse during early childhood developmental screening
- history of behaviour – ask about any behaviour Sarah is concerned about. Ask for a report from Billy's teacher about his classroom behaviour.

Clinical examination:

- check vital signs and look for signs of chronic disease
- check head circumference, weight and height and compare with previous data on Billy's percentile charts
- test hearing and vision.

General observations and growth parameters are summarised below in *Table 1* and *Table 2* .

Table 1. General observations

Examination feature	Potential clinical implication
Sad, clingy, separation anxiety	Emotional problems
Intrusive, interrupts conversation, impulsive, restless, very active	Disruptive behaviour problems – attention deficit hyperactivity disorder (ADHD)
Oblivious to the adults, poor response to name, no social smile, poor eye contact, inability to follow verbal instructions converse or communicate, unusual tone and pitch of voice	Autism spectrum disorder Developmental language disorder Hearing impairment
Unwashed hair, skin, nails and dirty unkempt look. Poor dental hygiene	Disadvantaged socio-economic factors and neglect
Many vacant staring episodes	Absence epilepsy

Adapted with permission from Von Hahn LE. Specific learning disabilities in children: Role of the primary care provider. In: UpToDate, Basow DS (Ed), UpToDate, Waltham, MA, 2013.[2]

Table 2. Growth parameters

Examination feature	Potential clinical implication
Weight percentile that has plateaued or crossed two percentiles, or weight for height below 80%	Indicates poor nutrition and may suggest chronic underlying disease
Height percentile that has plateaued or crossed two percentiles, or poor growth velocity	Indicates poor growth from underlying chronic disease such as coeliac disease, inflammatory bowel disease or endocrine disorders
Height, weight and head circumference below the 10th percentile with dysmorphic features	Suggests a genetic or chromosomal disorder such as Turner or Noonan syndrome, or foetal alcohol syndrome
Tall stature with large head	Soto syndrome
Tall stature with narrow face, prominent ears and jaw	Fragile X syndrome
Tall stature with small testes	Klinefelter syndrome
Short stature with webbed neck, widely spaced nipples, micrognathia, heart disease	Noonan or Turner syndrome

Adapted with permission from Von Hahn LE. Specific learning disabilities in children: Role of the primary care provider. In: UpToDate, Basow DS (Ed), UpToDate, Waltham, MA, 2013.[2]

Developmental assessment

It is important to use validated tools to assess behavioural problems, such as the Parents' Evaluation of Developmental Status: Developmental Milestones (PEDS-DM). This is a highly accurate, valid tool that provides developmental screening and behavioural screening, plus ongoing surveillance. The PEDS-DM takes 6–7 minutes to complete (see *Resources*).[7,8,9,10]

Specific screening for ADHD can be conducted using the Connors Questionnaire or Vanderbilt Assessment. (see *Resources*).[11,12]

Screening for autism can be performed using the Modified Checklist for Autism in Toddlers (M-CHAT) or by using the Autism Research Centre screening tools for different ages (see *Resources*).

ANSWER 3

The presence of dysmorphic features suggests a genetic disorder, or chromosomal change or foetal exposure to toxins. Look for unusual head shape; slant of the eyes; size of palpebral fissures; space between eyes; epicanthic folds; nasal bridge; size and shape of ears; low set ears; vermillion border of lips; size of mouth and tongue; high arched palate; length of neck; webbed neck; position of hairline; size, shape and number of fingers and toes; and palmar creases.

ANSWER 4

Billy should have his vision and hearing assessed. This should include assessment of short-term auditory memory and auditory figure ground assessment (ability to hear with background noise).

Investigations to consider are:

- full blood evaluation (FBE)
- C-reactive protein (CRP)
- urea, creatinine and electrolytes (UEC)
- thyroid stimulating hormone (TSH)
- iron studies
- liver function tests (LFTs)
- lead levels
- creatinine phosphokinase (CPK)
- urinary metabolic screen
- chromosome karyotype studies
- DNA specific tests (e.g. Fragile X syndrome, Prader–Willi syndrome, Williams syndrome)
- computerised tomography scan (CT scan) of the brain, magnetic resonance imaging (MRI) of the brain and/or electroencephalogram (EEG).

ANSWER 5

Whether Billy needs to be referred depends on your assessment. Often a multidisciplinary approach is needed. If your assessment suggests autism or ADHD, you could refer Billy to a developmental paediatrician or child psychiatrist.

If you have concerns about Billy's social and family situation, refer the family to family support services. If you are concerned about possible abuse, consult child protective services. Billy will need to be referred back to the school (Department of Education) for an educational assessment (including a cognitive assessment and speech and language assessment) and special educational support.

CASE 2

JACK HAS A LIMP

Jack, a 3-year-old boy, has been a patient of yours since his birth. His parents, James and Lisa, bring him into your practice today because they are concerned that he won't bear weight on his left leg.

You have only seen Jack previously for viral illnesses and routine vaccinations. His parents tell you today that 2 days ago they noticed Jack was no longer walking and had started crawling around the house. He commenced walking at 12 months of age and his parents hadn't noticed any issues with his mobility until this current incident.

QUESTION 1

What are your differential diagnoses in Jack?

QUESTION 2

What features would you look for on history and examination to help you make a diagnosis?

FURTHER INFORMATION

You find Jack is afebrile and otherwise well. There is no history of trauma. Clinically, he does not resist passive examination of his hip, which has a good range of movement, although he does complain of pain. His other lower limb joints appear normal and are not painful. You administer some simple analgesia (paracetamol and ibuprofen) and ask Jack's parents to sit with him in your waiting room for a short time.

You call Jack and his parents back into your room and examine Jack again. You note he appears to be putting more weight on his leg, although he still won't walk.

QUESTION 3

What investigations should you order for Jack?

QUESTION 4

How will you manage Jack?

FURTHER INFORMATION

Jack returns for review with you in 48 hours. He remains systemically well. He is still not walking normally, although he is more comfortable and has, on occasion, taken a few steps.

QUESTION 5

What is your working diagnosis?

QUESTION 6

What follow-up would you arrange for Jack?

CASE 2 ANSWERS

ANSWER 1

The potential differentials in a child of Jack's age are very broad. Age is the most important factor in helping decide potential diagnoses (see *Table 3*).

Rather than deciding on a definite diagnosis on the first visit, it is more important to consider dangerous pathologies that need urgent intervention (e.g. septic arthritis and malignancy), rather than more common self-resolving benign pathologies (e.g. transient synovitis).

As with all children with a potential injury, a non-accidental cause should be considered early.

Table 3. Primary differential diagnosis in a child with a traumatic limp

0–3 years of age
Septic arthritis or osteomyelitis
Developmental hip dysplasia
Fracture or soft tissue injury (toddler's fractures or non-accidental injury)
3–10 years of age
Transient synovitis or irritable hip
Septic arthritis or osteomyelitis
Perthes disease
Fracture or soft tissue injury (stress fracture)
10–15 years of age
Slipped upper femoral epiphysis (SUFE)
Septic arthritis or osteomyelitis
Perthes disease
Fracture or soft tissue injury (stress fracture)
Other diagnoses
Haematological disease, such as sickle cell anaemia
Infective disease, such as pyomyositis or discitis
Metabolic disease, such as rickets
Neoplastic disease, such as acute lymphoblastic leukaemia
Neuromuscular disease, such as cerebral palsy or muscular dystrophy
Primary anatomical abnormality, such as limb length inequality
Rheumatological disease, such as juvenile idiopathic arthritis

Reproduced with permission from Perry DC, Bruce C. Evaluating the child who presents with an acute limp. BMJ 2010;341:c4250.[13]

ANSWER 2

The features to be found on history and examination for conditions causing non-weight-bearing in children, particularly those who present with a painful or irritable hip, overlap significantly. The key to not missing dangerous diagnoses is to watch for 'red flags', such as a fever and severe pain, and perform timely clinical review. In particular, irritable hip should be regarded as a diagnosis of exclusion.

Some common presenting features are:[14]

Irritable hip (transient synovitis)

- Most common reason for a limp in the preschool-age group
- Usually occurs in 3–8-year-olds
- History of recent viral upper respiratory tract infections (URTI) (1–2 weeks)
- Child usually able to walk but with pain
- Child otherwise afebrile and well
- Mild-moderate decrease in range of hip movement – especially internal rotation (severe limitation of hip movement suggests septic arthritis).

Perthes disease

- Avascular necrosis of the capital femoral epiphysis
- Age range: 2–12 years (majority are 4–8 years of age)
- Twenty per cent bilateral
- Present with pain and limp
- Restricted hip motion on examination.

SUFE

- Late childhood/early adolescence
- Weight often above the 90th percentile
- Presents with pain in hip or knee, and associated limp
- Hip externally rotated and shortened
- Decreased hip movement – especially internal rotation
- May be bilateral.

Osteomyelitis and septic arthritis

These are the key diagnoses to exclude. A careful clinical history and examination with review is essential. There is significant overlap between these two conditions.

Osteomyelitis	Septic arthritis
• Subacute onset of limp/non-weight-bearing/refusal to use limb	• Acute onset of limp/non-weight-bearing/refusal to use limb
• Localised pain and pain on movement	• Pain on movement and at rest
• Tenderness	• Limited range/loss of movement (With septic arthritis, the hip is often held in flexion, abduction, external rotation to maximise the joint space.)
• Soft tissue redness/swelling may not be present and may appear late	• Soft tissue redness/swelling often present
• With or without fever	• Fever

ANSWER 3

Often no investigations are needed.

In Jack's case, his general appearance and clinical examination are compatible with transient synovitis – although this is a diagnosis of exclusion. Most investigations are non-diagnostic and need to be combined with clinical impression and follow-up.

If an urgent or time-critical diagnosis is being considered, consultation with a local orthopaedic service and referral to an emergency department at this stage is appropriate.

Some possible investigation choices are outlined below.

Blood tests

- CRP is useful for monitoring disease, but not sensitive or specific enough to make a diagnosis. In a small retrospective study, children with a CRP <1 mg/dL had a likelihood of 87% of not having septic arthritis.[15]
- FBE/ESR – white cell count (WCC) and erythrocyte sedimentation rate (ESR) are poorly sensitive and specific individually, although when combined (in Kocher's criteria[16] [see *Table 4*]), they perform moderately well[17] (although this finding failed to be supported on external validation[18]).

Table 4. Kocher's criteria for differentiating septic arthritis from transient synovitis

Factors for predicting septic arthritis
• Fever >38.5°C
• Cannot bear weight
• ESR >40 mm in the first hour
• WCC >12×10^9/L
Probability of septic arthritis
• No factors: <0.2%
• 1 factor: 3.0%
• 2 factors: 40%
• 3 factors: 93.1%
• 4 factors: 99.6%

Reproduced with permission from Kocher MS, Zurakowski D, Kasser JR. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. J Bone Joint Surg Am 1999 Dec;81(12):1662–70. PubMed PMID:10608376.[16]

Radiology

- Plain X-ray
  - Usually non-contributory in children younger than 10 years
  - Useful early if trauma is suspected
  - Essential (with frog leg lateral – supine position with femur externally rotated, flexed and abducted) if SUFE suspected (can be subtle)
  - Changes occur late (<10–14 days in osteomyelitis; <4 weeks in Perthes disease).
- Ultrasound
  - Sensitive for hip effusion, but will not distinguish aetiology (i.e. synovitis vs arthritis)
  - May demonstrate Perthes disease, SUFE
  - Operator dependent.
- Bone scan
  - Sensitive early, better for osteomyelitis than septic arthritis
  - Requires IV access, child needs to hold still, high radiation exposure.
- MRI
  - Provides a lot of detail, but can be difficult to access and often needs general anaesthesia
  - Usually not required to make diagnosis.

ANSWER 4

Jack should be managed with expectant observation. He should be reviewed within 24–48 hours. His parents should be warned about criteria for earlier review or attendance to an emergency department, which include progression of pain (rather than improvement), fever, lethargy or parental concern.

ANSWER 5

The working diagnosis is transient synovitis. Jack appears to be improving with simple analgesia, and he remains well.

ANSWER 6

Review Jack within 1–2 weeks. The natural history of transient synovitis is for return to normal activity within this time frame. If the limping persists past 2 weeks, other diagnoses (such as Perthes disease or bone malignancy) need to be considered, and suitable investigation and follow-up arranged. Plain X-ray, if not already performed, and discussion with an orthopaedic service would be appropriate.

CASE 3

LUCIA HAS A RASH

Lucia, a 2-year-old girl, is brought in by her mother Sue with an itchy rash that has been present for the past 4 months. Recently, the rash has been crusty and weeping and Lucia has sometimes scratched herself to the point where her skin bleeds. The rash is particularly troublesome around her elbows and behind her knees and ankles. She occasionally gets spots on her chin and cheeks.

Sue says the rash got worse after a family camping holiday when she regularly applied sunscreen to Lucia. She has tried a steroid cream, purchased over the counter at the local pharmacy, but is reluctant to use the cream as she has been cautioned about the side effects. When she applied the cream sparingly, it did not appear to make any difference. Sue is regularly applying a greasy moisturiser after Lucia's bath. She has, however, reduced the number of Lucia's baths to 2–3 per week as she has been told that bathing will dry her skin more.

Sue says Lucia had sensitive skin as a baby, and she would become 'rashy' after bubble bath was used in her bath or after being washed with soap. She now generally avoids these products.

Lucia is otherwise well. She was breastfed exclusively until she was 5 months of age, with Sue maintaining a normal diet herself while breastfeeding. Solid foods were introduced into Lucia's diet from 5 months, and Lucia now enjoys a varied family diet with no restrictions.

Her diet includes eggs, milk, wheat, soy, peanuts, tree nuts, fish and shellfish. Lucia's favourite food is peanut butter sandwiches.

Lucia has a baby sister Jessica, who is 3 months of age and well and thriving. She also has a 5-year-old brother, Harry, who recently had school sores.

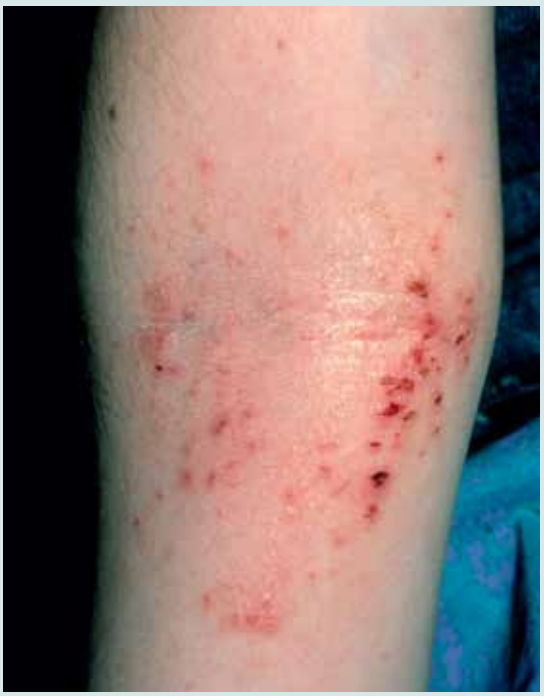

Figure 1. An example of Lucia's rash.
Photo from Science Photo Library

Lucia's father Paul had eczema as a child, and Sue gets hayfever during the pollen season.

Lucia has just commenced sleeping in a single bed. She sleeps with a lambs' wool underlay, woollen blankets and several soft cuddly toys. There are two family cats that sleep in the laundry.

Sue asks you whether she should withdraw milk and gluten-containing foods as well as peanut butter from Lucia's diet in case these are contributing to her rash. She also requests allergy testing to see what might be causing the rash.

QUESTION 1

What is the likely diagnosis?

QUESTION 2

Are any investigations required?

QUESTION 3

What treatments would you recommend?

QUESTION 4

Is there a role for allergy testing, and if so, what testing would you recommend?

QUESTION 5

Is there a role for dietary manipulation?

QUESTION 6

When should a patient be referred for specialist advice?

CASE 3 ANSWERS

ANSWER 1

Lucia's likely diagnosis is atopic dermatitis with staphylococcal super-infection.

Atopic dermatitis is an increasingly common condition with an estimated prevalence of up to 20% in infants and young children in Australia.[19] It is characterised by a relapsing and remitting itchy rash commonly affecting the flexures.

Lucia could have impetigo (school scores), although this diagnosis is less likely because the rash has been present for several months and has only recently become crusty and weepy.

Impetigo is a common and highly contagious bacterial skin infection due to *Staphylococcus aureus* or *Streptococcus pyogenes* or both. It commonly occurs in preschool and school-aged children and frequently affects multiple family members.

ANSWER 2

Atopic dermatitis is a clinical diagnosis and, in general, no investigations are required. In the setting of overt skin infection, empirical therapy with an anti-staphylococcal antibiotic is appropriate. A skin swab for microscopy, culture and sensitivities could be considered – this is particularly useful for recurrent infective episodes to ensure the chosen antimicrobial therapy is appropriate.

FEEDBACK

Alternative scenario: At 8 months of age, Lucia was given a teaspoon of runny scrambled egg and within 10 minutes she developed a 'welty' rash around her mouth and on her trunk. This was followed by a large vomit. She has refused to have whole egg since, but does tolerate egg baked in cake.

This history is consistent with an IgE-mediated egg allergy and should be confirmed by allergy testing (see *Question 4*). In the event of an IgE-mediated food allergy, an appropriate food allergy or anaphylaxis management plan, with indications of when to use an adrenaline auto-injector, should be implemented (see *Resources*).

ANSWER 3

There are two components to treatment for Lucia.

1. Treat the infection – use an appropriate antibiotic such as cephalexin or flucloxacillin.
2. Treat atopic dermatitis by:
    a. cleansing – bath daily. A significant proportion of patients with moderate atopic dermatitis have a filaggrin gene defect that predisposes to a defective skin barrier with resultant increased skin colonisation with *Staphylococcus aureus*.[20] This necessitates vigilant daily bathing. Adding bleach (sodium hypochlorite 6%) to the bathwater (12 mL per 10 L of water) has been shown to be effective in reducing skin staphylococcal colonisation levels with resultant improved atopic dermatitis control[21]
    b. moisturising – regularly apply an unperfumed product. The frequency of application should be dictated by degree of dryness, but morning and night as a minimum
    c. treating inflammation – apply topical corticosteroid ointments to sites of active inflammation until clear. Potent topical corticosteroids (e.g. mometasone [Elocon™ or Novasone™] or methylprednisolone [Advantan Fatty Ointment™]) for atopic dermatitis on the body, and 1% hydrocortisone (Sigmacort™ or DermAid™) are recommended for the more sensitive skin on the face (between the brow and chin). It is important to address parental concerns about topical corticosteroid use and steroid phobia
    d. minimising irritants – avoid soaps, bubble bath, certain sunscreens, woollen garments and blankets
    e. providing a management plan – if possible, give a written atopic dermatitis management plan to parents or carers detailing the measures above (see *Resources*).

ANSWER 4

Allergy testing is not required for mild to moderate eczema that is well controlled by the measures outlined in *Answer 3*. Allergy testing may be considered where there is a clear history of an associated immediate IgE-mediated food allergy or where environmental allergens such as house dust mites, pets or pollens are believed to be contributing to eczema flares.

Allergy testing can be either by skin prick testing (generally only available in specialist practice) or by blood test for serum-specific IgE (formerly RAST or radioallergosorbent test). Screening allergy testing to food allergens is not recommended in the absence of a history of an immediate allergic reaction to that food.[22]

ANSWER 5

Lucia has a full and varied diet inclusive of the usual commonly allergenic foods without reaction. Dietary manipulation in this situation is unlikely to be of benefit and is not recommended. It could also pose a risk for nutritional inadequacy. Atopic dermatitis and food allergy are commonly associated, but food allergy does not cause atopic dermatitis. The risk of coexistent food allergy is increased in early onset (under 12 months of age) moderate to severe eczema.[22]

ANSWER 6

Any patient who has ongoing significant atopic dermatitis who fails to respond to appropriate topical therapies, or who has associated multiple food allergy should be referred for specialist advice.

CASE 4

JOEL'S BEHAVIOUR CONCERNS HIS PARENTS

Joel, a 4-year old boy, has been your patient since his birth. He presents with symptoms of a mild respiratory tract infection. He has come in today with his mother, Alison, although his father, Jake, also brings him to your surgery sometimes. Six months ago Joel's parents had another baby, who they named Carla.

After you examine and assess Jake, Alison appears reassured, but then confesses she is not coping with Joel's behaviour. She says she is frustrated and confused about how to manage his defiant behaviour and tantrums.

Joel was born at term by normal delivery after an uneventful pregnancy. His growth and development were appropriate through infancy and early childhood, although he was admitted for feeding and settling issues at a clinic for new mothers when he was a baby. His vaccinations are up-to-date, he has a history of viral infections and two emergency department presentations for croup at 2 years of age. His croup required oral prednisone but no admission.

You heard Joel in the waiting room before you called him and his mother in to see you.

Alison speaks to Joel in your presence in an irritable and negative fashion. Joel's behaviour reflects a difficult temperament, including characteristics of impulsivity and restlessness. He appears defiant and resists Alison's attempts to control him. He tries to open and explore the drawers of your desk and occasionally grabs the computer mouse from your desk. Alison's reaction is to shout at her son and grab his arm forcibly, which Joel ignores. Alison appears embarrassed by his behaviour and her inability to deal with it.

QUESTION 1

What safety aspects of this situation should you explore?

QUESTION 2

How would you assess this situation?

QUESTION 3

What sociocultural aspects need to be considered?

QUESTION 4

What advice could you give Alison on how to improve her communication with Joel, and what other resources could she access?

QUESTION 5

What advice could you give with respect to discipline issues?

QUESTION 6

What diagnoses and possible referrals would you consider for Joel?

CASE 4 ANSWERS

ANSWER 1

Safety assessment should always come first for all members of the family. For Joel, there could be a risk of physical abuse that may be happening out of sight. Punishments occurring at home, either from Alison or Jake, may be inappropriate. A physical examination may reveal bruising or signs of physical abuse.

Alison's behaviour and poor coping skills may reflect a perinatal mood disorder. Carla's safety is also an issue as Alison has confided in you that she is having trouble coping. It is important to sensitively but firmly explore safety issues for the entire family. This should include directly talking to and physically examining Joel, and also examining Carla at the next opportunity.

ANSWER 2

A great deal of your assessment has already taken place, by virtue of your position as their family doctor. You have knowledge of the family dynamics, which extends to family and friends. An Ecological Framework for Human Development, developed in the 1970s by Bronfrenbrenner, reflects this dynamic.[23]

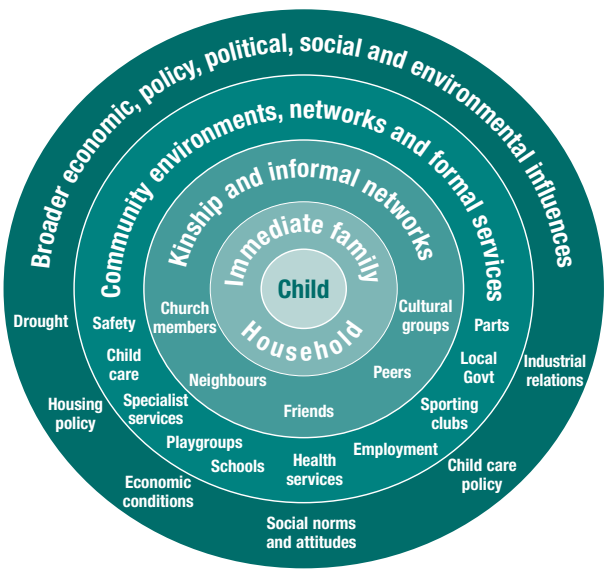

Figure 2. Reproduced with permission from Bronfenbrenner, U. (1977). Toward an experimental ecology of human development. American Psychologist, 32, 513–531[23]

Assuming safety has been established, assessment should follow a patient- and family-centred approach. Exploring the parents' views on behaviour, communication and discipline in a non-judgemental manner is the most likely way to explore the family dynamic. It will also make the patient (in this case the parent) feel they are being taken seriously, and this will develop a sense of ownership and improve parent compliance. You should ask Alison how she and Jake discipline Joel. Do they share the same views on discipline?

ANSWER 3

Sociocultural issues strongly influence attitudes to discipline of children, as the ecology model illustrates. Developing an understanding of these issues will help in our assessment, and also enable us to be a more effective guide. Exploring the parents' schema (how they view the world based on their own experiences) will help this. How were they brought up? Do the parents' attitudes to discipline match up? What are the parents' cultural backgrounds, and how do these influence their own beliefs about parenting?

ANSWER 4

Communication requires consideration of how people listen and respond to each other. A prerequisite for effective listening skills is taking what others say seriously, and treating what they say with respect even if they are small children. This is demonstrated by showing an interest in what children say, and exploring and expanding any leads they provide. Alison should be counselled that this 'teasing out' approach requires effort and patience, but will ultimately enhance Joel's confidence and self-image.[24]

A useful publication from the Raising Children Network website 'Positive ways to talk to children' (see *Resources*) could be provided to Alison. When Joel misbehaves encourage her to stop what she is doing for a moment, kneel down and make eye contact with him. She can acknowledge his feelings, for example, by asking: 'You look sad, can you tell me what happened?' Suggest to Alison that she repeats what Joel tells her and asks him more specific questions.

Emotional delivery can get in the way of understanding, so sticking to the facts, explaining why you are saying what you are saying and doing so without negative emotion will enhance communication. Negativity can manifest as threats, orders, sarcasm, accusations and name-calling.[24,25]

As well as reducing negativity, increasing positive language – such as recognising and praising desired behaviours – will help reduce what has been called a 'praise deficit' (see Brooks and Goldstein[24]).

ANSWER 5

Children learn by watching adults respond to their behaviour, both wanted and unwanted. Because a central driver of child behaviour is parental attention, paying attention to wanted behaviour and withholding attention from unwanted behaviour is the 'ace in our hand'. Convincing parents such as Alison of this can prove challenging, but will ultimately enhance their ability to appropriately discipline and, consequently, nurture their children.

For more extreme unwanted behaviour the use of consequences is effective. Consequences only work with children over the age of 3 years, and should only be used in response to a small fraction of the child's behaviour, regardless of the child's temperament (a small fraction means that the majority of unwanted behaviour is ignored, and only the more extreme behaviours receive a consequence).[25]

If consequences are overused, their effectiveness diminishes and relationships can be damaged.

There are three types of consequences and they are listed below.

1. **Natural consequences** where there is a natural flow from the misdemeanour to the consequence, for example, if a child refuses to have dinner they go to bed hungry. Natural consequences have a momentum of their own and, assuming they do not result in harm, can provide many valuable lessons.
2. **Related consequences** where there is some link between the unwanted behaviour and the result (this is also sometimes called a 'logical consequence'). For example, if a child makes a mess, she must clean it up, or if two children are fighting over a toy, the toy gets taken away for a while. Related (and natural) consequences allow a child to link the unwanted behaviour with the consequence.
3. **Losing a privilege** because of an unwanted behaviour. This can be a very powerful technique as it focuses the child's attention, but is more punitive than natural and related consequences and there is no logical link between behaviour and consequence. Parents should only withdraw a privilege with care as it can cause resentment, and the child should be warned beforehand that they are going to lose a particular privilege for a particular unwanted behaviour.

What doesn't work is screaming, constantly explaining, repeatedly warning, threatening, pleading, arguing, bribing, giving in and smacking.[25,26] Smacking undermines the parent's respect for their child (hence eroding their ability to teach), teaches the child that violence is a good solution to a problem, and denies the parent what they actually want – their child to learn from the experience – as the message is lost in the emotional mess.[25,26,27]

ANSWER 6

Joel's behaviour may be a result of his temperament, the parenting style used to date, or a combination of the two. However, sometimes a diagnosis may need to be considered such as conduct disorder, oppositional defiant disorder or ADHD. Further assessment and input by a paediatrician or a psychologist may be warranted, depending on local and family resources.

CASE 5

JADE IS VOMITING

Jade, a 4-year-old girl, is brought to see you by her mother Joanna. She has been vomiting since 3 am this morning. Jade is not interested in eating and is only sipping water. Joanna tells you that 'everything she drinks she brings straight up'. Jade has not opened her bowels and has no urinary symptoms. Joanna says that Jade 'was perfectly well yesterday'.

Jade does not appear to be her usual self, but is still able to return your smile and walk independently to be examined. You check for dehydration by pinching her cheeks and abdomen to ensure adequate elasticity and capillary refill. The physical examination is unremarkable and her growth charts show no signs of weight loss.

QUESTION 1

What is the most likely diagnosis?

QUESTION 2

What is the differential diagnosis?

QUESTION 3

Would your provisional diagnosis change if Jade was febrile?

QUESTION 4

What are the main concerns in diagnosing gastroenteritis in Jade at this stage?

QUESTION 5

How would you manage Jade?

FURTHER INFORMATION

Several days later, Joanna brings Jade back to see you. She is concerned that there is blood in Jade's stools, which are quite loose.

QUESTION 6

What is the most likely explanation for blood in Jade's stool?

QUESTION 7

Is there a place for medication?

QUESTION 8

Has the introduction of rotavirus vaccines had a significant public health benefit?

CASE 5 ANSWERS

ANSWER 1

The most likely diagnosis is viral gastroenteritis. However, it is important to remember that 'all that vomits is not gastro'. It is important to exclude other pathology.

ANSWER 2

Acute vomiting may be a symptom of:

- sepsis – e.g. septicaemia, pneumonia, UTI
- head injury – e.g. meningitis, raised intracranial pressure
- abdominal conditions – e.g. surgical obstruction, appendicitis, torsion of testes
- endocrine and metabolic disorders – e.g. ketoacidosis, Addison disease and inborn errors of metabolism
- medication – e.g. antibiotics, non-steroidal anti-inflammatory drugs (NSAIDS), laxatives
- poisoning
- envenomation
- cyclical vomiting syndrome.[28]

FEEDBACK

Experience suggests that children with gastroenteritis are perfectly well prior to the onset of vomiting. It is important to exclude ketoacidosis as this is misdiagnosed as gastroenteritis often enough to be of concern.[29] If a child is vomiting because of ketoacidosis there should be a history of deteriorating wellbeing over recent time. If that is the case, ask about polyuria, polydipsia, weight loss or recent onset of bedwetting.

Signs and symptoms of other illnesses may be non-specific in very young children and they are at risk of dehydrating quickly. Children younger than 6 months of age with acute onset of vomiting should initially be managed in hospital.

Admission is also indicated for children who are malnourished or who have depressed immunity.

ANSWER 3

The presence of fever does not change the provisional diagnosis. However, it would trigger a search for sepsis as well as ongoing monitoring.

FEEDBACK

Urine culture is indicated in younger, pre-verbal children with fever and vomiting even if they look well at presentation. Children less than 6 months of age with acute onset of vomiting should be managed in hospital initially, irrespective of fever or current wellbeing.

An Australian guideline[30] regards the trio of vomiting, diarrhoea and fever as characteristic of gastroenteritis. Experience in the community suggests that fever is not a universal feature. The authors of a UK guideline[31] did not find a cohort study that was conclusive in the clinical presentation of gastroenteritis in children.

ANSWER 4

Two principle concerns early in an episode of gastroenteritis are getting the diagnosis correct and excluding dehydration. There are no symptoms or signs of early dehydration. However, Jade's history suggests that her fluid balance is negative and she is at risk of developing detectable dehydration.

FEEDBACK

The National Institute for Health and Clinical Excellence (NICE) suggests determining where the patient lies on a continuum from no dehydration to detectable dehydration to clinical shock[31] rather than attempting to assess severity.

Table 5. Symptoms and signs of clinical dehydration

Symptoms of clinical dehydration
• Appears to be unwell or deteriorating • Altered responsiveness (e.g. irritable, lethargic) • Decreased urine output
Signs of clinical dehydration
• Altered responsiveness (e.g. irritable or lethargic) • Sunken eyes • Dry mucous membranes • Reduced skin turgor

Adapted with permission from the NICE guideline.[31] NICE includes tachycardia and tachypnoea under signs of clinical dehydration.

ANSWER 5

It is important that Joanna leaves your surgery both confident and competent to look after Jade and monitor her progress.

Her tasks are to:

- promote adequate hydration and nutrition
- ensure medical review if needed
- reduce the risk of others becoming infected.

Step 1.

It is crucial to empathise and engage Jade's mother as your partner in managing Jade's illness.

Step 2.

Explain the expected course of the illness and ask Joanna to return if Jade's progress varies from this course.

The expected course of gastroenteritis is:

0–6 hours	frequent vomiting, sometimes to dry retching but with no blood or bile in the vomitus
6–30 hours	vomiting becomes less frequent and mostly resolves completely in 3 days
6–30 hours	diarrhoea begins. Initially frequent, most resolves within 2 weeks

Be aware of the following:

- persistent and frequent vomiting beyond 6 hours. The risk of dehydration increases and so does the need to rethink the provisional diagnosis
- blood or bile in the vomitus
- a progressive unrelieved deterioration in the four As (alert, aware, active and appropriate) of social interaction
- episodes of unusual pallor need urgent review. Consider intussusception and sepsis
- reduced skin elasticity/reduced capillary return.

Checking for skin turgor over the mastoids eliminates the effect of subcutaneous fat.

Reduced capillary return sought centrally on the abdomen suggests clinical shock.[31]

Step 3.

Explain how to use fluids designed to reduce the risk of dehydration.

Breastfed infants should continue breast-feeding with frequent small feeds. They can be offered additional sips of oral rehydrating solution (ORS).

In the early period of frequent vomiting Jade should be offered 0.5mL per kg of ORS every 5 minutes.[30] This may be offered by teaspoon, cup or syringe. As small frequent feeds are tolerated, volumes can increase and frequency decrease.

The aim is to reduce vomiting and maximise water absorption from the upper intestine – water follows sodium – and sodium absorption is facilitated by glucose. ORS have an appropriate amount of electrolytes and glucose with an osmolality that does not promote diarrhoea. Hydralyte™ has slightly less sodium than other ORSs and being less salty may be more palatable, especially when frozen.[30]

A less desirable option, and only for a child with no clinically detectable dehydration, is dilute juice or lemonade diluted one part of juice or lemonade to four parts of water.[30]

According to the NICE clinical guideline there is 'great variation in the concentration of sodium and potassium and in the osmolality of readily available juices, soups and carbonated drinks'.[31]

FEEDBACK

The recommendations for preventing dehydration are based on expert consensus.[31]

Some parents withhold and some children refuse fluids for fear of causing further vomiting. It is important to explicitly address this fear if it is present.

Low calorie beverages are sweetened with sorbitol, which can cause osmotic diarrhoea – these are best avoided.

When a child's appetite returns, reintroduce solids. The NICE guideline recommends the reintroduction of palatable solids as early as possible. Some parents are reluctant to do this in case it promotes diarrhoea. Delay may result in calorie-protein malnutrition. The NICE guideline does not support diluting milk or the use of lactose-free fluids.[31]

Step 4.

Remind Joanna of the importance of hand washing, not sharing towels, avoiding school and childcare until 48 hours after last vomit or diarrhoea. Also avoid swimming pools until there has been no diarrhoea for 2 weeks.

ANSWER 6

Blood in diarrhoea is called dysentery. Blood in the stool suggests a bacterial cause for the gastroenteritis. *Campylobacter* and *Salmonella* are common pathogens causing childhood gastroenteritis.

FEEDBACK

The management of a bacterial gastroenteritis in the community is the same as for a viral gastroenteritis. However, it is worth culturing the stool in this situation as a positive culture alleviates concern about other causes of blood in the stool, e.g. inflammatory bowel disease (IBD). Four per cent of childhood IBD presents in children less than 5 years old.[32]

ANSWER 7

Until recently, best practice has been to avoid antibiotics, antiemetics and anti-diarrhoeal medications in childhood gastroenteritis. Antibiotics are not required for bacterial gastroenteritis that can be managed at home.

Studies are investigating the use of ondansetron as an antiemetic. While the evidence is not conclusive it may have some clinical benefit as a single dose when used by an experienced clinician.[30]

The roles of probiotics and other dietary supplements are currently being investigated.[31]

ANSWER 8

Rotavirus is the most common cause of severe gastroenteritis in children less than 5 years of age. From July 2007, two vaccines were included in the National Immunisation Program. By June 2010, there was a 71% reduction in rotavirus hospital admissions in children in this age group. The impact of vaccination in Indigenous children in hyperendemic areas was not as high.[34] It is anticipated that vaccination will significantly reduce hospital admissions for rotavirus gastroenteritis by 85–100%, as well as reducing any rotavirus gastroenteritis by up to 70%.[33]

CASE 6

EVIE HAS A FEVER

Evie, a 7-month-old baby, is brought into your surgery by her mother Laura.

Evie completed her third vaccination 3 weeks ago.

Laura reports that Evie had a high fever of 39°C overnight. She tells you there are no other associated symptoms, and no history of vomiting or diarrhoea.

On examination, Evie looks relatively well, does not have any tachypnoea, cough or increased work of breathing, has good peripheral perfusion and is well hydrated. Ear, nose, throat and respiratory examinations are clear. There is no abdominal tenderness and no skin changes present.

QUESTION 1

How would you manage Evie, and what advice would you give to Laura?

FURTHER INFORMATION

You suggest to Laura that Evie has a viral infection, and request that she brings her back for review the following day.

Evie is reviewed the following day. She is reported to be still miserable but active, and she has been taking reasonable amounts of fluids. Her temperature is 39°C, and the physical examination has not changed, with no new signs of a focal infection being present.

You order a FBC, CRP and urine microscopy and culture. The blood count identifies a WCC of 15 000, CRP of 50 and urine clean-catch specimen shows 2+ bacteria with white cells and red cells.

QUESTION 2

What is your management plan, and are any further investigations needed at this stage?

FURTHER INFORMATION

Evie was prescribed trimethoprim+sulfamethoxazole in two divided doses. On the following day, Laura reports that Evie appears a little better, but she is still having fevers. You advise that the antibiotics should be continued without change pending definitive culture results and antibiotic sensitivities. The next day Evie is well and afebrile, with the urine culture results identifying an *E. coli* organism with broad sensitivities, including to trimethoprim+sulfamethoxazole.

QUESTION 3

What immediate management is necessary?

QUESTION 4

Should Evie have follow-up management?

FURTHER INFORMATION

Evie is followed up with a renal ultrasound, which is completely normal.

QUESTION 5

What other investigations could be considered?

FURTHER INFORMATION

Evie responds well to antibiotics, but she presents 2 months later with symptoms suggestive of another urinary tract infection, which is confirmed on urine culture. Treatment is again effective, using amoxicillin for 10 days, and Laura enquires about the need for further investigation.

QUESTION 6

What would you advise Laura?

CASE 6 ANSWERS

ANSWER 1

Fever is the most common symptom among children attending for medical care. The majority of young children who present with a significant febrile illness (>38.5°C) have an infection, usually a self-limiting viral infection. It is important, however, that the possibility of occult or emerging bacterial infection be considered to prevent or detect potentially serious and life-threatening conditions early (e.g. UTI, pneumonia, meningitis, septicaemia, cellulitis and osteomyelitis).

The approach to a child who has a fever is greatly influenced by the following factors:[35,36,37]

- age of the child (0–3 months, 3–36 months, older than 36 months)
- immunisation status
- whether a focal source of infection can be identified on history or physical examination
- whether the child appears ill and toxic or appears well-looking.

Examples of a focal source of infection include otitis media, URTI and gastroenteritis. Toxic and unwell infants need hospital treatment, irrespective of whether a focal or specific cause of their infection is identified.

Even in the case of a relatively well-looking child with a fever, the possibility of occult bacterial infection, which may further develop into a serious and life-threatening infection, needs to be considered.[36]

Younger children are more likely to be bacteraemic when febrile than older children. The infant younger than 3 months of age is at significant risk, and should always be referred to a hospital for investigation and treatment if they present with fever, even if they appear well.

The risk is significantly lower when the child has completed their initial course of immunisations (the third of the initial immunisations is due at 6 months of age). This risk may be less than 1% in these children, but if the child is non-immunised or incompletely immunised, the risk of bacteraemia can be as high as 10%.

It is important in this group of patients to review their condition frequently, usually on a daily basis, to ensure they are not developing invasive bacterial disease.

When evaluating a child who presents with a fever, the doctor should look for symptoms and signs related to the respiratory, neurological, musculoskeletal and urological systems to make as accurate a diagnosis as possible on clinical grounds.

However, most importantly, the degree of illness must be assessed as this is the best indicator for risk of bacterial sepsis being present.[35,37]

ANSWER 2

No further investigations are needed at this stage for Evie.

Before making a definitive diagnosis of UTI, it is important that the urine taken for microscopy and culture is a reliable specimen. Collection of urine in the young infant can be very difficult. It is important to recognise the limitations of the various modes of collection and not make a definitive diagnosis of UTI based on inappropriately collected specimens. A clean-catch urine is currently the preferred method of collection in the outpatient or general practice setting.[38,39,40]

Bag urine specimens are not reliable and have a high incidence of false positive culture results. They can be used for screening purposes with urine dipstick testing or when a negative culture result is obtained although, in general, bag specimens should not be sent for culture.[38,39,40]

Dipstick tests positive for nitrites or leukocytes need to be confirmed with an appropriately collected clean-catch voiding urine, either collected by the pathology service or by the parent at home. It must be sent immediately for culture. Clean-catch specimens are easily obtained from children who are toilet-trained. Sick infants and children who require hospital care will have a catheter or suprapubic aspirate (bladder tap) urine specimen collected urgently and sent for microscopy and culture prior to commencing antibiotic therapy.

Evie's results were obtained from a clean-catch specimen. Hence the results were reliable and consistent with a diagnosis of UTI. Evie should be prescribed trimethoprim+sulfamethoxazole in two divided doses. Amoxicillin or a cephalosporin would also provide suitable cover for urinary pathogens.

FEEDBACK

Reviewing febrile children until they are better is most important as their condition may change, focal infection may emerge, or an unexpected diagnosis evolve.

Presenting symptoms can be nonspecific, and physical examination in the young child can be very difficult. Signs of focal disease can be easily missed or not be present, and if the situation is not clear, the GP may need to perform some simple investigations to aid management.

The most useful investigations that can help detect bacteraemia in a child are a FBC, CRP, urine microscopy and culture and, in some situations, a chest X-ray. A WCC of >15 000 leukocytes is highly suggestive of bacterial infection requiring antibiotic treatment. A properly collected urine culture is often the only way of determining whether a UTI is present. A chest X-ray may identify pneumonia in young children even when respiratory symptoms and signs are minimal.

ANSWER 3

The course of antibiotics must be completed. The course should be between 7 and 14 days and is most usually of 10 days duration. After completion of the antibiotic course, if the child is well, there is no need to repeat the urine culture to confirm eradication of infection.[38,39,40]

Some children do not respond to treatment as expected, despite being given an appropriate antibiotic to which the identified organism is sensitive. In this situation, a referral to a hospital emergency department or paediatric specialist should be made. In this setting, a renal ultrasound should be immediately included in the investigations because non-response to treatment may indicate the presence of an underlying renal abnormality, which may need urgent surgical intervention in addition to antibiotic treatment. The size and shape of the kidneys, renal pelvis dilatation (which may indicate obstruction), renal or peri-nephric abscess, pyelonephritis, ureteric abnormalities, or a dilated or abnormal bladder can be identified with ultrasound, and may help explain failure to respond to initial therapy.

ANSWER 4

An elective renal ultrasound is a worthwhile follow-up investigation of young children who have had their first UTI. It is a simple and non-invasive test used mainly to screen for renal abnormalities, particularly renal pelvis dilatation, ureteric dilatation and bladder abnormalities. Renal ultrasound is less likely to detect vesicoureteric reflux and renal scarring.

ANSWER 5

A micturating cystourethrogram is used to detect vesicoureteric reflux, but currently it is not recommended for all children as follow-up of a UTI because it is an invasive and expensive procedure involving significant amounts of radiation exposure.

The traditional view that vesicoureteric reflux causes renal scarring has changed. Its presence is of questionable significance in the development of progressive renal damage. This has altered the approach to follow-up investigation of children who have had a UTI.

Increasing grades of vesicoureteric reflux do increase the risk of the development of renal scars, but the benefits of detection and treatment of vesicoureteric reflux are not clear. The role of surgical intervention and the use of long-term prophylactic antibiotics are not proven in the prevention of renal scarring, even for high grades of vesicoureteric reflux. A micturating cystourethrogram is therefore no longer routinely performed. It is recommended only for children who have significant renal tract abnormalities identified on ultrasound or, possibly, in those who have recurrent UTIs.[38,39,40]

Children who have recurrent UTIs and normal renal ultrasounds probably do have at least low-grade vesicoureteric reflux; however, this can usually be managed without the need for documentation using a micturating cystourethrogram.

Other investigations can further evaluate the urinary tract. Of these, radio-isotope renal scintigraphy, DMSA, DTPA or MAG3 scans are specifically used to define kidney structure and function.

The DMSA isotope scan is used to identify renal structure and particularly identify renal scarring. Its role in the investigation of UTI remains controversial. It involves exposure to radiation, is expensive, and certainly plays no role in the treatment of those children who have had their first UTI and who have normal renal ultrasounds.

DTPA or MAG3 scintigraphy is used to examine renal function and also investigate for pelviureteric obstruction or vesicoureteric obstruction. It is only needed when an abnormality is identified on renal ultrasound, which may suggest obstruction. It can also help distinguish a poorly functioning dysplastic kidney from an obstructed kidney.

ANSWER 6

You advise Laura that even though it is likely that Evie has low-grade vesicoureteric reflux predisposing her to UTIs, further investigation is not required. As Evie's renal ultrasound is normal, there is no need to perform a micturating cystourethrogram. You should explain that the use of preventive antibiotics is not warranted as it does not prevent the development of renal scars, although there may be a role in prevention of further UTIs.

It is worth mentioning that should a further urinary infection occur, referral to a paediatrician or paediatric nephrologist would be indicated.

1. High PC, American Academy of Pediatrics Committee on Early Childhood, Adoption, and Dependent Care and Council on School Health. School Readiness. Pediatrics. 2008;121(4):1008–15.
2. von Hahn LE. Specific learning disabilities in children: Role of the primary care provider. Available at http://bit.ly/ZeuB9y [accessed on 7 March 2013].
3. Bravender T. School performance: the pediatrician's role. Clin Pediatr (Phila). 2008;47(6):535.
4. Phillips DM, Longlett SK, Mulrine C, Kruse J, Kewney R. School problems and the family physician. Am Fam Physician. 1999;59(10):2816.
5. Hagerman RJ. Pediatric assessment of the learning-disabled child. J Dev Behav Pediatr. 1984;5(5):274.
6. Mclnerny TK. Children who have difficulty in school: a primary pediatrician's approach. Pediatr Rev. 1995;16(9):325.
7. Glascoe FP. Parents' concerns about children's development: prescreening technique or screening test? Pediatrics. 1997;99(4):522.
8. Brothers KB, Glascoe FP, Robertshaw NS. PEDS: developmental milestones–an accurate brief tool for surveillance and screening. Clin Pediatr 2008;47(3):271.
9. Dworkin PH. Detection of behavioral, developmental, and psychosocial problems in pediatric primary care practice. Curr Opin Pediatr. 1993;5(5):531.
10. Johnson CP, Myers SM, American Academy of Pediatrics Council on Children with Disabilities. Identification and evaluation of children with autism spectrum disorders. Pediatrics. 2007;120(5):1183.
11. ADHD: Clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. Process of Care Supplemental Appendix. Available at: http://pediatrics.aappublications.org/content/128/5/1007/suppl/DC1 [accessed 7 March 2013].
12. Wolraich ML, Lambert W, Doffing MA, et al. Psychometric properties of the Vanderbilt ADHD diagnostic parent rating scale in a referred population. J Pediatr Psychol. 2003;28(8):559.
13. Perry DC, Bruce C. Evaluating the child who presents with an acute limp. BMJ 2010;341:c4250. PubMed PMID:20729271.
14. Royal Children's Hospital Melbourne. Clinical Practice Guidelines. Available at http://www.rch.org.au/clinicalguide/guideline_index/The_Acutely_Painful_Hip/ [Accessed 7/12/2012].
15. Levine MJ, McGuire KJ, McGowan KL, Flynn JM. Assessment of the test characteristics of C-reactive protein for septic arthritis in children. J Pediatr Orthop 2003 May-Jun;23(3):373–7. PubMed PMID: 12724603.
16. Kocher MS, Zurakowski D, Kasser JR. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. J Bone Joint Surg Am 1999 Dec;81(12):1662–70. PubMed PMID:10608376.
17. Kocher MS, Mandiga R, Zurakowski D, Barnewolt C, Kasser JR. Validation of a clinical prediction rule for the differentiation between septic arthritis and transient synovitis of the hip in children. J Bone Joint Surg Am 2004 Aug;86-A(8):1629–35. PubMed PMID:15292409.
18. Luhmann SJ, Jones A, Schootman M, et al. Differentiation between septic arthritis and transient synovitis of the hip in children with clinical prediction algorithms. J Bone Joint Surg Am 2004 May;86-A(5):956–62. PubMed PMID: 15118038.
19. Williams H, Stewart A, von Mutius, E, Cookson W, Anderson HR. Is eczema really on the increase worldwide? J Allergy Clin Immunol 2008;121:947–54, e915.
20. Cork MJ, Danby SG, Vasilopoulos Y, et al. Epidermal barrier dysfunction in atopic dermatitis. J Invest Dermatol 2009;129:1892–908.
21. Huang JT, Abrams M, Tlougan B, Rademaker A, Paller AS. Treatment of colonization in atopic dermatitis decreases disease severity. Pediatrics 2009;123(5):e808–e814.
22. Campbell DE. Role of food allergy in childhood atopic dermatitis. J Paediatr Child Health 2012;48(12):1058–64.
23. Bronfenbrenner, U. Toward an experimental ecology of human development. American Psychologist, 1977;32,513–531
24. Brooks R, Goldstein S. Raising resilient children. New York: McGraw-Hill, 2001,198–199
25. Turecki S, Wernick S. Normal children have problems too. New York: Bantam, 1994.
26. Siegel DJ, Hartzell M. Parenting from the inside out. New York: Penguin, 2003.
27. Committee of Psychosocial Aspects of Child and Family Health, American Academy of Pediatrics. Guidance for effective discipline. Pediatrics 1998(reaffirmed 2012);101;723-728.
28. Forbes D., Fairbrother S. Cyclic nausea and vomiting in childhood. Aust Fam Physician 2008;37:33–36.
29. Ali K, Harnden A., Edge J. A. Easily missed? Type 1 diabetes in children. BMJ 2011; 342: d294.
30. NSW HEALTH, Clinical practice guidelines. Infants and children: acute management of gastroenteritis. Third edition. November 2009 http://www.nslhd.health.nsw.gov.au/ppg/PD2010_009%20-%20Flowchart.pdf [accessed 7 March 2013].
31. The National Institute for Clinical Excellence (NICE). Diarrhoea and vomiting caused by gastroenteritis: diagnosis, assessment and management in children younger than 5 years. Clinical guideline. April 2009. http://www.nice.org.uk/nicemedia/live/11846/43817/43817.pdf [accessed 7 March 2013].
32. Sawczenko A, Sandhu BK, Logan RFA, et al. Prospective survey of childhood inflammatory bowel disease in the British Isles. Lancet 2001 (357):1093–4.
33. NCIRS Fact Sheet: November 2009. Rotavirus Vaccines for Australian Children: Information for Immunisation Providers. Available at www.ncirs.edu.au/immunisation/fact-sheets/rotavirus-fact-sheet.pdf [accessed 7 March].
34. Dey A, Wang H, Menzies R, Macartney K. Changes in hospitalisations for acute gastroenteritis in Australia after the national rotavirus vaccination program. MJA 2012;197(8):453–7.
35. Hewson PH, Humphries SM, Roberton DM, McNamara JM, Robinson MJ. Markers of serious illness in infants under 6 months old presenting to a children's hospital. Arch Dis Child 1990;Jul;65(7):750–6.
36. Haddon RA, Barnett PL, Grimwood K, Hogg GG. Bacteraemia in febrile children presenting to a paediatric emergency department. Med J Aust 1999;170(10):475–8.
37. Van den Bruel A, Thompson M, Buntinx F, Mant D. Clinicians' gut feeling about serious infections in children: observational study. BMJ 2012;345:e6144. doi: 10.1136/bmj.e6144.
38. NICE (National Institute for Health and Clinical Excellence) clinical guidelines. Urinary tract infection in children: Diagnosis, treatment and long-term management. Issued: August 2007.
39. Larcombe J. Urinary tract infection in children. Clin Evid (online). 2010Feb 9;2010.pii:0306.
40. Roberts KB; Subcommittee on Urinary Tract Infection; Steering Committee on Quality Improvement and Management. Urinary tract infection: clinical practice guideline for the diagnosis and management of the initial UTI in febrile infants and children 2 to 24 months. Pediatrics 2011;128(3):595–610.doi:10.1542/peds.2011-1330. Epub 2011 Aug 28.

RESOURCES FOR DOCTORS

- The Parents Evaluation of Developmental Status Developmental Milestones (PEDS and PEDS-DM) are two highly accurate, valid tools providing developmental screening and behavioural screening, as well as ongoing surveillance. The PEDS-DM takes 6–7 minutes to complete and can be purchased at www.pedstest.com.
- The Pediatric Symptom Check List detects 85–90% of children with behaviour problems and is available at www.brightfutures.org. It is endorsed by the American Academy of Pediatrics.
- ADHD may be screened using the Conners Questionnaires or Vanderbilt Assessment Scales available at the National Initiative for Children's Healthcare Quality. Visit www.nichq.org/adhd.html.
- See management plans and indications for an adrenalin auto-injector at the Australian Society of Clinical Immunology and Allergy. Visit www.allergy.org.au.
- The Royal Children's Hospital's clinical practice guideline for atopic dermatitis is available at www.rch.org.au/rchcpg/hospital_clinical_guideline_index/Eczema_management/.
- The Autism Research Centre offers screening tools for individuals of different ages. Visit www.autismresearchcentre.com/arc_tests for more information.
- A publication of The Committee of Psychosocial Aspects of Child and Family Health at the American Academy of Pediatrics called *Guidance for effective discipline* is available from http://bit.ly/1674rw4.
- A good article to help parents develop discipline techniques is 'A brief primary care intervention helps parents develop plans to discipline' by Scholer SJ, Hudnut-Beumler J, Dietrich MS. in *Pediatrics* 2010;125;e242-249.
- *Awakening children's minds: how parents and teachers can make a difference* by Laura Berk, published by Oxford University Press in 2001, examines the importance of communication between children and adults.
- For research on the effect of communication on the devleoping brain read *Parenting from the inside out*, published by Penguin in 2003, by Daniel Siegel and Mary Hartzell.
- The NSW Department of Health have published clinical guidelines for the management of acute gastroenteritis at http://www.nslhd.health.nsw.gov.au/ppg/PD2010_009%20-%20Flowchart.pdf.
- NICE have published guidelines for the management of diarrhoea and vomiting caused by gastroenteritis at http://www.nice.org.uk/nicemedia/live/11846/43817/43817.pdf.
- The National Centre for Immunisation and Research Surveillance has a useful factsheet on rotavirus vaccination for immunisation providers at www.ncirs.edu.au/immunisation/fact-sheets/rotavirus-fact-sheet.pdf.

RESOURCES FOR PATIENTS

- A useful patient handout on normal development in children can be found in *Murtagh's Patient Education*, 5th edition on page 45.
- The Parents Evaluation of Developmental Status Developmental Milestones (PEDS-DM) takes 6–7 minutes to complete. The PEDS-DM can be purchased at www.pedstest.com.
- The Raising Children Network provides information that can help parents with the day-to-day decisions of raising children. This information is available at www.raisingchildren.net.au.
- The American Academy of Paediatricians hosts a comprehensive online resource for parents at www.healthychildren.org.
- *How to talk so kids will listen and listen so kids will talk* by Adele Faber and Elaine Mazlish, published by Piccadilly Press in 1999, explores communicating with children effectively.
- *Raising resilient children: fostering strength, hope and optimism in your child* by Robert Brooks and Sam Goldstein, published by McGraw-Hill in 2002, looks at how to raise resilient children.
- *Normal children have problems too* by Stanley Turecki and Sarah Wernick, published by Bantam in 1994, offers insights into the social difficulties children can have and suggests ways to find solutions.

Paediatrics

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and
- log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au.

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.
FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

QUESTION 1

Tim, a 12-month-old boy, presents with a temperature of 38.5ºC. He is otherwise well. Urinary dipstick is positive for nitrites and you suggest a clean catch specimen of urine. This confirms a diagnosis of a UTI. Six months later Tim returns with another UTI, and 3 months later he returns with a third UTI. What is the most appropriate initial test to exclude renal abnormalities?

A. Abdominal X-ray
B. DMSA isotope scan
C. MAG3 scintigraphy
D. Micturating cystourethrogram
E. Renal ultrasound.

QUESTION 2

Bo, a 4-year-old boy, has just started preschool. His mother brings him in to see you because he started limping yesterday and says it hurts to walk. Bo had a cold 2 weeks ago, but has otherwise been well. On examination he has slightly decreased range of movement of his left hip. What feature in Bo's history would be of most concern to you?

A. Sudden onset of hip pain
B. Painful range of hip movement
C. History of recent viral URTI
D. Fever of 38ºC
E. Walking with a limp.

QUESTION 3

Violet, a 4-year-old girl, is brought in by her mother Selina who is struggling with her behaviour. Violet has a younger 2-year-old brother. She often has tantrums where she falls to the floor and screams. She is wilful and defiant to her parents' requests and often has meltdowns in public places, such as the supermarket. Selina is embarrassed to visit her friends because Violet is difficult to control, noisy and loves to open cupboards and make a mess. Selina is frustrated and close to tears. What would your priority be when assessing Violet?

A. Checking for developmental delay
B. Assessing child and mother safety
C. Exploring parenting style
D. Checking for underlying illness
E. Assessing cultural background.

QUESTION 4

You examine Violet (from *Question 3*) and find no evidence of child abuse, developmental delay or underlying illness. What is the most useful intervention in dealing with unwanted behaviour in this age group?

A. Constantly explaining
B. Repeatedly warning
C. Smacking
D. Pleading
E. Improving communication and using consequences.

QUESTION 5

Moshe, a 9-month-old baby boy, is brought in to see you by his parents Ester and Joshua. They have noticed an intermittent dry rash on his cheeks and his abdomen over several months. Moshe tends to scratch the rash, which then becomes worse. Ester has a history of hay fever and Joshua had asthma as a child. You make a diagnosis of atopic dermatitis. What treatment options do you suggest to Ester and Joshua?

A. Cleanse and bathe daily.
B. Apply non perfumed moisturiser.
C. Minimise irritants and avoid soaps, bubble bath, woollen garments, blankets and certain sunscreens.
D. Apply topical cortisone cream.
E. All of the above.

QUESTION 6

Jet, a 5-year-old boy, presents with vomiting over the past 6 hours. His mother tells you Jet hasn't been his usual self over the past few days, and has not wanted to go to school. He has been drinking

more than usual and his appetite has decreased. Jet wet the bed last night and his breath smells. He looks pale and unwell, and his temperature is 37ºC. What is the most likely diagnosis?

A. Diabetic ketoacidosis
B. Viral gastroenteritis
C. School refusal
D. Cyclical vomiting syndrome
E. Food poisoning.

QUESTION 7

Jamila, a 2-year-old girl, presents with a 24-hour history of vomiting and diarrohea. Her temperature is 37.5ºC. Jamila is able to drink small sips of fluids only. You are concerned that she is becoming dehydrated. How would you assess Jamila's level of dehydration?

A. Assess skin elasticity at the mastoid.
B. Check capillary refill in the abdomen.
C. Check for altered responsiveness.
D. Check for loss of weight.
E. All of the above.

QUESTION 8

Ivy, a 3-month-old baby, is brought into the surgery by her mother Jane. She is breastfed and has not had her 2-month immunisations. Jane tells you Ivy was difficult to settle last night. On examination Ivy looks flushed, lethargic and unwell, and has a temperature of 39ºC. There appear to be no focal signs of infection. What is the most appropriate management for a baby of Ivy's age?

A. Regular paracetamol
B. Tepid sponging
C. Review the following day
D. Refer to hospital
E. Reassure Jane that this is normal.

QUESTION 9

Harry, a 3-year-old boy, is brought in by his mother May who is concerned by his behaviour. Harry had a normal vaginal delivery and was breastfed for 9 months. He crawled at 6 months and commenced walking at 14 months. Harry was slow to speak and does not have a large vocabulary now. May has noticed he does not always respond to his name and tends to avoid eye contact. She is frustrated by Harry's apparent inability to follow simple commands. Which of the following is the most likely diagnosis?

A. ADHD
B. Absence epilepsy
C. Fragile X syndrome
D. Autism spectrum disorder
E. Foetal alcohol syndrome.

QUESTION 10

Jesse, a 14-year-old boy, limps into the surgery. He is finding it difficult to walk and hobbles slowly to the examination couch. Jesse complains of pain in his right hip and some discomfort in his right knee. On examination Jesse is 167 cm tall and weighs 75 kg. He is afebrile. His right hip is shortened and he has decreased right hip movement on internal rotation. What is the most likely diagnosis?

A. Osteomyelitis
B. Transient synovitis (irritable hip)
C. Perthes disease
D. SUFE
E. Stress fracture of the hip.

check

Independent learning program for GPs

Unit 494 May 2013

Dementia

RACGP

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check

Independent learning program for GPs

Dementia

Unit 494 May 2013

The five domains of general practice
Communication skills and the patient–doctor relationship
Applied professional knowledge and skills
Population health and the context of general practice
Professional and ethical role
Organisational and legal dimensions

RACGP

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UNDERSTAND ALZHEIMER'S
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A diagnosis of dementia can be devastating for the patient and their family. Dementia is the second most feared disease, behind cancer. We fear no longer being in control of our mind. As well as this fear there is, unfortunately, a stigma attached to the diagnosis of dementia. While many people consider it a natural part of ageing, it is in fact a chronic disease process.

An early diagnosis of dementia has many benefits. It can potentially delay progress of the disease and treat reversible causes. Support systems can be put into place early, and planning can be organised before the patient's competency is compromised.

Dementia is a progressive disease that requires a multidisciplinary approach. It involves not only the patient but their family and carers as well. Comorbidities such as hypertension and diabetes need to be managed, and the health of carers needs to be monitored. The family GP is ideally placed to provide this support.

Alzheimer's Australia coordinated the original manuscripts, and further information on early intervention for people with dementia can be found at www.detectearly.org.au.

We would like to thank the following authors, contributors and reviewer for their valuable input.

Allan Shell MBBS, Grad Dip PH, general practitioner and visiting fellow at the Dementia Collaborative Research Centre, School of Psychiatry, University of New South Wales. He has a particular interest in continuing professional development and education for GPs and the wider community.

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We would also like to thank Henry Brodaty, Diana Fayle, Carmelle Peisah and Lee-Fay Low for their valuable contributions to this issue of *check*.

The reviewer for this unit was Hadia Haikal-Mukhtar Bsc (Hons), MB BS (Melb), FRACGP, Dip Ger. Med, LLB (Hons) (Melb), Grad Cert Health Prof Ed (Monash), general practitioner, Fellow of the RACGP and head of Auburn Clinical School, University of Notre Dame Australia.

The learning objectives of this unit are to:

- display increased knowledge of the different types of dementias and their presentations
- understand the importance of treating comorbidities in patients diagnosed with dementia to improve health outcomes and patient wellbeing
- become familiar with cognitive testing tools that can be used in culturally and linguistically diverse (CALD) populations
- identify the difference between delirium and dementia
- display an increased understanding of the testamentary issues involved with dementia
- advise patients of protective lifestyle factors such as exercise and mental stimulation, to decrease the rate of disease progression.

We hope you find this edition of *check* useful in the diagnosis and management of dementia.

Kind regards

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GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

ACAT	Aged Care Assessment Team
ACD	advanced care directive
AD	Alzheimer disease
APOE ε4	apolipoprotein E epsilon
AMTS	Abbreviated Mental Test Score
BADL	basic activities of daily living
BP	blood pressure
BPSD	behavioural and psychological symptoms of dementia
bvFTD	behavioural variant frontotemporal dementia
CALD	culturally and linguistically diverse
CASS	Chinese Australian Services Society
CT	computerised tomography
CXR	chest X-ray
DBMAS	Dementia Behaviour Management Advisory Service
DSM IV	Diagnostic and Statistical Manual of Mental Disorders IV
ECG	electrocardiogram
eGFR	estimated glomerular filtration rate
EUC	urea and creatinine
FBE	full blood count
FDG-PET	fluorodeoxyglucose positron emission tomography
FTD	frontotemporal dementia
FTLD	frontotemporal lobar degeneration
GPCOG	General Practitioner assessment of Cognition
KICA	Kimberley Indigenous Cognitive Assessment tool
LBD	Lewy body dementia
LFT	liver function test
MCI	mild cognitive impairment
MMSE	Mini-Mental State Examination
MRI	magnetic resonance imaging
MSU	midstream urine
NDD	neurodegenerative disease
PBS	Pharmaceutical Benefits Scheme
PET	positron emission tomography
RBG	random blood glucose
REM	rapid eye movement
RSBD	REM sleep behaviour disorder
RUDAS	Rowland Universal Dementia Assessment Scale
SPECT	single photon emission computed tomography
TC	testamentary capacity
TFT	thyroid function test
UTI	urinary tract infection
VaD	vascular dementia
WACHA	Western Australian Centre for Health and Ageing

CASE 1

DAISY IS FORGETTING HOW TO COOK HER FAVOURITE MEALS

Daisy, aged 78 years, migrated from Hong Kong 10 years ago with her husband Wu under Family Reunion status to be with their two children in Sydney. They both live with their married daughter Ling in her home, and speak Chinese and limited English.

Daisy assists Ling, who runs a small business, by doing the household chores, shopping and looking after her three teenage grandchildren. Daisy's son lives in another part of town. He is not always around, but he and his family keep in touch regularly.

Ling brings her mother to see you for regular check-ups because Daisy has moderate hypertension and has recently developed type 2 diabetes. You manage both these conditions with oral medication. Daisy also takes ginseng tea, Ginkgo biloba and a Chinese medicinal tea for general wellbeing.

Daisy occasionally attends a social club to meet with other Chinese friends for lunch, but she is not otherwise physically active.

Recently, Ling has noticed that Daisy is more anxious, is making mistakes when she cooks her favourite meals and is becoming more forgetful, particularly when doing more than one preparation task for dinner. Ling is concerned about her mother's memory and has brought her to see you with a specific request to review this.

QUESTION 1

What are the key features in your history and examination of Daisy?

QUESTION 2

What is the most likely diagnosis?

QUESTION 3

What criteria need to be satisfied in order to make a diagnosis of dementia?

FURTHER INFORMATION

On examination, Daisy's weight is stable and her neurological examination is normal. Her blood pressure (BP) is 130/70 mmHg, her pulse rate is 72 beats per minute and her random blood glucose (RBG) is 5.5 mmol/L.

You use the online Chinese version of the General Practitioner assessment of Cognition (GPCOG), with the assistance of Ling. Daisy scores 5/9. This assessment only takes a few minutes to complete.[1]

You request that Daisy returns a week later with Ling and Wu to discuss the results of investigations you order today. You ask Daisy to complete a Geriatric Depression Scale form[2] before her next visit to exclude depression.

Investigations show haemoglobin of 13.2 g/L with a normal full blood count (FBE). Daisy's urea is 6.1 mmol/L (3–8.00 mmol/L) and her estimated glomerular filtration rate (eGFR) is 65 (>60 mL/min/1.72 m^2). All other electrolytes are normal. There is no evidence of a urinary tract infection (UTI) and Daisy has a normal chest X-ray (CXR) and a normal electrocardiogram (ECG).

QUESTION 4

With Daisy's permission, what further office-based assessment tool could you use to evaluate her memory in the presence of Wu and Ling?

FURTHER INFORMATION

You explain that Daisy has some form of memory loss or mild cognitive impairment, or even early dementia, and that you feel a computerised tomography (CT) scan of Daisy's brain would be useful. You advise Daisy's family that she may need to be referred to a memory clinic.

Ling is relieved by this explanation, but Wu is initially adamant that there is nothing wrong with his wife. He doesn't think Daisy needs any further testing and he says he will watch over her when she cooks. You explain that further testing may help Daisy and referral to a memory clinic could assist with providing medication, support and advice for Daisy and her family.

QUESTION 5

What factors can affect the diagnosis of dementia?

FURTHER INFORMATION

Wu is reassured by your explanation and has agreed to Daisy having a CT scan of her brain, as well as being referred to a memory clinic.

The CT scan shows generalised global atrophy with small lacunar infarcts consistent with vascular dementia (VaD).

QUESTION 6

What are your expectations of Daisy's memory clinic visit?

QUESTION 7

What else can be done to improve Daisy's health?

CASE 1 ANSWERS

ANSWER 1

It is important to take a complete medical and family history.

Specifically ask about forgetfulness, orientation, problem solving, coping with everyday life, mood, alcohol consumption, depression and new physical symptoms.

Ask about a time frame. When were the changes first noticed? Were they sudden or gradual? Are the difficulties getting worse?[3]

With Daisy's consent, ask other family members what they have noticed. Ask them to complete the 'informants questionnaire' from the General Practitioner assessment of Cognition (GPCOG).[1] It is a useful resource for GPs that is available in several languages, including Chinese.

Conduct a general and neurological examination.

ANSWER 2

The most likely diagnosis is mild cognitive impairment or early dementia requiring further investigation.

It is also important to consider that Daisy may not have dementia. It is important to rule out renal failure, chronic low-grade infection (e.g. UTI), depression, drugs, traditional Chinese medicines, cerebrovascular disease and other cerebral causes (e.g. tumour).

ANSWER 3

For a diagnosis of dementia there must be multiple cognitive deficits that commonly, but not always, involve memory loss. In some forms of dementia, memory may be preserved in the early stages.

- As well as having some form of cognitive impairment, there must also be impairment in one of the following domains.[4]
  - **Aphasia**: problems with language. This is often heard during the history more than elicited directly as a sign (e.g. being able to identify a watch but being unable to describe what it does, such as tell the time).
  - **Agnosia**: failure to recognise what objects are used for. Failure to recognise family members or well-known friends is a late feature of agnosia. This is not so much struggling to remember a name as it is struggling to recall that the person is known at all.
  - **Apraxia**: inability to carry out purposeful movements even though there is no sensory or motor impairment. This can present as difficulty with dressing in which there is a loss of detail.
  - **Executive dysfunction**: impaired planning, sequential organisation and attention. This may present as poor initiation, difficulty with problem solving and a reluctance to change routines.

Cognitive deficits must be severe enough to interfere with occupational and/or social functioning. They must also represent a decline from a previously higher level of function, as well as not occurring exclusively during the course of delirium.[4]

There needs to be a variation from the normal 'score' in one of the following standard assessment tools, all of which have Chinese translations available.

- Mini-Mental State Examination (MMSE)[5]
- Rowland Universal Dementia Assessment Scale (RUDAS)[5,6]
- GPCOG[1]

In this particular case, RUDAS would be the tool of choice because it is particularly appropriate for patients who are from CALD backgrounds (see *Resources*).

Investigations to include as part of a dementia screen are: FBE, electrolytes, urea and creatinine (EUC), liver function tests (LFTs), calcium, RBG, thyroid function tests (TFTs), B12 and folate levels, midstream urine (MSU), ECG, CT brain and CXR.[4]

ANSWER 4

A significant proportion of elderly people in Australia are born overseas with English as their second language. Standard assessment tools, such as the MMSE, are not easy to translate and administer to this demographic. Culturally appropriate dementia assessment tools are now available and include RUDAS and the Kimberley Indigenous Cognitive Assessment tool (KICA) for the rural and remote Indigenous population.[7,8] Daisy's RUDAS score is 23/30.

ANSWER 5

There are several barriers to the diagnosis of dementia, which include differentiating between normal ageing and dementia, a perceived lack of need to make a specific diagnosis and the lack of perceived treatment options. There can be a negative stigma attached to a diagnosis of dementia and the patient may have impaired ability, which can hinder an accurate history and participation in self care. There may also be denial by the patient or their family.[3]

There are many benefits to making an early diagnosis of dementia. These include treating any reversible causes and modifying risk factors. Education and safety issues can be addressed, and legal planning can be undertaken while the patient is still competent. The diagnosis is often a relief to carers. They can be supported in their role and their own health needs can be addressed by their GP.

ANSWER 6

Your expectation of Daisy's memory clinic visit should be to confirm the diagnosis of dementia, and to provide an overall management plan for Daisy. This would facilitate her staying at home for as long as possible.

The memory clinic can reassess Daisy's memory at a later stage, as well as referring her to a local geriatrician.

Daisy's family should be provided with education about dementia and ongoing support in caring for her.

ANSWER 7

Daisy's diabetes and hypertension need to be controlled and monitored regularly.

You can encourage Daisy to increase her exercise by walking or doing resistance exercise. Regular resistance exercise may help reduce her anxiety and improve her overall wellbeing. It has the added benefit that it can be done with her husband Wu.[9,10]

You can also discuss support services such as Alzheimer's Australia, (which offers resources for families and carers of culturally diverse backgrounds), the Chinese Australian Services Society (CASS) and the Dementia Behaviour Management Advisory Service (DBMAS) (see *Resources*).

An assessment from the Aged Care Assessment Team (ACAT) may also be helpful.

CASE 2

HARRY HAS BECOME MORE FORGETFUL

Harry, aged 75 years, is brought to see you by his wife Doris, who reports a 6-month history of forgetfulness. This includes Harry leaving taps running in the bathroom and losing his wallet and keys. Harry now has difficulty balancing his chequebook and has lost interest in watching sport on television. Harry is an irregular attendee at your practice. He has a history of hypertension and tends to be noncompliant with his medication. Doris has to consistently remind him to take his medication, which he resists at times.

On examination Harry's BP is 170/90 mmHg. After a thorough assessment you perform an MMSE. Harry's MMSE score is 21/30. You make a provisional diagnosis of dementia.

QUESTION 1

What are the main causes of cognitive decline in the elderly?

QUESTION 2

When would you refer patients with suspected dementia to a specialist or memory clinic?

FURTHER INFORMATION

You recommend that Harry be seen by an appropriate specialist or attends a memory clinic. He is assessed and a diagnosis of mixed Alzheimer disease (AD) and VaD is made. Harry is given a prescription for an acetyl cholinesterase inhibitor.

QUESTION 3

What drugs are available for the treatment of AD?

QUESTION 4

What are the indications for prescribing an acetyl cholinesterase inhibitor? What are the contraindications and the possible side effects?

FURTHER INFORMATION

Doris asks what she can do in terms of slowing the progression of Harry's dementia, and asks specifically about activities to keep Harry healthy. She also asks about the expected progression of the disease, and is concerned about what will happen if and when Harry deteriorates and needs more care than she can offer.

QUESTION 5

What are the key features in the management of Harry's dementia?

QUESTION 6

What is the role of exercise and recreational pursuits in dementia care, and what activities would you recommend for Harry?

CASE 2 ANSWERS

ANSWER 1

The main causes of cognitive decline in the elderly include the four Ds – delirium, depression, dementia and drugs. It can also be caused by normal ageing as well as mild cognitive impairment (MCI).[11,12]

ANSWER 2

Referral to a specialist is indicated if you are unsure of the diagnosis, or if the patient is relatively young. You would refer if multiple complex comorbidities were present and if the presentation was atypical. A psychotic episode or severe behavioural disturbance would also be indications for referral.

ANSWER 3

There are currently four drugs approved in Australia for the treatment of AD to slow cognitive decline. Donepezil (Aricept®), rivastigmine (Exelon®) and galantamine (e.g. Reminyl®) are cholinesterase inhibitors that make more of the neurotransmitter acetylcholine available at brain synapses and help to enhance memory function. Memantine (e.g. Ebixa®) acts on the neurotransmitter glutamate and can reduce the rate of symptom progression in the middle and later stages of AD.[3] A diagnosis of AD must be confirmed or made in consultation with a specialist for the first 6 months of treatment with a cholinesterase inhibitor[12,13] and/or memantine. These drugs can reduce the rate of decline of cognitive function for a period of time, but the effect tends to wear off.

ANSWER 4

Cholinesterase inhibitors such as donepezil, rivastigmine and galantamine can be prescribed on an authority prescription under the Pharmaceutical Benefits Scheme (PBS). A diagnosis of AD needs to be made and confirmed by a specialist and cognitive testing results with the MMSE must be greater than 10/30. Memantine has similar restrictions, but the MMSE needs to be between 10–14/30. Memantine can also be used with cholinesterase inhibitors, but is only subsidised as monotherapy. However, GPs can prescribe these drugs with no prior testing if the patient wants to pay for the nonsubsidised medication.[13] For patients with a diagnosis of AD, the restrictions and re-assessment process after the initial 6 months of treatment have been removed, as of 1 May 2013, for the use of those medications subsidised by the PBS.

The relative contraindications to cholinesterase inhibitors include bradyarrhythmias, active peptic ulcer disease and severe asthma.

Common side effects of cholinesterase inhibitors include gastro-intestinal disturbances such as anorexia, nausea, diarrhoea and vomiting. Other side effects include the urge to defaecate or urinate, sleep disturbances, nightmares and muscle cramps. There is also an increased risk of epileptic fits.

ANSWER 5

While there is currently no cure for dementia[3] there is much that can be done to maintain Harry's health and wellbeing, as well as delay his cognitive decline. It is important to reduce his cardiovascular risk factors, in particular his hypertension,[14,15] as well as managing any comorbidities such as hyperlipidaemia and diabetes. In addition to the medication he has been prescribed to delay cognitive decline, it is important to promote exercise, socialisation and some form of cognitive stimulation.

Preventative management includes immunisation and ensuring adequate hygiene, rest/sleep, hydration, nutrition and dental care.

Psychosocial treatments and support such as attention to safety, activities of daily living, mental activity and stimulation, and medication supervision are important.

Carer education and support are essential. This may include a referral to a support organisation such as Alzheimer's Australia that can offer resources for Harry and his family.[16] As his functional impairment increases, Doris may need to be directed to support services in the community to help with his care.

Harry will need to be assessed for his fitness to drive and legal issues will need to be raised while he is still competent. These include having an up-to-date will, an enduring power of attorney (for financial matters) and appointing an enduring guardian for health decisions, as well as completing advanced care directives (s).[17]

It is also important to consider Doris' health, because the stress and added burden of care at an older age can affect her overall health and mental wellbeing.

ANSWER 6

Exercise has a positive effect on memory and health.[9,10,18] Consider sports interests and activities that were part of Harry's premorbid lifestyle (e.g. Harry was a keen lawn bowler and gave it up because he could not remember the score or the names of visiting bowlers). Encourage Harry to resume this activity with another member who can act as his 'minder' by keeping the score and knowing the names of the other players.

You may like to suggest that Harry and Doris join a local community exercise group. This may include activities such as hydrotherapy, walking and strength training.

Encourage Harry to be physically active, and encourage ongoing mental stimulation (e.g. choirs, art classes or joining a men's shed group). You may also suggest that Harry and Doris join a seniors' group for ongoing social support, group activities and outings.

CASE 3

ANNE IS GETTING MORE AGGRESSIVE

Anne, aged 80 years, presents at your surgery with her daughter Jane, who is worried that her mother is becoming more aggressive. Jane tells you that Anne is becoming irritable and shouts at her and her children for no reason. Anne is also wandering around at night and waking the family.

Anne was diagnosed with AD 3 years ago. Until then, she had lived in her own apartment, even after the death of her husband Bruce. After her diagnosis she moved into a purpose-built 'granny flat' attached to Jane's home.

Jane is married and has two teenage children. Jane brought Anne to the initial consultation with you 3 years ago, as she had been concerned about her becoming more forgetful. Jane has attended all subsequent consultations with Anne, both at your practice and at the memory clinic.

When Anne was diagnosed with AD, Jane expressed relief – 'Finally we have an answer. I knew something was wrong with mum.'

At the time of diagnosis, Jane discussed with you her plan to care for Anne for as long as possible.

Jane asked you how AD progresses and what features might present at a later stage, as well as asking about a time frame for the development of these features.

QUESTION 1

What were you able to tell Anne and Jane about the symptom progression in AD?

QUESTION 2

What were the safety features that Jane needed to consider in terms of the physical environment inside the home for Anne?

FURTHER INFORMATION

Jane is aware that her role as Anne's carer will, at various stages, include many different responsibilities. She knows she will be responsible for managing medications, therapies and medical emergencies. She will also provide supervision and emotional support, and assist with personal care, mobility and household tasks.

At this consultation with you, Jane said she is concerned that she may not be coping as her mother's condition progresses.

QUESTION 3

What are the issues that may affect carers as a result of their role?

QUESTION 4

What issues do carers generally feel they would like GPs to manage?

QUESTION 5

What tool can be used to assess the caregiver burden?

QUESTION 6

What term is used collectively to describe irritability, aggression and wandering as they present in dementia? List other features that may also occur.

QUESTION 7

What are the differential diagnoses of behavioural and psychological symptoms of dementia (BPSD)?

QUESTION 8

What advice would you give Jane to help her deal with Anne's aggressive incidents?

CASE 3 ANSWERS

ANSWER 1

It was important to address Anne as well as Jane when answering questions. You told Jane and Anne that the symptoms and time frame of AD are variable. You may have used *Figure 1* to explain the general trends of AD using scores on the MMSE.

The basic activities of daily living (BADL) are bathing, continence, dressing, eating, toileting and transferring (walking). The degree to which the person with AD loses control over any two or more of these activities will affect the level of care required by that person, and the possibility of obtaining subsidised community 'packages' of care for them.

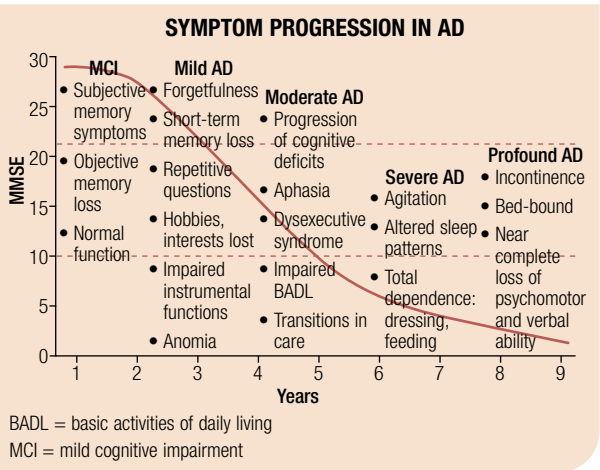

Figure 1. Symptom progression in AD
From Feldman HH, 2007.[19] Reproduced with permission.

ANSWER 2

The home environment needed to be tailored to assist Anne to be independent. It needed to be familiar. Changes to the environment could produce or exacerbate Anne's confusion.[20,21]

Safety in the home required consideration of the physical features of the building. These include those listed below.

- Access to the home: Is it level, or does it need a ramp?
- The house itself: Is each room safe?
- Falls prevention: Are there grab rails in the bathroom and shower, and no 'scatter rugs'?
- Safety in the kitchen: Is there an electric, rather than gas, stove, and is a fire extinguisher easily accessible?

Jane may also have considered using local social work and occupational health services for assessment of the home. The ACAT can help to assess the needs of the person with dementia and their carers, as well as the home environment.

Jane also needed to consider safety outside the home (e.g. did Anne need an identity bracelet, or should there be bells on the doors?) and whether Anne had access to social and leisure activities (e.g. clubs, bowls, shopping mall).

ANSWER 3

Jane's roles, at different times, might include managing medications, therapies and medical emergencies; providing supervision and emotional support; and assisting with personal care, mobility and household tasks.[22]

Carers may feel considerable satisfaction when caring for relatives; however, they often feel exhausted, isolated and burdened by their responsibilities. In a survey of carers,[22] more than half reported that their physical health had been adversely affected, a third said they had sustained a physical injury, and over half reported depression, anxiety, high levels of stress and other effects on their mental health.

Carers often used words such as tiring, demanding, depressing and frustrating. Half the caregivers surveyed said they sometimes suffered depression. The losses and sacrifices they listed included having no social life or holidays, having less time available for children or other dependents, having a greater financial burden and needing to give up or limit their employment.[23]

ANSWER 4

Carers want GPs to be proactive in addressing their needs. Ways in which this can be done are shown in *Table 1*. In addition to the issues in the table, consider whether the carer is eligible for federal government funded assistance – a carer's allowance and/or a carer's pension.

Table 1. What carers would like general practitioners to do

- Recognise their carer status and care responsibilities and include them in care planning and decision-making.
- Avoid assumptions about carer's capacity, confidence and willingness to provide home care.
- Provide plain-language information to the carer on the patient's condition, prognosis, treatment, care needs and management (including behaviour management).
- Provide information and referrals relevant to carers (e.g. in-home and residential respite care options, counselling, peer support groups, financial entitlements, self care and coping strategies).
- Give referrals to carer associations and state-wide condition specific bodies as a starting point.
- Discuss and, where appropriate, assess the carer's own physical and psychosocial health needs.
- Engage other family members in understanding and sharing care responsibilities.
- Recognise grief and loss on cessation of caring.

From Nankervis JM, Waxman PJ, O'Hara DA, Burbidge M. Caring for family carers in general practice. Med J Aust 2002;177(8):408–10.
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ANSWER 5

Counselling and psychosocial interventions for caregivers can have a positive effect on patients with AD, and other dementias, as well as their caregivers. However, one study showed that a semi-tailored program of counselling, education and support for patients with mild AD and their caregivers may not improve outcomes.[24]

The burden of care may have a significant impact on the carer and should be assessed sporadically, where possible, as the person with dementia progresses in their illness. The Caregiver Burden Scale (see *Resources*) is a 22-item self-administered questionnaire that can be used to assess the 'experience of burden'.

ANSWER 6

Irritability, aggression and wandering when present in dementia are called BPSD. They may also be referred to as neuropsychiatric symptoms.

Other BPSD include:[25]

- symptoms of disturbed perception (hallucinations), thought content (delusions), mood (depression) and anxiety
- behavioural changes such as wandering, aggression, sexual disinhibition, screaming and hoarding
- aberrant motor behaviours including pacing, rummaging, wandering and pointless hyperactivity
- apathy, sleep disturbance and agitation.

The most common BPSD are apathy, wandering, aggression and agitation.

ANSWER 7

Differential diagnoses of BPSD include:[25]

- delirium
  - from infection, pain or medication
- drugs
  - anticholinergic medications
  - anti-Parkinson medication
- changes to the environment or routine.

ANSWER 8

There are several ways to assess and manage BPSD, and there is no consensus on the optimum management process. One helpful model is the ABC model, which is described below.[26]

- **A**ntecedent or triggering event that preceded the behaviour
- **B**ehaviour itself
- **C**onsequence of that behaviour

This is a way of characterising events and resultant behaviour.

When Anne is aggressive, if Jane could intervene using non-aggressive words in a calming voice with gentle body language, it would be more likely that Anne's behaviour might be modified.

CASE 4

ROBERT IS CONFUSED

Robert, aged 76 years, is brought in to see you by his wife, Mavis. She is concerned that over the past 6 months Robert has begun to move more slowly and is prone to bouts of confusion. Last month he underwent an elective cholecystectomy, which was complicated by post-operative confusion and behavioural disturbance. He was diagnosed with delirium and given haloperidol to reduce his symptoms. Mavis tells you that his memory has not returned to its pre-operative level.

On further questioning, Mavis says Robert's memory may have been poor for up to 18 months before his admission to hospital. Despite this, he has remained at home and she had attributed his decline to 'old age'.

On examination, Robert is bradykinetic, with moderate cogwheeling in his upper limbs. Tremor is absent. You notice that his thought processes appear to be slow (bradyphrenia) and that it takes him time to answer your questions. His MMSE score is 23/30. Robert loses 2 points on orientation to time, 4 points on the serial sevens task, and 1 point on the picture copying task.

QUESTION 1

What differential diagnoses would you consider at this point?

QUESTION 2

What further history would you seek in order to narrow the diagnostic options?

QUESTION 3

What investigations might be helpful?

QUESTION 4

What is the role of neuroimaging in the diagnosis of dementia?

QUESTION 5

What are the key clinical features of Lewy body dementia (LBD)?

QUESTION 6

What other clinical features should be sought if a diagnosis of LBD is suspected?

QUESTION 7

How would you treat a psychotic episode in a patient with LBD?

QUESTION 8

How is LBD different from Parkinson dementia?

CASE 4 ANSWERS

ANSWER 1

It is important to consider persisting delirium, Parkinson disease, dementia (including AD, VaD, LBD and Parkinson-related dementia), medication and stroke.

ANSWER 2

Obtaining a time frame of Robert's symptoms is vital. If parkinsonism has been present only since the use of haloperidol then it is most likely a side effect of this medication. However, 'physical slowing' was reported by Mavis several months prior to Robert's operation. It is important to clarify what she means by this as Robert may have had undiagnosed Parkinson disease. The use of haloperidol may have exacerbated a pre-existing parkinsonism and raises the possibility of underlying Parkinson disease or LBD. If Robert's cognitive decline came before his physical slowing then LBD becomes more likely than Parkinson disease. A dramatic decline in his cognition post-operatively may suggest an ongoing delirium or stroke. A history of smoking, hypertension, diabetes or hypercholesterolaemia is more suggestive of VaD or AD.

ANSWER 3

It is important to exclude reversible conditions that might be causing Robert's symptoms. A standard 'dementia screen' includes FBE, EUC, RBG, LFT, TFT, B12 and folate levels, and CT brain. Ongoing delirium is a possible cause of Robert's symptoms, so excluding infection with urine microscopy and culture, wound swab and CXR is important. If a vascular cause is suspected (i.e. the patient is found to be in atrial fibrillation or has carotid bruits) consider an ECG and carotid ultrasound.

ANSWER 4

The role of standard neuroimaging in the diagnosis of dementing illnesses is controversial.[27] However, some form of structural neuroimaging is recommended by the *American Academy of Neurology Guidelines*.[28] CT scanning is useful for eliminating the possibility of the presence of mass lesions and stroke, and for detecting the presence of significant atrophy. Atrophy of the hippocampus is a classic finding in AD (best seen in the coronal view). The presence of lacunar infarcts and extensive deep white matter ischaemic changes suggest a vascular cause. Low resolution is a limitation of CT scanning.

The improved resolution that magnetic resonance imaging (MRI) can offer is a significant advantage for the detection of vascular changes (especially if fluid attenuated inversion recovery imaging is requested) and provides more accurate visualisation of the medial temporal lobe structures. Functional imaging, such as single photon emission computed tomography (SPECT) and positron emission tomography (PET) scanning can aid in the differential diagnosis of AD, LBD and VaD, but SPECT is limited by false negative findings[29] and PET is hard to access outside of tertiary care centres.

ANSWER 5

LBD accounts for 10–15% of all cases of dementia. There are three key diagnostic features for LBD. These are visual hallucinations, parkinsonism and significant fluctuation in cognition. The presence of two or more features in someone with dementia indicates probable LBD.[30]

Visual hallucinations, when present, are classically described as 'cinematic', in that they convey a striking sense of realism. Interestingly, they most commonly take the form of small animals, small children or adult figures, and they are often paradoxically non-distressing to the patient.

The parkinsonism seen in LBD tends to be akinetic in nature, with tremor often being absent. It is often dopamine non-responsive, and blunting of affect, bradykinesia and bradyphrenia are commonly observed.

While cognitive fluctuation can occur in a number of different dementing illnesses, it is particularly striking in LBD, occurring not only from day to day but throughout the day as well.

Table 2. Consensus criteria for the clinical diagnosis of probable and possible LBD[31]

I. The central feature required for a diagnosis of LBD is progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational function. Prominent or persistent memory impairment may not necessarily occur in the early stages, but is usually evident with progression. Deficits on tests of attention and of fronto-subcortical skills and visuospatial ability may be especially prominent.

II. Two of the following core features are essential for a diagnosis of probable LBD:

- fluctuating cognition with pronounced variations in attention and alertness
- recurrent visual hallucinations that are typically well formed and detailed
- spontaneous motor features of parkinsonism.

III. Features supportive of the diagnosis are:

- repeated falls
- syncope
- transient loss of consciousness
- neuroleptic sensitivity
- systemised delusions
- hallucinations in other modalities.

IV. A diagnosis of LBD is less likely in the presence of:

- stroke disease, evident as focal neurologic signs or on brain images
- evidence on physical examination and investigation of any physical illness or other brain disorder sufficient to account for the clinical picture.

From McKeith IG, O'Brien JT, Burn DJ, et al. Dementia with Lewy bodies. In Davis KL, Charney D, Coyle, JT(eds) et al. Neuropsychopharmacology: the fifth generation of progress. Reproduced with permission.

ANSWER 6

There are a number of other symptoms that support a diagnosis of LBD even though they do not form part of the diagnostic criteria.

These include those listed below.

- Autonomic instability – this tends to manifest either as falls (resulting from postural hypotension) or as urinary incontinence.
- Daytime somnolence – spouses will often describe their partners falling asleep as soon as they sit down to watch television.
- Increased tendency to develop delirium – this could reflect the relatively greater cholinergic deficiency that is present in LBD compared to AD.[31]
- Exquisite vulnerability to neuroleptic agents such as haloperidol – even relatively small doses can cause dramatic extrapyramidal side-effects.
- REM sleep behaviour disorder (RSBD) – this condition is characterised by a loss of the paralysis that normally accompanies stage IV REM sleep. The key to diagnosis is the presence of vivid dreams that are often violently acted out. This can result in bedclothes being in disarray when a patient awakes and a patient's partner needing to sleep in a separate bed.

ANSWER 7

If an antipsychotic must be given to a patient with LBD due to behavioural disturbance, low-dose quetiapine is generally the agent of choice. Quetiapine has minimal dopamine-blocking properties, and is therefore less likely to precipitate parkinsonian side effects than other agents. Doses in the range of 12.5–25 mg nocte should be trialled initially, and only rarely is a dose in excess of 100 mg nocte required. Quetiapine itself is not without risk. Its main side effects are sedation and postural hypotension in this patient group, who may already suffer from autonomic instability and daytime somnolence. Clozapine is also effective for psychotic symptoms in LBD, but its use is best restricted to specialist services.

ANSWER 8

The distinction is largely arbitrary. They can be viewed as a continuum, with Parkinson disease at one end and LBD at the other end. Parkinson dementia is diagnosed if motor symptoms precede the onset of cognitive decline by at least 12 months. Vice versa, LBD is diagnosed if cognitive symptoms either precede, or occur within 12 months of the onset of motor parkinsonism.[32] Lewy bodies are cellular inclusions composed of ubiquitin and alpha-synuclein. They are the pathological hallmark of Parkinson disease, where they are found in the deep cerebral striatal structures of the caudate and putamen. At autopsy, however, scattered Lewy bodies are also present in the cerebral cortex. Patients who have suffered from Parkinson disease for at least 20 years almost invariably show signs of dementia. This reflects Lewy body[33] spread to the cortical regions. LBD is characterised by the presence of Lewy bodies predominantly in the cortex with relatively few found in the deeper striatal structures. It is likely that Lewy bodies within these deeper structures can explain both the parkinsonism and extreme neuroleptic sensitivity that LBD patients demonstrate.

CASE 5

DIANNE PRESENTS WITH ODD BEHAVIOUR

Dianne, aged 64 years, presents with her son Peter. Dianne appears nonplussed when entering the room, in stark contrast to the concern evident on Peter's face. Dianne wonders why she has been brought to the doctor, as she feels well. Peter's body language shows that he has concerns, so you politely ask Dianne if she would mind if you spoke with Peter alone for 5 minutes while she returns to the waiting room. You reassure her that this is common practice.

Peter expresses his concern regarding changes in his mother's behaviour, personality and memory over the past 3 years. The initial changes were in the social context. Dianne began to show poor judgement and display inappropriate behaviour. Once a reserved lady, Peter says his mother has become overfamiliar and offensive at times. Once an empathic mother and friend, she has become more distant, and will sometimes look through a person rather than at them. At the same time, his mother's personal hygiene has deteriorated, and she has lost nearly all social contacts because 'they all annoy her'. Peter mentions that he thinks his mother's social isolation might stem from 'paranoia'. She has accused friends of stealing items from her house, and of later returning them.

Dianne returns to the room and you take a history from her while Peter is present. There is no personal or family history of neurological or psychiatric illness. Dianne's developmental history was unremarkable, and there is no clinically significant alcohol or drug history. There is no history of head trauma.

On examination, you find no signs of neurological or movement disorders.

Dianne's MMSE is 28/30. Her orientation is mostly intact; her attention and concentration are poor. On the clock-drawing test she shows poor planning in inserting the numbers, and finds it difficult to conceptualise where to place the hands to indicate 10 minutes past 11 o'clock.

QUESTION 1

What diagnoses could explain Dianne's presentation?

QUESTION 2

How would you differentiate between these diagnoses?

QUESTION 3

What investigations could help confirm the diagnosis?

QUESTION 4

What are the frontotemporal dementias (FTDs)?

QUESTION 5

What treatment is available for FTDs?

CASE 5 ANSWERS

ANSWER 1

The differential diagnosis for Dianne includes a neurodegenerative disease (NDD) or a psychiatric disorder.

NDDs to consider would be frontotemporal dementia (FTD), a frontal variant of AD or VaD.

Psychiatric disorders to consider would be depression, late-onset schizophrenia, schizoaffective disorder and bipolar affective disorder.

The gradual changes in personality and behaviour in addition to cognitive decline and psychosis favour a diagnosis of FTD for Dianne.

ANSWER 2

A focused history, mental-state examination and physical examination can help to form a diagnostic hierarchy. Dianne's presentation, with behaviour and personality changes along with memory deficits, suggests a process involving the frontal and temporal lobes. Other features to look for are further manifestations of executive dysfunction, language problems (aphasias) and new onset movement disorders.

FTD is often difficult to diagnose. It is one of the dementias that can occur in younger people and needs to be considered in those aged under 60 years. There are numerous areas of clinical overlap between NDDs and psychiatric disorders such as schizophrenia and depression.

- NDDs are commonly associated with neuropsychiatric phenomena such as delusions, hallucinations and mood disorders. FTD can be associated with all of these phenomena.[34]
- Chronic psychotic illnesses such as schizophrenia and NDDs may both present with altered personality, behaviour and cognition, and may both be associated with neuropsychiatric phenomena.
- Schizophrenia (although usually presenting at an earlier age) can be thought of as having multiple symptom domains – positive (e.g. delusions, hallucinations), negative (e.g. flat affect, alogia, amotivation, anhedonia, asociality), cognitive and disorganised. The negative features of schizophrenia are most likely deficits in frontal lobe function (pre-frontal areas).[35] Hence, there is clinical overlap between negative symptoms of schizophrenia and NDDs affecting the pre-frontal cortices, which commonly manifest as apathy.

Having reviewed the areas of clinical overlap, you may now explore the presenting features and look for supporting information.

You need to elicit more detail about Dianne's altered behaviour and personality. Why is she behaving as she is, and why do her friends 'annoy' her? The behaviour may be due to either delusions regarding her friends (such as fixed, false persecutory ideas) or impulsivity. If impulsivity is present, its extent should be sought, as it can manifest in multiple ways (e.g. a loss of manners, rash decisions, hypersexuality, complex behaviours such as gambling). You must also exclude psychosocial issues.

It is important to explore Dianne's apparent paranoia. The distinction between true paranoid delusions and 'delusions of theft' is a useful one. Although boundaries may blur, delusions of theft are common in those with short-term memory deficits and reflect a primary memory deficit rather than a psychotic process. The patient merely forgets that they have moved an item in their house, and rationalises the absence of the item by accusing others of pilfering. Such 'delusions' are thus theoretically unlikely to respond to antipsychotics, although there is limited evidence that risperidone is effective.[36]

ANSWER 3

Dianne has pronounced behaviour and personality change with cognitive impairment. There is also an absence of marked positive symptoms of schizophrenia. Hence, if investigations fail to reveal features of a dementia, an NDD would still remain a provisional diagnosis.

Blood tests will help determine whether a reversible cause for cognitive decline and behaviour change is present. A standard dementia screen would be appropriate, comprising FBE, EUC, RBG, LFTs, TFTs, C-reactive protein, and vitamin B12 and folate levels.

Structural imaging in dementia, once used mainly to exclude surgical lesions, can now aid in confirming a particular diagnosis. Structural neuroimaging is an appropriate starting point, looking for frontotemporal atrophy, often asymmetrical with preservation of posterior structures. CT would be the investigation of choice in the primary care setting. This can be followed by referral to a specialist or to a memory clinic for ongoing investigation.

FEEDBACK

Other tests include MRI, which has greater sensitivity and specificity than CT.[37] A negative scan does not exclude an FTD, particularly in the earlier stages of the disease.[38] Functional scans such as SPECT and fluorodeoxyglucose positron emission tomography (FDG-PET) have greater sensitivity than structural scans[39] and may show a characteristic pattern of frontal hypoperfusion.[39]

ANSWER 4

The FTDs are a group of dementias that primarily affect the frontal and temporal lobes. As neurones die in this area, the frontal and temporal lobes atrophy and shrink. With time, this causes behavioural change, problems with cognition, difficulty with walking and other movements, and problems with communication. FTD disorders are complex because they have a number of different features that do not necessarily correlate well with each other.

Clinically, FTDs can present in three different ways.

- Behaviour and personality changes can predominate. In this case, the entity is called behavioural variant frontotemporal dementia (bvFTD) (see *Table 3*). FTDs can also present with symptoms consistent with a functional psychiatric disorder such as mania or schizophrenia.[40]
- FTDs can present with primary language dysfunction. These clinical syndromes are subsumed under the name 'primary progressive aphasia'. Each syndrome has its characteristic aphasia, associated anatomical underpinnings and neuropathology. They are a non-fluent/agrammatic type and a semantic type.
- FTDs can present clinically with neurological signs. These variants include motor neurone disease, progressive supranuclear palsy and corticobasal degeneration.[41]

ANSWER 5

Management of FTDs may be very challenging for patients, carers and doctors. As with most dementias, there is no disease-modifying treatment available. Furthermore, some experts recommend strongly against the use of cholinesterase inhibitors, as studies have revealed a deterioration in

behaviour associated with treatment. However, medications are available to ameliorate specific symptoms. Liaison with a specialist in the field and a multidisciplinary team approach to support the patient and carers are of paramount importance. Guidance and support for managing behavioural problems can be accessed through support groups such as Alzheimer's Australia and FRONTIER (see *Resources*).

Table 3. International consensus criteria for bvFTD

I. Neurodegenerative disease
The following symptom must be present to meet criteria for bvFTD.
A. Shows progressive deterioration of behaviour and/or cognition by observation or history (as provided by a knowledgeable informant).
II. Possible bvFTD
Three of the following behavioural/cognitive symptoms (A–F) must be present to meet criteria. Ascertainment requires that symptoms be persistent or recurrent, rather than single or rare events.
A. Early* behavioural disinhibition [one of the following symptoms (A.1–A.3) must be present]:
A.1.Socially inappropriate behaviour
A.2.Loss of manners or decorum
A.3.Impulsive, rash or careless actions
B. Early apathy or inertia [one of the following symptoms (B.1–B.2) must be present]:
B.1.Apathy
B.2.Inertia
C. Early loss of sympathy or empathy [one of the following symptoms (C.1–C.2) must be present]:
C.1.Diminished response to other people's needs and feelings
C.2.Diminished social interest, interrelatedness or personal warmth
D. Early perseverative, stereotyped or compulsive/ritualistic behaviour [one of the following symptoms (D.1–D.3) must be present]:
D.1.Simple repetitive movements
D.2.Complex, compulsive or ritualistic behaviours
D.3.Stereotypy of speech
E. Hyperorality and dietary changes [one of the following symptoms (E.1–E.3) must be present]:
E.1.Altered food preferences
E.2.Binge eating, increased consumption of alcohol or cigarettes
E.3.Oral exploration or consumption of inedible objects
F. Neuropsychological profile: executive/generation deficits with relative sparing of memory and visuospatial functions [all of the following symptoms (F.1–F.3) must be present]:
F.1.Deficits in executive tasks
F.2.Relative sparing of episodic memory
F.3.Relative sparing of visuospatial skills
III. Probable bvFTD
All of the following symptoms (A–C) must be present to meet criteria.
A. Meets criteria for possible bvFTD
B. Exhibits significant functional decline (by caregiver report or as evidenced by Clinical Dementia Rating Scale or Functional Activities Questionnaire scores)
C. Imaging results consistent with bvFTD [one of the following (C.1–C.2) must be present]:
C.1.Frontal and/or anterior temporal atrophy on MRI or CT
C.2.Frontal and/or anterior temporal hypoperfusion or hypometabolism on PET or SPECT
IV. bvFTD with definite frontotemporal lobar degeneration (FTLD) pathology
Criterion A and either criterion B or C must be present to meet criteria.
A. Meets criteria for possible or probable bvFTD
B. Histopathological evidence of FTLD on biopsy or at post-mortem
C. Presence of a known pathogenic mutation
V. Exclusionary criteria for bvFTD
Criteria A and B must be answered negatively for any bvFTD diagnosis. Criterion C can be positive for possible bvFTD but must be negative for probable bvFTD.
A. Pattern of deficits is better accounted for by other non-degenerative nervous system or medical disorders
B. Behavioural disturbance is better accounted for by a psychiatric diagnosis
C. Biomarkers strongly indicative of AD or other neurodegenerative process
*As a general guideline 'early' refers to symptom presentation within the first 3 years.
From Rascovsky K, Hodges JR, Knopman D, et al. Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. Brain 2011;134(9):2456–77. Reproduced by permission of Oxford University Press.

CASE 6

MARGARET PRESENTS WITH ONGOING CONFUSION

Margaret, aged 75 years, is brought in to see you by her daughter, Rose. Margaret is happily married and until recently volunteered at the local soup kitchen. You have known her for many years, and immediately you notice a distinct change. Usually an impeccably dressed lady, Margaret appears somewhat dishevelled. Her gait is slowed and her usually warm smile lacks depth.

Margaret has a history of hypertension, insulin resistance and urinary incontinence, and has had a tendency towards anxiety and depression in the past. Her current medications are perindopril 4 mg mane, oxybutynin 5 mg bd and amitriptyline 150 mg nocte. Three months ago Margaret suffered a UTI, for which she required hospitalisation due to associated delirium (acute organic brain syndrome). Due to the severity of the delirium a CT scan of the brain was performed, which showed 'age appropriate generalised atrophy'.

Rose is worried about her mother. At the hospital, the doctors assured her the delirium would pass. However, Margaret has only partially improved. The 'strange thoughts' have gone, but she remains more confused and forgetful than before the UTI. Rose wants to know if the confusion will resolve.

QUESTION 1

What are the features of delirium?

QUESTION 2

Which cognitive feature will be seen on MMSE in delirium?

QUESTION 3

What is the neuropathogenesis of delirium?

FURTHER INFORMATION

You reassure Rose as best you can, telling her that delirium can take some time to resolve. You explain that you would first like to rule out any ongoing medical problems. You perform an MMSE and Margaret scores 21/30. Six months ago her score was 30/30 on routine screening. Margaret lost 2 points on orientation, and although she was once a bookkeeper, she only scored 1/5 on the serial sevens task. She also scored 0/3 on the recall task. To confirm that the serial sevens task was performed poorly due to poor attention, rather than a limitation in mathematics, you try another test requiring attention. Margaret has great difficulty saying the months in reverse order, and is frequently distracted from the task.

A repeat MSU and delirium screen are ordered, and the following week Rose and Margaret return for the results. They are all essentially normal.

Rose turns to you and asks, 'Will my mum get better?'

QUESTION 4

Is delirium reversible?

QUESTION 5

What do you tell Rose?

QUESTION 6

What can be done for Margaret?

CASE 6 ANSWERS

ANSWER 1

Delirium is an acute confusional state that is also known as an 'acute brain syndrome' or an 'acute organic reaction'.

It is characterised by acute onset with inattention the most common cognitive deficit.[43] It is also characterised by disorganised thinking and an altered level of consciousness.

Table 4. Delirium – DSM IV definition

- Disturbed consciousness
- Reduced ability to focus, sustain or shift attention
- Change in cognition or development of perpetual disturbance
- Rapid onset (hours to days), fluctuates during the course of the day
- Evidence that it is a result of illness

From Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR Fourth Edition, American Psychiatric Association.

It is important to consider two main presentations of delirium – hyperactive and hypoactive delirium. Hyperactive patients may be loud, restless, excitable and hyper-vigilant. They are more prone to falls. Hypoactive delirium can easily be overlooked as the patient lies passively in bed and can appear to be the 'ideal patient'. They are quiet and tend to drift off to sleep, and have slow, incoherent speech. Mixed, fluctuating presentations are common. Dementia and depression are common differential diagnoses.

ANSWER 2

Patients with delirium tend to perform poorly on MMSE due to their limited attention span. In particular, patients will perform poorly on the serial sevens task, which requires focused thought. Short-term memory is also impaired, probably as a direct consequence of inattention.[44] Executive functions such as planning, organisation and abstraction are affected by delirium, but this will not be picked up on an MMSE.

ANSWER 3

The underlying mechanisms of delirium remain elusive. However, the most robust evidence to date supports a relative cholinergic deficit and dopaminergic excess in the brain.[45]

ANSWER 4

The question of prognosis is a challenge. It is clear that delirium is associated with adverse outcomes such as increased hospital stay, cognitive decline, functional decline, institutionalisation and mortality.[46]

There is a common association between dementia and delirium and evidence shows that many people who appear cognitively

well, but who develop delirum, have cognitive problems long-term. This is supported by a recent study published in the *Archives of Internal Medicine*,[47] which showed that controlling for pre-existing dementia and medical disease burden, patients with delirium had a significant and sustained deterioration in cognition for up to 5 years compared to the control group. Although the study was not without its shortcomings[48], the authors concluded that delirium per-se is neurotoxic and causes long-term cognitive decline. While particular aspects of delirium, such as psychosis and severe attentional deficits, seem to be readily reversible, there is a non-reversible, negative impact on cognition.

ANSWER 5

It would be appropriate to explain to Rose that the understanding of delirium is evolving. While we have thought for many years that delirium is reversible, it is becoming clear that this is not always the case. While the majority of cognitive and neuropsychiatric phenomena of a delirium are reversible in most cases, it may be that Margaret will not improve further. In fact, Margaret may continue to deteriorate.

ANSWER 6

A review of Margaret's current medication is appropriate. It is important to reduce or eliminate any medication with an anticholinergic effect.[49] Margaret is currently prescribed two strongly anticholinergic medications – oxybutynin for bladder instability and amitriptyline for depression. Anticholinergic burden has a clear association with cognitive decline,[49] which is explained by the current cholinergic deficit theory mentioned in *Answer 3*.

The clinical need for the oxybutynin and its current dose need to be re-evaluated. As a first step you decide to halve the dose. Similarly, the indication for amitriptyline requires revision. Until now you have been loath to wean the amitriptyline as Margaret has had long-term issues with anxiety, insomnia and depression. In discussion with Margaret and Rose a decision is made to wean and cease the antidepressant. Should Margaret's mental state deteriorate you will try another, less anticholinergic medication.

If Margaret's cognition does not improve significantly after altering her medication, it would be appropriate to refer Margaret for assessment of her cognitive state, in particular for dementia. She may be prescribed a cholinesterase inhibitor if she has AD.

FEEDBACK

The presence of delirium does complicate and make the treatment of serious illnesses more difficult, but it can also result in permanent, irreversible brain damage. It is wise to show equal concern for the brain as we do for the other organs of the body.[50]

CASE 7

ALBERT IS WORRIED HE MAY DEVELOP DEMENTIA

Albert, a secondary school teacher aged 49 years, presents for a check-up and prescription renewal. He tells you he is very worried about getting AD, which his mother has. He and his 54-year-old sister had been caring for their mother for several years until she moved into residential aged care a month ago. Albert wants to know if he and his sister can be tested to determine whether they are destined to develop dementia, and what he can do to avoid his mother's fate. Albert lives with his wife and two teenage children and is relatively healthy. However, he has been taking medication for hypertension for 2 years and has mild hypercholesterolaemia.

Two months ago Albert's fasting lipids were total cholesterol 6.0 mmol/L, HDL 1.2 mmol/L (normal >1.0 mmol/L), LDL 4.3 mmol/L (normal <2.5 mmol/L), TG 2.3 mmol/L (normal <1.7 mmol/L). You told him at that time to lower his cholesterol intake and to return in 2 months for repeat blood tests.

On this visit, Albert's BP is 147/95 mmHg.

QUESTION 1

What do you ask Albert about his family history to assess whether he may be at risk of a genetic form of dementia?

FURTHER INFORMATION

Albert believes his mother first displayed symptoms of dementia at age 74 years. She was diagnosed with AD 3 years later. To his knowledge, no other family member has been affected. His mother's father and sister lived to around 75 years of age, but her mother and brother died 'young'.

QUESTION 2

What do you advise Albert about his genetic risk for dementia and the possibility of genetic testing?

QUESTION 3

How do Albert's vascular risk factors affect his dementia risk?

QUESTION 4

What do you advise Albert about managing his vascular risk factors in relation to his dementia risk?

QUESTION 5

What other potential dementia risk factors should you ask Albert about?

FURTHER INFORMATION

Albert's work is intellectually demanding and he is learning Spanish. His diet is relatively healthy and his body mass index is in the normal range. He has never smoked and only occasionally drinks alcohol. He is on his feet a few hours a day at work, but does no regular exercise.

QUESTION 6

What do you advise Albert about lifestyle strategies to reduce his risk of dementia?

QUESTION 7

Albert wants to know if he will definitely avoid dementia if he does all these things. What do you tell him?

CASE 7 ANSWERS

ANSWER 1

In the rare familial form of AD, genetic mutations on three chromosomes have been identified that cause autosomal dominant transmission and younger onset (typically before 60 years of age). These genes account for perhaps 1% of AD cases.[51] Familial FTD accounts for around 10–15% of cases of FTD.[52] There are also rare genetic forms of LBD, cerebrovascular disease and other causes of dementia.

Ask Albert about his family history and the age of onset of his mother's symptoms, and whether other family members have been affected.

ANSWER 2

There is nothing in Albert's family history to suggest autosomal dominant transmission or younger onset dementia, so there is no indication his family is affected by a genetic cause of AD.

For families who are affected by single gene mutations that cause dementia, genetic counselling and predictive genetic testing are available through state-based genetics services (see *Resources*).

In sporadic AD, the apolipoprotein E epsilon 4 allele (APOE ε4) has been identified as a major risk factor, and other susceptibility genes have been identified. APOE ε4 may also increase risk of cerebrovascular disease and possibly of LBD. Clinical APOE testing is not currently recommended because it is not possible to predict who will or will not develop AD.

Albert can be reassured that only a small proportion of dementia cases are thought to be inherited. In his case there is no genetic test available to determine whether or not he will develop AD. However, epidemiological studies suggest he is at 2–3 times higher risk because he has an affected first-degree relative.[53] While he can't change this, there are many other risk factors that he can do something about.

ANSWER 3

Hypertension is a risk factor for cerebrovascular disease and VaD. Hypertension in midlife can also increase the risk of AD. Studies assessing long-term use of antihypertensives from midlife suggest a cumulative reduction in risk of dementia for each year of treatment.[54]

High midlife total serum cholesterol is associated with increased risk of any dementia and of AD, and statin use may be associated with reduced risk.[55]

Albert's hypertension and high cholesterol potentially increase his risk of later developing dementia. They may contribute to cerebrovascular disease and may also exacerbate AD pathology.

FEEDBACK

Diabetes and pre-diabetes syndromes are also risk factors for all dementia, AD and VaD. Few studies have examined the effect of treatment of diabetes on dementia risk, and for those that have, the results are mixed.[56]

ANSWER 4

Albert should be advised to control his high blood pressure and cholesterol using evidence-based strategies. Vascular risk factors require treatment for a range of reasons, and potentially reducing the risk of dementia may be an added benefit and provide motivation for Albert to adhere to medication and lifestyle recommendations.

ANSWER 5

Higher participation in cognitively stimulating activities is associated with reduced dementia risk.[57] Mental challenge and learning contribute to increased brain volume and efficient cognitive function.

Regular physical exercise is associated with lower risk of developing dementia.[58] The mechanisms by which physical activity protects against dementia likely include reduction of vascular risk factors, neurogenesis and neuroprotection.

Diet may play a role in dementia risk, but the evidence for specific nutrients is inconclusive. Diets low in saturated fat and high in vegetable consumption may be beneficial. Obesity and underweight in midlife are associated with increased dementia risk.[59]

Moderate alcohol consumption is associated with reduced dementia risk, but there is no evidence that non-drinkers should take up alcohol.[60] Smoking is a risk factor for dementia.[61]

ANSWER 6

Albert should be encouraged to maintain regular participation in cognitively challenging activities. He should be advised to be more physically active. The Australian Physical Activity Guidelines (see *Resources*) recommend adults perform at least 30 minutes per day of moderate-intensity physical activity to reduce their risk for a range of conditions.

Serious head injury is associated with increased dementia risk. It is therefore appropriate to encourage Albert to wear head protection should he engage in sports or activities that could result in head injuries, such as bicycling.

Albert should be advised to follow the Australian Dietary Guidelines (see *Resources*), limit his saturated fat intake and eat plenty of fruit and vegetables. He should maintain his drinking and smoking status.

ANSWER 7

Albert cannot be guaranteed that these interventions will decrease his risk of AD, despite their other health benefits. The recommendations are based on epidemiological research evidence of what, on average, puts people at lower risk, rather than clinical trials that prove interventions can reduce risk. *Table 5* is a summary of factors associated with dementia risk.

Table 5. Summary of factors associated with dementia risk

Health or lifestyle factor	Risk for cognitive decline and dementia
Brain	
Mental activity	Higher mental stimulation through education, occupation or leisure is associated with lower risk.
Social activity	Higher social interaction in late life is associated with lower risk.
Body	
Alcohol	Moderate alcohol consumption is associated with lower risk, but comes with a warning – alcohol consumption can cause other health problems.
Diet	Findings for individual nutrients are inconsistent. Higher intakes of fruit, vegetables and fish seem to be associated with lower risk.
Physical activity	Regular physical exercise at all ages is associated with lower risk.
Heart	
Blood pressure	Untreated midlife high blood pressure is associated with increased risk.
Cholesterol	Untreated midlife high cholesterol is associated with increased risk.
Diabetes	Type 2 diabetes is associated with increased risk.
Smoking	Current smoking is associated with increased risk, former smoking is not.
Weight	Midlife obesity and underweight are associated with increased risk.

Reproduced with permission from Farrow M, O'Connor E. Targeting brain, body and heart for cognitive health and dementia prevention. Alzheimer's Australia Paper 29. 2012. Alzheimer's Australia: Canberra.

This is included in Alzheimer's Australia's Your Brain Matters program, which encourages a holistic approach to looking after your brain, body and heart at all ages.

CASE 8

MARK IS HAVING DIFFICULTY MAKING DECISIONS

Mark, aged 75 years, was diagnosed with VaD 5 years ago. His wife Lillian is his primary carer and they have continued to live independently in their unit. They have two adult children who live close by. Their daughter Sarah is more involved in her parent's care, assisting with driving to the shops and appointments, and helping with household chores. Sarah's role has expanded over the past 3 years as Mark's dementia has progressed and Lillian's ability to manage their household has decreased. The family is now considering placing Mark in a residential care facility.

When Mark was diagnosed with VaD, you, as the longstanding family doctor, were invited and took part in a family meeting called by their son Travis. The family wished to discuss longer-term planning, ongoing care and what Travis called 'legal issues' with you. The family wanted to make sure that Mark's wishes were respected, and that he participated in the decision-making process while he still had capacity.

QUESTION 1

What issues may have been appropriate to discuss at this meeting? How should legal issues be addressed?

QUESTION 2

What key features should you cover in a meeting to deliver bad news, and what system may assist you with delivering these messages?

FURTHER INFORMATION

About 1 year after Mark's diagnosis of VaD, Sarah had a baby. You received a call from Mark's attorney asking if you were able to assess Mark's testamentary capacity (TC) because Mark and Lillian wanted to update their will, as they had done with the birth of each new grandchild.

QUESTION 3

What is required to assess TC in terms of the understanding of a testator, and what are the pitfalls related to the use of the MMSE you might encounter in this area?

FURTHER INFORMATION

Shortly after the call from the lawyer, Lillian phoned you and asked how she could stop Mark from driving. She was concerned that he sometimes forgot where he parked the car at the shops, got lost on the way to places he used to drive to regularly and scraped the car a few times while trying to park. Mark was adamant that he was still a good driver, and that he intended to continue driving, as he was not ready to surrender his licence.

QUESTION 4

List the impact that dementia may have on driving ability and skills.

QUESTION 5

What are the key features to elicit in the assessment of fitness to drive?

FURTHER INFORMATION:

Mark has progressed further and has now been placed in a local residential care facility. He no longer recognises Lillian or his children, and he has lost considerable weight. The staff call you to tell you that they have noticed some blood in Mark's stools. You order a series of investigations, and on examination of the results notice that he has significant anaemia and a large lesion next to his caecum on CT abdomen.

QUESTION 6

What features differentiate an ACD from a plan of care?

CASE 8 ANSWERS

ANSWER 1

Issues that might have been discussed at an initial meeting with the family include:

- current living arrangements and the need for and role of a carer
- education and training for carers to assist with managing both the carer and the patient, and to limit the impact on the carer and to delay placement of the patient into a facility[63,64,65,66]
- legal issues that facilitate planning.

Legal issues (e.g. enduring power of attorney for financial issues, enduring guardianship for accommodation and healthcare decisions, ACDs for future treatment, including end-of-life care) should be addressed at an early stage. Decisions about these issues may have already been made.

Assessment of decision-making capacity should focus only on the decisions that need to be made or the documents that need to be signed. Patients should only be encouraged to make the decisions they are capable of making, and their ability to do so will depend on the complexity of the decision and their situation in light of the nature and severity of their cognitive impairment (see *Figure 2*). Even people with early dementia may be unable to make some complex decisions, whereas people with severe dementia may still be able to make simple decisions.

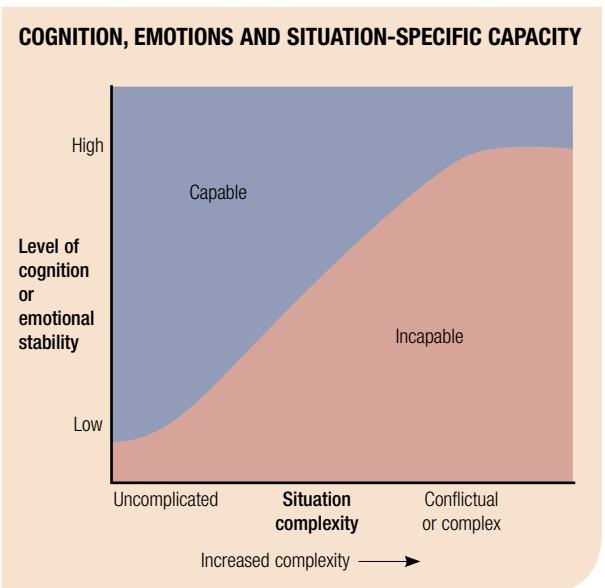

Figure 2. Assessment of testamentary capacity and vulnerability to undue influence
From Shulman, 2007.[62]

Decision-making capacity depends on the decision to be made. It may be different for every decision made, even within one domain. It depends on the complexity of the decision.[67]

In this case, the family's agenda was clearly to facilitate planning and to give voice to Mark's decisions and wishes. However, it is important to be aware of the vulnerability of people with dementia to undue influence. Be wary when people with dementia are encouraged, by others seeking to gain from them, to gratuitously change documents such as wills and powers of attorney that have been properly executed in the past.

Finally, it is important to include comprehensive documentation of discussions with the patient and family members, including telephone conversations, in the patient's medical record. The patient assessment, including the tools relied upon for the assessment, also need to be documented in the records.

ANSWER 2

The four main objectives of an interview to disclose bad news are to:

- gather information from the patient
- transmit medical information
- provide support to the patient
- develop a collaborative strategy and management plan for the future.

The SPIKES system is a system to help with delivering bad news to patients. It may be modified to assist carers and families, as the patient may not have the cognition to fully participate. However, the patient has the right to information about their condition, and you need to skilfully ascertain how much information the patient would like to know.

The SPIKES system consists of six steps.[68,69]

1. **S**etting up the interview
2. Assessing the **P**atient's **P**erception
3. Obtaining the patient's **I**nvitation (for information)
4. Giving **K**nowledge and information to the patient
5. Addressing the patient's **E**motions with **E**mpathic responses
6. **S**trategy and **S**ummary

Other important principles include the following.

- Break bad news gradually.
- Titrate the amount of information according to the reaction and expressed wishes of the patient and carers.
- Hold out some hope (i.e. research into possible cures).
- Ask the patient if they would like to see you alone or with family member/s.
- You may need more than one session with the patient to discuss diagnosis and consequences.
- Strong emotions will preclude the patient attending to information, so it may be best to schedule another visit.
- Follow-up with another interview. The patient and their family will think of many questions when they go away. They may want to come back with other family members.

ANSWER 3

The assessment of TC in someone with dementia depends on an assessment of the complexity of the task and the person's situation, in the context of the severity of their cognitive impairment (see *Figure 2*).

TC, as defined by the Banks v. Goodfellow Criteria, requires that the testator has:[62,70]

- understanding of the nature of a will
- knowledge of the nature and extent of one's assets
- knowledge of persons who have a reasonable claim to be beneficiaries
- understanding of the impact of the distribution of the assets of the estate
- a confirmation that the testator is free of any delusions that influence the disposition of assets
- ability to express wishes clearly and consistently in an orderly plan of disposition.

Pitfalls related to the use of the MMSE and the clock-drawing test to assess TC are that they assess higher-level brain functions and are screening tests for cognitive impairment and used to show changes over time. They are not diagnostic of dementia and cannot be used as a measure of TC.[62]

It is not recommended that GPs, or any other healthcare professional, assess or comment on TC unless they are trained to do so. You might refer to an expert in the field of assessing TC, such as a neurologist, psychiatrist, psychologist or geriatrician.[67]

Again, thorough documentation of all discussions in the patient's medical record is essential.

ANSWER 4

Dementia may affect the ability to drive in many ways, including:[71]

- navigational errors (e.g. forgetting familiar routes or becoming lost in a familiar environment)
- limited attention span or breaks in concentration (e.g. not seeing road signs or not responding to them)
- judgement errors (e.g. misjudging distances when parking or stopping, misjudging speed)
- confusion in a stressful situation (e.g. hitting the accelerator instead of the brake)
- impaired decision-making and problem-solving (e.g. not giving way or stopping at an inappropriate time)
- lack of insight and denial of limited ability
- slow reaction time and not responding to directions
- inadequate eye–hand coordination.

ANSWER 5

Issues that might be helpful in assessing a patient's ability to drive include the following.[71]

- Be aware of different state and territory requirements.
- Use a combination of medical/specialist and practical assessment.
- Ask about and assess:
  - driving history, including accidents or referral by police or other authority for assessment
  - vision, both front and side
  - hearing
  - reaction time
  - problem solving
  - coordination
  - praxis
  - alertness and perception
  - insight
  - other aspects of driving performance such as telling left from right, confusion on familiar routes, confusion at roundabouts, staying in the correct lane, 'stop' and 'go' at the lights, reading a map or directions, mood and confidence while on the road
  - if in doubt, send the patient for an on-road assessment, preferably with an occupational therapist.
- Cognitive tests correlate poorly with driving ability.
- Patients will need to tell their insurance company when they are given a diagnosis of dementia, as their policy may no longer be valid once they obtain this diagnosis.

In New South Wales, nobody with dementia can have an unconditional driving licence.

ANSWER 6

ACD

An ACD is a written or oral statement made by a capable adult regarding wishes, preferences, values and beliefs about future treatment decisions, including end-of-life treatment. It may include instructions about future use or restriction of particular medical treatments ('treatment directive') and/or the details of a preferred substitute decision-maker ('proxy directive'). It is only used when the person loses the capacity to make decisions about their healthcare.[72]

Plan of care

A plan of care is a consensus-based discussion involving the patient (who, regardless of not having capacity, may want to have some input into this discussion), carer (usually the person responsible) and medical staff around best interests, as the patient is no longer able to provide informed consent about their future treatment. The carer or person responsible can state their wishes for the patient's healthcare, based on what they believe is in the patient's best interest and reflecting what the patient would have wanted.[72]

1. General Practitioner assessment of Cognition. Available at www.gpcog.com.au [accessed 27 March 2013].
2. Geriatric Depression Scale. Available at www.stanford.edu/~yesavage/GDS.english.short.score.html [accessed 27 March 2013].
3. Phillips J, Pond D, Shell A. Quality dementia care: no time like the present: the importance of a timely dementia diagnosis. Alzheimer's Australia 2010;7:13–14. Available at www.fightdementia.org.au/common/files/NAT/20100900_Nat_QDC_QDC7NTLTP.pdf [accessed 02 April 2013].
4. Yates M, Pond D. The dementia pathway. Australian Doctor 2012;17 Sept. Available at www.australiandoctor.com.au/clinical/therapy-update/the-dementia-pathway [accessed 02 April 2013].
5. Basic D, Khoo A, Conforti D, Rowland J, Vrantsidis F, LoGiudice D, et al. Rowland Universal Dementia Assessment Scale, Mini-Mental State Examination and General Practitioner assessment of Cognition in a multicultural cohort of community-dwelling older persons with early dementia. Aust Psychol 2009;44(1):40–53.
6. Stolk Y. Approaches to the influence of culture and language on cognitive assessment instruments: the Australian context. Aust Psychol 2009;44(1):1–5.
7. Smith K, Flicker L, Dwyer A, et al. Assessing cognitive impairment in Indigenous Australians: re-evaluation of the Kimberley Indigenous Cognitive Assessment in Western Australia and the Northern Territory. Aust Psychol 2009;44(1):54–61.
8. LoGiudice D, Smith K, Thomas J, et al. Kimberley Indigenous Cognitive Assessment tool (KICA): development of a cognitive assessment tool for older indigenous Australians. Int Psychogeriatr 2006;(18)02:269–80.
9. Ahlskog JE, Geda YE, Graff-Radford NR, Petersen RC. Physical exercise as a preventive or disease-modifying treatment of dementia and brain aging. Mayo Clin Proc 2001;86(9):876–84.
10. Gates N, Valenzuela M, Sachdev P, et al. Study of Mental Activity and Regular Training (SMART) in at risk individuals: a randomised double blind, sham controlled, longitudinal trial. BMC Geriatr 2011;11(19):1–15.
11. Reutens S, Peisah C, Brodaty H. Dementia: to screen or not to screen. Medicine Today 2009;10(5):14–22. Available at www.medicinetoday.com.au/home/article/view/330 [accessed 02 April 2013].
12. Brodaty H. 14 essentials in the practice & art of diagnosis & management of dementia. 2011. Available at www.dementiaresearch.com.au/images/dcrc/presentations/2011/GPs+-14+Essentials_Homebush_21.pdf [accessed 02 April 2013].
13. Australian Government, Department of Health and Ageing. Review of Pharmaceutical Benefits Scheme anti-dementia drugs to treat Alzheimers disease. Available at www.pbs.gov.au/info/reviews/anti-dementia-drugs [accessed 02 April 2013].
14. Igases M, Kohara K. Miki T. The association between hypertension and dementia in the elderly. Int J Hypertens 2012. Available at www.hindawi.com/journals/ijht/2012/320648 [accessed 02 April 2013].
15. Nagai M, Hoshide S, Kario K. Hypertension and dementia. Am J Hypertens 2010;23(2):116–24.
16. Alzheimer's Australia. Available at www.fightdementia.org.au [accessed 02 April 2013].
17. Pond D. Dementia – an update on management. Aust Fam Physician 2012;41(12):936–9.
18. Alford L. What men should know about the impact of physical activity on their health. Int J Clin Pract 2010;64(13):1731–4.
19. Feldman HH, ed. Atlas of Alzheimer's disease. London: Informa Healthcare; 2007.
20. Better Health Channel. Dementia. Available at www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Dementia_explained [accessed 02 April 2013].
21. Alzheimer's Australia. Safety issues. Available at www.fightdementia.org.au/services/safety-issues.aspx [accessed 02 April 2013].
22. Nankervis JM, Waxman PJ, O'Hara D, Burbidge M. Caring for family carers in general practice. Med J Aust 2002;177(8):408–10.
23. Wilkinson D, Stave C, Keohane D, Vincenzino O. The role of general practitioners in the diagnosis and treatment of Alzheimer's disease: a multinational survey. J Int Med Res 2004;32(2),149–59.
24. Waldorff FB, Buss DV, Eckermann AM, et al. Efficacy of psychosocial intervention in patients with mild Alzheimer's disease: the multicentre, rater blinded, randomised Danish Alzheimer Intervention Study (DAISY). BMJ 2012;345:e4693.
25. Brodaty, H. Update on management of BPSD. 2012. Available at www.dementia.unsw.edu.au/images/dcrc/output-files/41-bpsd_dtsc_290212.pdf [accessed 02 April 2013].
26. Bendigo Health. ABC of behaviour management. Available at www.dementiamanagementstrategy.com/Pages/ABC_of_behaviour_management.aspx [accessed 02 April 2013].
27. Sullivan V, Majumdar B, Richman A, Vinjamuri S. To scan or not to scan: neuroimaging in mild cognitive impairment and dementia. Adv Psych Treatment 2012;18:457–66.
28. Knopman DS, DeKosky ST, Cummings JL, et al. Practice parameter: diagnosis of dementia (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2001;56(9);1143–53.
29. Jagust M, Thisted R, Devous MD Sr, et.al. SPECT perfusion imaging in the diagnosis of Alzheimer's disease: a clinical-pathologic study. Neurology 2001;56(7):950–6.
30. McKeith IG, Dickson DW, Lowe J, et.al. Diagnosis and management of dementia with Lewy bodies: third report of the DLB Consortium. Neurology 2005;65(12):1863–72.
31. McKeith IG, O'Brien JT, Burn DJ, Perry RH, Perry EK. Dementia with Lewy bodies. In: Davis KL, Charney D, Coyle JT, Nemeroff C, eds. Neuropsychopharmacology: the fifth generation of progress – an official publication of the American College of Neuropsychopharmacology. Philadelphia: Lippincott Williams & Wilkins, 2002.
32. McKeith IG, Mintzer J, Aarsland D, et al. Dementia with Lewy bodies. Lancet Neurol 2004;3(1):19–28.
33. Hely MA, Reid WG, Adena MA, Halliday GM, Morris JG. The Sydney multicenter study of Parkinson's disease: the inevitability of dementia at 20 years. Mov Disord 2008;23(6):837-44.
34. Mendez MF, Lauterbach EC, Sampson SM. ANPA Committee on Research. An evidence-based review of the psychopathology of frontotemporal dementia: a report of the ANPA Committee on Research. J Neuropsychiatry Clin Neurosci 2008;20(2):130–49.
35. Semkovska M, Bédard MA, Stip E. Hypofrontality and negative symptoms in schizophrenia: synthesis of anatomic and neuropsychological knowledge and ecological perspectives; Encephale 2001;27(5):405–15.
36. Shigenobu K, Ikeda M, Fukuhara R, Komori K, Tanabe H. A structured, open trial of risperidone therapy for delusions of theft in Alzheimer disease. Am J Geriatr Psychiatry 2003;11(2):256–7.
37. Cardarelli R, Kertesz A, Knebl JA. Frontotemporal dementia: a review for primary care physicians. Am Fam Physician 2010;82(11):1372–7.
38. Piguet O, Hornberger M, Mioshi E, Hodges JR. Behavioural-variant frontotemporal dementia: diagnosis, clinical staging, and management. Lancet Neurol 2011;10(2):162–72.
39. Varma AR, Adams W, Lloyd JJ, et al. Diagnostic patterns of regional atrophy on MRI and regional cerebral blood flow change on SPECT in young onset patients with Alzheimer's disease, frontotemporal dementia and vascular dementia. Acta Neurol Scand 2002;105(4):261–9.
40. Scheltens P, Rockwood K. How golden is the gold standard of neuropathology in dementia? Alzheimers Dement 2011;7(4):486–9.
41. Ringman JM, Vinters HV. Alzheimer's disease and the frontotemporal

dementia syndromes. In: Coffey CE, Cummings JL, eds. Textbook of geriatric neuropsychiatry. 3rd edn. Virginia: American Psychiatric Publishing, 2011, 389–406.

42. Rascovsky K, Hodges JR, Knopman D, et al. Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. Brain 2011;134(9):2456–77.
43. Meagher DJ, Moran M, Raju B, Gibbons D, Donnelly S. Phenomenology of delirium: assessment of 100 adult cases using standardised measures. Br J Psychiatry 2007;190:135-41.
44. Hodges JR. Cognitive assessment for clinicians. 2nd ed. New York: Oxford University Press; 2007.
45. Tune LE, DeWitt MA. Delirium. In: Coffey CE, Cummings JL, eds. Textbook of geriatric neuropsychiatry. 3rd edn. Virginia: American Psychiatric Publishing, 2011; 389–406.
46. Martins S, Fernandes L. Delirium in elderly people: a review. Front Neurol. 2012;3:101.
47. Gross AL, Jones RN, Habtemariam DA, et al. Delirium and long-term cognitive trajectory among persons with dementia. Arch Intern Med 2012;172(17):1324–31.
48. Vasilevskis EE, Ely EW. The danger of delirium: comment on "Delirium and long-term cognitive trajectory among persons with dementia". Arch Intern Med 2012;172(17):1331–2.
49. Chew ML, Mulsant BH, Pollock BG, et al. Anticholinergic activity of 107 medications commonly used by older adults. J Am Geriatr Soc 2008;56(7):1333–41.
50. Engel GL, Romano J. Delirium, a syndrome of cerebral insufficiency. J Chronic Dis 1959;9(3):260–77.
51. Dominantly Inherited Alzheimer Network (DIAN). Available at www.dian-info.org [accessed 19 June 2012].
52. FRONTIER (Frontotemporal Dementia Research Group). Available at www.neura.edu.au/frontier [accessed 19 June 2012].
53. Jarvik LF, Blazer D. Children of Alzheimer patients: an overview. J Geriatr Psychiatry Neurol 2005;18:181–6.
54. Haag MD, Hofman A, Koudstaal PJ, Breteler MM, Stricker BH. Duration of antihypertensive drug use and risk of dementia. A prospective cohort study. Neurology 2009;72(20):1727–34.
55. Anstey KJ, Lipnicki DM, Low L-F. Cholesterol as a risk factor for dementia and cognitive decline: a systematic review of prospective studies with meta-analysis. Am J Geriatr Psychiatry 2008;16(5):343–54.
56. Lu F-P, Lin K-P, Kuo H-K. Diabetes and the risk of multi-system aging phenotypes: a systematic review and meta-analysis. Available at www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0004144 [accessed 02 April 2013].
57. Valenzuela MJ, Sachdev P. Brain reserve and dementia: a systematic review. Psychol Med 2006;36(4):441–54.
58. Lautenschlager N, Cox K, Cyarto EV. The influence of exercise on brain aging and dementia. Biochim Biophys Acta 2012;1822(3):474–81.
59. Anstey KJ, Cherbuin N, Budge M, Young J. Body mass index in midlife and late-life as a risk factor for dementia: a meta-analysis of prospective studies. Obes Rev 2011;12(5):e426–37.
60. Anstey KJ, Mack HA, Cherbuin N. Alcohol consumption as a risk factor for dementia and cognitive decline: meta-analysis of prospective studies. Am J Geriatr Psychiatry 2009;17(7):542–55.
61. Anstey KJ, von Sanden C, Salim A, O'Kearney R. Smoking as a risk factor for dementia and cognitive decline: a meta-analysis of prospective studies. Am J Epidemiol 2007;166(4):367–78.
62. Shulman KI, Cohen CA, Kirsh FC, Hull IM, Champine PR. Assessment of testamentary capacity and vulnerability to undue influence. Am J Psychiatry 2007;164:722–7.
63. Brodaty H, Gresham M. Effect of a training programme to reduce stress in carers of patients with dementia. BMJ 1989;299(6712), 1375–9.
64. Brodaty H, McGilchrist C, Harris L, Peters KE. Time until institutionalization and death in patients with dementia. Role of caregiver training and risk factors. Arch Neurol 1993;50(6):643–50.
65. Mittelman MS, Ferris SH, Steinberg G, et al. An intervention that delays institutionalization of Alzheimer's disease patients: treatment of spouse-caregivers. Gerontologist 1993;33(6):730–40.
66. Marriott A, Donaldson C, Tarrier N, Burns A. Effectiveness of cognitive-behavioural family intervention in reducing the burden of care in carers of patients with Alzheimer's disease. Br J Psychiatry 2000;176:557–62.
67. Macnab J, Peisah C. Capacity toolkit: How do I decide whether a person has the capacity to make their own decisions? 2011. Available at http://gpnn.org.au/wp-content/uploads/2011/06/Jenna-Macnab-Capacity.pdf [accessed 02 April 2013].
68. Baile WF, Buckman R, Lenzia R, et al. SPIKES – a six-step protocol for delivering bad news: application to the patient with cancer. Oncologist 2000;5(4):302–11.
69. Mueller PS. Breaking bad news to patients. The SPIKES approach can make this difficult task easier. Postgrad Med 2002;112(3):15–16,18.
70. Jacoby R, Steer P. How to assess capacity to make a will. BMJ 2007;335(7611):155–7.
71. National Transport Commission Australia. Assessing fitness to drive for commercial and private vehicle drivers. 2013. Available at www.austroads.com.au/images/stories/assessing_fitness_to_drive_2013.pdf [accessed 02 April 2013].
72. Central Coast Division of General Practice. Advance care planning fact sheet. 2006. Available at www.nscchealth.nsw.gov.au/carersupport/resources/otherpublication/003772155.pdf [accessed 02 April 2013].

RESOURCES FOR DOCTORS

- GPCOG is available at www.gpcog.com.au, and is a useful, short cognitive assessment tool.
- The Geriatric Depression Scale is a useful screening tool for depression and is available at www.stanford.edu/~yesavage/GDS.english.short.score.html [accessed 27 March 2013].
- KICA is available at www.wacha.org.au/docs/misc/KICA-Tool.pdf [accessed 27 March 2013].
- Alzheimer's Australia provides culturally appropriate assessment tools, which are available at www.fightdementia.org.au/understanding-dementia/culturally-appropriate-dementia-assessment-tools-1.aspx [accessed 27 March 2013].
- The Dementia Collaborative Research Centre provides advice and copies of assessment tools for cognition, function and BPSD. It is available at www.dementia-assessment.com.au [accessed 27 March 2013].
- RUDAS is available at www.fightdementia.org.au/understanding-dementia/rowland-universal-dementia-assessment-scale.aspx [accessed 27 March 2013].
- PBS information on drugs can be found at www.pbs.gov.au/browse/medicine-listing [accessed 27 March 2013].
- The Caregiver Burden Scale can be found at www.dementia-assessment.com.au or at www.scfmresidency.com/SCFM_Curriculum/Neurology/Alzheimers_AAFP_2004.pdf [both accessed 27 March 2013].
- Alzheimer's Australia's brain health and dementia risk reduction program, Your Brain Matters at http://yourbrainmatters.org.au provides evidence-based information about strategies associated with better brain health, better cognitive function and reduced dementia risk.
- For a comprehensive summary of the state of the evidence for dementia risk reduction, see Targeting brain, body and heart for cognitive health and dementia prevention: current evidence and future directions. It can be found on the Alzheimer's Australia website, available at http://yourbrainmatters.org.au [accessed 27 March 2013].
- For a summary of the evidence, references and resources for GPs and patients for 12 dementia risk factors, see Dementia risk reduction: a practical guide for general practitioners. It can be found on the Alzheimer's Australia website, available at http://yourbrainmatters.org.au [accessed 27 March 2013].
- The AlzRisk database at www.alzrisk.org provides a comprehensive, publicly available collection of epidemiologic reports that evaluate environmental (i.e. non-genetic) risk factors for Alzheimer disease.
- Each state and territory offers state-based genetics services. These can be found by entering your state or territory and 'genetics services' into an internet browser search.
- The Australian Physical Activity Guidelines offer advice about appropriate levels of exercise for all Australians, and are available at www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-phys-act-guidelines [accessed 27 March 2013].
- The Australian Dietary Guidelines offer advice about diet and nutrition for Australians, and are available at www.nhmrc.gov.au/guidelines/publications/n55 [accessed 27 March 2013].
- WACHA e-ageing is a self-directed learning package for health professionals in rural and remote settings. It has 10 interactive modules on age-related care, including delirium. It is available at http://e-ageing.wacha.org.au/index.php?id=1521 [accessed 27 March 2013].
- The Clinical Practice Guidelines for the Management of Delirium in Older People are available at www.health.vic.gov.au/acute-agedcare [accessed 27 March 2013].
- A recent article in the *Australian Family Physican*, 'Frontotemporal dementia, features, diagnosis and management,' is a useful resource and overview of FTD for GPs.
- The article by P Shelton and K Rockweeod[40], How golden is the gold standard of neuropathology in dementia?, in the *References* section gives a good overview of dementia.
- See *check* January/February 2012, units 478/479 'Fitness to drive' and the Alzheimer's Australia website at www.fightdementia.org.au [accessed 27 March 2013].
- For information on advance planning, see The Advance Care Directive Association's website, www.advancecaredirectives.org.au, or contact them by phone or email on 0423 157 003 and info@advancecaredirectives.org.au.
- The Respecting Patient Choices – Advanced Care Planning website, available at www.respectingpatientchoices.org.au, provides resources and information on general and state-specific advanced care planning.
- The Office of the Public Advocate or Public Guardian in each state and territory provides a range of fact sheets that can assist patients and their families in understanding legal issues related to capacity, such as the legal requirements for power of attorney. For example, the New South Wales Government has created an excellent Capacity Toolkit, available at www.publicguardian.lawlink.nsw.gov.au/agdbasev7wr/publicguardian/documents/pdf/capacity_toolkit0609.pdf [accessed 27 March 2013], which uses case studies to illustrate the meaning and assessment of capacity.
- The *Australian Medico-Legal Handbook* published by Elsevier Health is an excellent resource for issues surrounding capacity and decision making. Also see AFP *Medicolegal handbook for general practice* by Sara Bird, published by the RACGP in 2006.

RESOURCES FOR PATIENTS

- The AlzRisk database (www.alzrisk.org) provides a comprehensive, publicly available collection of epidemiologic reports that evaluate environmental (i.e. non-genetic) risk factors for Alzheimer disease.
- Each state and territory offers state-based genetics services. These can be found by entering your state or territory and 'genetics services' into an internet browser search.
- The Australian Physical Activity Guidelines offer advice about appropriate levels of exercise for all Australians, and are available at www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-phys-act-guidelines [accessed 27 March 2013].
- The Australian Dietary Guidelines offer advice about diet and nutrition for Australians, and are available at www.nhmrc.gov.au/guidelines/publications/n55 [accessed 27 March 2013].
- Alzheimer's Australia's website provides resources and information about services, support and education to assist families and carers. It includes cultural diversity resources. It is available at www.fightdementia.org.au [accessed 27 March 2013].
- The Alzheimer's Australia National Dementia Helpline (1800 100 500) provides carer support, living with memory loss programs and other support services, including a Chinese helpline. Further information on the services provided by the helpline it is available at www.fightdementia.org.au/services/national-dementia-helpline.aspx [accessed 27 March 2013].
- Information about dementia-related safety issues can be found at www.fightdementia.org.au/services/safety-issues.aspx [accessed 27 March 2013].
- Culturally specific Chinese dementia resources can be found at the CASS. Their head office is located in New South Wales and they can be contacted via their website (www.cass.org.au) or by telephone 02 9789 4587.
- DBMAS provides a helpline (1800 699 799) and a website (www.dbmas.org.au) with information for carers and families of people with dementia.
- Carers Australia (www.carersaustralia.com.au) provides support and resources for carers.
- The Better Health Channel provides a good basic overview of dementia, covering many frequently asked questions. It is available at www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Dementia_explained [accessed 27 March 2013].
- Independent Living Centres in each state and territory provide information and resources to enable people to continue to live independently. Independent Living Centres Australia also provides a national helpline (1300 885 886).
- FRONTIER is a clinical research group in Sydney dedicated to the study of FTD and related disorders. They have a useful link for carers, available at www.neura.edu.au/frontier/carer-support [accessed 27 March 2013].
- The National Institute of Health website gives an excellent overview of FTD and is a useful resource for patients. It is available at www.nia.nih.gov/alzheimers/publication/frontotemporal-disorders-resource-list [accessed 27 March 2013].
- The Respecting Patient Choices – Advanced Care Planning website, available at www.respectingpatientchoices.org.au, provides resources and information on general and state-specific advanced care planning.
- The Office of the Public Advocate or Public Guardian in each state and territory provides a range of fact sheets that can assist patients and their families in understanding legal issues related to capacity, such as the legal requirements for power of attorney. For example, the New South Wales Government has created an excellent Capacity Toolkit, available at www.publicguardian.lawlink.nsw.gov.au/agdbasev7wr/publicguardian/documents/pdf/capacity_toolkit0609.pdf [accessed 27 March 2013], which uses case studies to illustrate the meaning and assessment of capacity.
- The Australian Institute of Health and Welfare released *Dementia in Australia* in 2012, which provides a comprehensive overview of dementia and the issues that may arise for both patients and carers. It is available for free download at www.aihw.gov.au/publication-detail/?id=10737422958 [accessed 27 March 2013].
- The Department of Health and Ageing website provides a list of resources (available at www.health.gov.au/internet/main/publishing.nsf/content/dementia-resources) and information (available at www.health.gov.au/internet/main/publishing.nsf/Content/dementia-information) about dementia for patients, families and carers [both accessed 27 March 2013].
- Aged Care Australia's website provides excellent information on aged care assessment teams, resources and support programs for people with dementia, their families and their carers. It is available at www.agedcareaustralia.gov.au/internet/agedcare/publishing.nsf/Content/Streaming+page [accessed 27 March 2013].

Dementia

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and
- log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au.

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.

FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

QUESTION 1

Jarli, an Indigenous man from a rural and remote community aged 67 years, is brought in to see you by his daughter Kirra. He has a history of hypertension, type 2 diabetes and cataracts. Kirra is concerned that Jarli seems increasingly forgetful. He is sometimes unable to name things and no longer enjoys family gatherings. You suspect Jarli may have dementia. What is the most appropriate assessment tool to screen for dementia in this situation?

A. MMSE
B. RUDAS
C. GPCOG
D. Abbreviated Mental Test Score (AMTS)
E. KICA.

QUESTION 2

Amy, aged 80 years, comes in for her annual influenza vaccine with her husband, Bob. He tells you that Amy has been forgetting the names of her grandchildren, she sometimes leaves the gas on the stove on and is having difficulty with dressing. Amy's MMSE last year was 27/30 when she had her annual health assessment. Her MMSE today is 24/30. You make a provisional diagnosis of dementia. What is currently the most common type of dementia?

A. Mixed dementia
B. AD
C. VaD
D. FTD
E. LBD.

QUESTION 3

Amy's husband Bob from *Question 2* is reluctant to accept a diagnosis of dementia and suggests that Amy may just be 'getting old'. What features in her history and examination need to be included to make a diagnosis of dementia?

A. A decline in multiple areas of cognition
B. Significant impairment in occupational functioning
C. Frequent falls
D. Daytime somnolence
E. Both A and B.

QUESTION 4

Bruce, aged 78 years, is admitted to hospital with respiratory distress. He is a smoker and has a history of hypertension and gout. His wife, Bev, calls you because she is worried about her husband. He looks flushed and seems agitated and distressed. Bev can't always understand what he is saying and his attention moves from one subject to another. He has already pulled out his IV twice. He is usually a keen reader and plays golf regularly. You suspect Bruce may have delirium. What are the features of delirium?

A. Acute onset with inattention, disorganised thinking and an altered level of consciousness
B. Progressive memory loss
C. Visual hallucinations, parkinsonism and fluctuations in cognition
D. Autonomic instability and sensitivity to neuroleptic agents such as haloperidol
E. RSBD.

QUESTION 5

Lynette, aged 50 years, comes to see you. Her widowed mother, Mavis, was diagnosed with mixed AD and VaD 6 months ago. Mavis is no longer able to live independently and has just moved into a nursing home. Lynette has found it distressing to see her mother lose her memory. She asks if there is anything she can do to avoid developing dementia when she gets older. What are some lifestyle factors that appear to be protective against dementia?

A. A varied and healthy diet
B. Physical activity
C. Mental and social activity
D. Control of vascular risk factors (diabetes and hypertension)
E. All of the above.

QUESTION 6

Mary, aged 75 years, is brought to your clinic by her daughter Jane, who wants to know whether her mother is able to change her will. Mary was diagnosed with AD 4 years ago, and stopped driving her car 2 years ago. She has been in a residential aged-care facility for 12 months. Six months ago, Mary gave power of attorney to Jane. Which of the following is correct regarding Mary's capacity to change her will?

A. Mary cannot change her will because she has AD.
B. Mary's capacity to change her will depends on her MMSE score.
C. Mary's capacity to change her will depends on her clock-drawing ability.
D. Mary's capacity to change her will depends on her ability to understand the issues involved.
E. Mary cannot change her will because Jane has power of attorney.

QUESTION 7

Which of the following is NOT true of ACDs and plans of care?

A. An ACD about future treatment decisions is only used when a person loses capacity to make their own decisions about healthcare.
B. An ACD about future treatment decisions is made while the person has decision-making capacity.
C. A plan of care about future treatment decisions can only be made while the person has decision-making capacity.
D. A plan of care is made by relatives, carers and medical staff of a person who does not have decision-making capacity. It may involve the person.
E. An ACD is useful to have in place for people with dementia.

QUESTION 8

Joan, aged 80 years, is brought to see you by her daughter Cathy. Cathy says that Joan has been growing more forgetful over the past 3 years. Joan lives at home with her husband, John, and they have meals delivered. Joan used to be a keen gardener, but has lost interest. She also does not read as much as she previously did. She repeatedly asks the same questions, despite seeming to listen to the replies each time. She has trouble recalling names of objects and people. You refer Joan to a memory clinic where a diagnosis of AD is made. What stage of AD does Joan have?

A. MCI
B. Mild AD
C. Moderate AD
D. Severe AD
E. Profound AD.

QUESTION 9

Rob comes to see you to discuss whether he and his siblings should have genetic testing for suspected familial AD. His mother and aunt both developed AD in their 50s, although their two brothers are still living, aged 75 and 78 years, with no symptoms of dementia. Rob's grandmother on his mother's side had dementia early in life. He is unsure of other relatives. What is the mode of transmission of familial AD? Of Rob and his three siblings, how many would be expected to develop the disease?

A. Autosomal dominant; 2 of 4
B. Autosomal dominant; 1 of 4
C. Autosomal recessive; 2 of 4
D. Autosomal recessive; 1 of 4
E. X-linked recessive; males only.

QUESTION 10

Julie, aged 48 years, comes into your clinic. She has had testing overseas and is carrying one APOE ε4 allele. Julie wants to know what this means in relation to her risk of developing AD. Which of the following is true?

A. Those with one APOE ε4 allele will definitely develop AD.
B. APOE ε4 is not a risk factor for AD.
C. Some people with APOE ε4 will not develop AD.
D. Those with APOE ε4 do not have a greater risk of developing AD at a younger age.
E. APOE ε4 is not a AD susceptibility gene.

check

Independent learning program for GPs

Unit 495 June 2013

Imaging

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This unit of *check* on radiological imaging looks at the most up-to-date and current techniques used for the diagnosis of common problems in general practice. Advances in technology mean that we now have imaging modalities that are less invasive, limit patients' exposure to ionising radiation and have better visualisation. Another benefit of new imaging modalities is that some can be used for therapeutic interventions and may be less invasive than traditional methods.

We would like to thank the authors, who are all specialists at MIA Radiology, a member of I-MED Radiology Network, for providing a wealth of information on current imaging techniques for this unit of *check*.

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The learning objectives of this unit are to:

- develop increased confidence in using appropriate imaging techniques to investigate breast pathology
- assess postmenopausal bleeding as well as identify risk factors for endometrial carcinoma
- identify the red flags in patients presenting with headache
- list the imaging modalities used for different brain pathologies
- use evidence-based guidelines to determine the probability of a patient having a pulmonary embolism
- discuss the different types of non-traumatic knee pain presenting in a young adolescent
- list the causes, investigation and treatment of greater trochanteric pain sydrome
- develop improved competence in the diagnosis and management of shoulder pain.

We hope this edition of *check* will be helpful in selecting the most appropriate investigation for your patients.

Kind regards

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GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

AC	acromioclavicular	ELISA	enzyme linked immunosorbent assay	OA	osteoarthritis
AP	anterior posterior	FNA	fine needle aspiration	PD	proton density
BIH	benign intracranial hypertension	GTPS	greater trochanteric pain syndrome	PE	pulmonary embolism
BMI	body mass index	IIH	idiopathic intracranial hypertension	PIOPED	Prospective Investigation of Pulmonary Embolism Diagnosis
BP	blood pressure	ITB	iliotibial band	SE	stress echocardiogram/ echocardiography
CAD	coronary artery disease	LV	left ventricle	SS	supraspinatus
CC	craniocaudal	MBS	Medicare Benefits Schedule	UOQ	upper outer quadrant
CSF	cerebrospinal fluid	MLO	mediolateral oblique	V/Q	ventilation/perfusion
CT	computed tomography	MPI	myocardial perfusion imaging		
CTPA	CT pulmonary angiography	MRI	magnetic resonance imaging		
CVST	cerebral venous sinus thrombosis	MRV	magnetic resonance venogram		
ECG	electrocardiogram	NSAID	non-steroidal anti-inflammatory drug		

CASE 1

JANE IS WORRIED ABOUT BREAST CANCER

Jane, aged 36 years, presents to you having discovered a thickening with tenderness in her right breast on self-examination. She has not had any previous breast imaging. Jane's mother died of breast cancer aged 52 years, and Jane's older sister had treatment for breast cancer aged 40 years and is currently alive and well. Jane has a son and a daughter, and has no current health issues. She is not on any medication.

When you examine Jane you find symmetrical areas of thickening in the upper outer quadrant (UOQ) of both breasts, with no discrete lumps. There is no lymphadenopathy in the axillae. General examination reveals no abnormalities.

QUESTION 1

What is the most appropriate initial set of investigations?

QUESTION 2

Jane asks about going to BreastScreen. What do you recommend?

QUESTION 3

Jane is concerned about the radiation dose from a mammogram. What do you tell her?

FURTHER INFORMATION

Jane agrees to have a mammogram and ultrasound. The mammogram shows breast density of >75% with an area of subtle distortion in the left UOQ, but no other abnormality (see *Figure 1*). On ultrasound, the area of concern within both lateral breasts corresponds with normal dense breast tissue, and there are scattered simple cysts within both breasts (category 2). There are two further small, irregular hypoechoic masses more laterally within the left axillary tail, each measuring about 5–6 mm in diameter and 2 cm apart (category 4). There is also a left axillary lymph node with prominent cortical thickness of 3.1 mm (see *Figure 2*). Core biopsy of the two irregular breast masses and fine needle aspiration (FNA) of the axillary node are recommended.

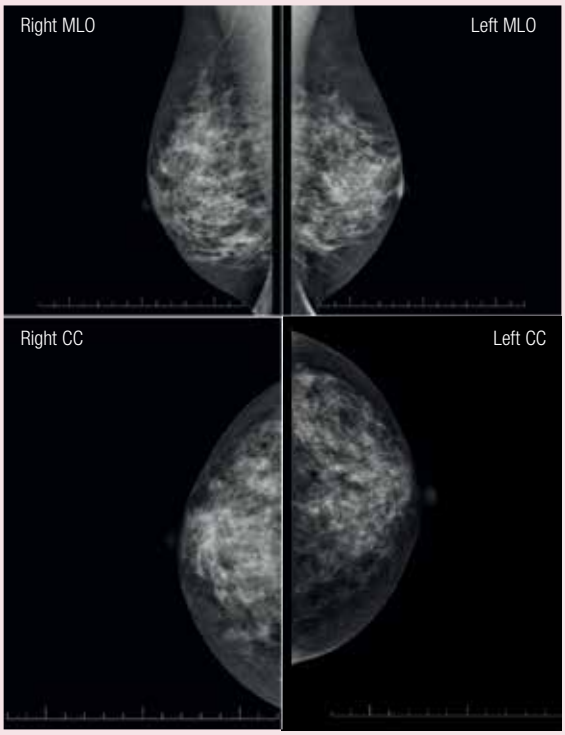


Figure 1. Bilateral mammogram showing dense fibroglandular tissue and subtle architectural distortion in the axillary tail on the left mediolateral oblique (MLO) projection. The craniocaudal (CC) view is also shown.

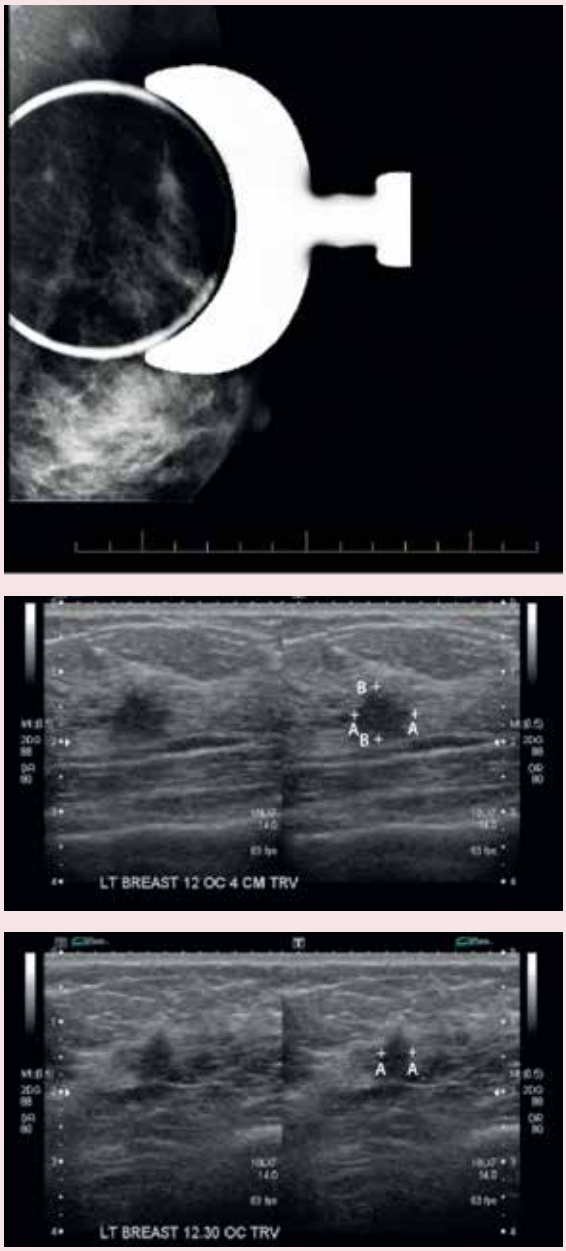


Figure 2. Work-up mammographic view showing two small spiculate masses. In the bottom and middle images, ultrasound also demonstrates two small, ill-defined hypoechoic masses with suspicious features.

QUESTION 4

What is the relevance of the categories 1–5 in breast imaging synoptic reports?

QUESTION 5

What is the relevance of breast density as stated in the mammogram report?

FURTHER INFORMATION

You refer Jane to a breast surgeon and she subsequently undergoes a breast core biopsy and axillary node FNA, both under ultrasound guidance, at a local radiology practice.

Both breast masses are invasive lobular cancers, but the axillary node is normal.

Further imaging is performed and CT and bone scans are clear of metastasis.

Jane elects to undergo bilateral mastectomy and reconstruction because of her strong family history of breast cancer and the imaging findings. Following the surgery she will take anti-oestrogen therapy but will not require any other adjuvant treatment.

QUESTION 6

Jane has a younger sister, Lily, aged 30 years. What action would you advise her to take regarding her breast health?

CASE 1 ANSWERS

ANSWER 1

Jane should have a diagnostic bilateral mammogram and ultrasound. If Jane had been younger than 35 years, an ultrasound would have been the first investigation, and a mammogram would have been performed when the suspicious masses were seen in the left breast. Women younger than 35 years are more likely to have dense breasts, therefore it is less likely that abnormalities will be detected on mammogram. When there is a breast symptom, a targeted breast ultrasound should always be performed.[1]

ANSWER 2

BreastScreen is a screening program and is not an appropriate place to investigate a breast symptom. A screening program assumes no symptoms. There is increased wait time for results and biopsy, and an ultrasound is only performed if the woman is recalled for assessment of a mammographic abnormality.

You tell Jane that she needs both mammogram and ultrasound, and that this is better undertaken at a diagnostic imaging facility.

ANSWER 3

Digital mammography requires an extremely low dose of radiation to acquire good quality images. The average radiation dose for two images of each breast (four images in total) is around 0.7 mSv, or the equivalent of approximately 4 months of environmental background radiation. For a woman younger than 40 years, this suggests a lifetime additional risk of cancer of between 1:10 000 and 1:100 000 per examination. This compares with the general risk of one in three of developing any cancer in a woman's lifetime.[2]

ANSWER 4

A standardised, management-based classification system for breast imaging findings will help to prevent interpretation errors and should improve the appropriate management of women with breast symptoms.[3]

- Category 1: no significant imaging abnormality (does not preclude biopsy of any clinically suspicious area)
- Category 2: benign findings (does not preclude biopsy of any clinically suspicious area)
- Category 3: indeterminate/equivocal findings. Requires further investigation (e.g. FNA/core biopsy). Management should be based on the outcomes of a triple test (clinical examination, imaging and biopsy or FNA). There may be a limited role for early follow-up
- Category 4: findings suspicious of malignancy. Requires further investigation. May require excisional biopsy
- Category 5: findings are consistent with malignancy. Requires further investigation (e.g. biopsy).

ANSWER 5

Breast density relates to the proportion of fibroglandular tissue within the breast, which varies considerably among women. In young women, breast tissue is highly glandular, dense and looks white on a normal mammogram. As women age, the glandular tissue generally becomes involuted and is replaced by fatty tissue. As cancers and other breast masses are white on a mammogram, the greater the background density, the lower the sensitivity for cancer detection on mammogram alone. Ultrasound is therefore of increasing importance to aid in the detection of cancer in denser breast tissue.[1]

ANSWER 6

Lily is considered at high risk of developing breast cancer because she has more than two primary relatives (mother and two sisters) who have developed the disease. Her risk of developing breast cancer is 3–6 times the normal female population, with a 25–50% lifetime risk of developing breast cancer. Lily should be referred to a cancer specialist or family cancer clinic for risk assessment, possible genetic testing and management plan. Ongoing surveillance strategies may include regular clinical breast examination, breast imaging with mammography, MRI or ultrasound and consideration of ovarian cancer risk. Individualised surveillance program may include regular clinical breast examination, and annual breast imaging with mammography, MRI or ultrasound.[4]

CASE 2

BARBARA HAS INTERMITTENT VAGINAL BLEEDING

Barbara, aged 70 years, presents with intermittent vaginal bleeding over the past 3 months. She has a history of breast cancer, which was treated with tamoxifen for 5 years. Barbara also has a history of obesity, hypertension and diabetes. She lives with her husband, John; they have no children.

QUESTION 1

What is the differential diagnosis for postmenopausal uterine bleeding?

QUESTION 2

What are the risk factors for endometrial carcinoma?

QUESTION 3

What imaging tests should you order for Barbara?

QUESTION 4

What is the risk of endometrial carcinoma for a patient on tamoxifen therapy?

CASE 2 ANSWERS

ANSWER 1

Causes of postmenopausal uterine bleeding include endometrial atrophy, benign endometrial hyperplasia, benign endometrial polyps, submucous uterine fibroids, oestrogen withdrawal and endometrial carcinoma. Of women presenting with postmenopausal bleeding, 10% will have an underlying malignancy such as endometrial carcinoma, atypical endometrial hyperplasia and cervical carcinoma.[5] Endometrial atrophy is the commonest cause of postmenopausal uterine bleeding.[6]

ANSWER 2

Risk factors for endometrial carcinoma include prolonged administration of unopposed oestrogen hormone replacement therapy (more than 5 years), tamoxifen use, hereditary non-polyposis colorectal carcinoma, obesity combined with diabetes, hypertension and exogenous or endogenous increased oestrogen.[7] Nulliparity, late menopause, early menarche and polycystic ovaries have also been implicated as risk factors. Pregnancy and the use of oral contraceptives reduce risk.

It is important to remember that 12% of endometrial cancer occurs in pre-menopausal women.[8]

In addition to being a risk factor for endometrial carcinoma, tamoxifen treatment is also associated with increased incidence of endometrial polyps and hyperplasia.[9]

ANSWER 3

Transvaginal ultrasound is the investigation of choice for women with postmenopausal uterine bleeding. An endometrial thickness of greater than 4 mm requires further investigation with an endometrial biopsy.[10] There is no benefit for ultrasound screening of asymptomatic women, even in high-risk groups such as women taking tamoxifen.[11]

Saline infusion sonohysterography is a transvaginal ultrasound performed after distension of the endometrial cavity with fluid via a fine cervical catheter. The hypoechoic fluid in the endometrial cavity provides contrast with the echogenic endometrial lining, which allows differentiation between diffuse endometrial pathology such as endometrial hyperplasia and focal pathology such as endometrial polyps or carcinoma (see *Figure 3*).[12]

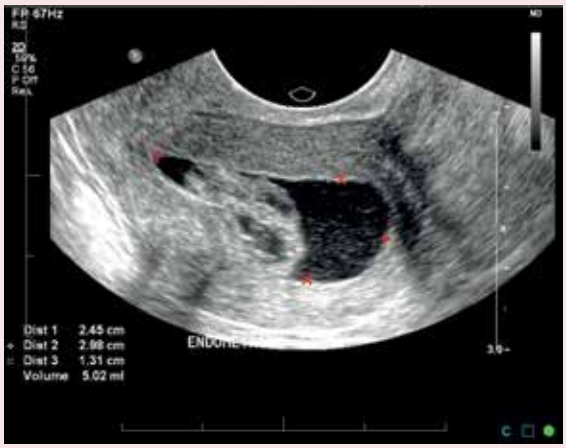


Figure 3. Sagittal ultrasound image of the uterus showing a polypoid endometrial carcinoma outlined by hypoechoic fluid (bright focus surrounded by black).

Endometrial hyperplasia affects the whole endometrium, causing diffuse thickening of greater than 5 mm. Endometrial polyps appear as focal endometrial thickening, occasionally with a vascular stalk being identified. Endometrial carcinoma may appear as endometrial thickening, a polypoid lesion or an irregular mass (see *Figures 4* and *5*).[13]

Ultrasound cannot reliably distinguish between benign endometrial hyperplasia and malignancy.[14]

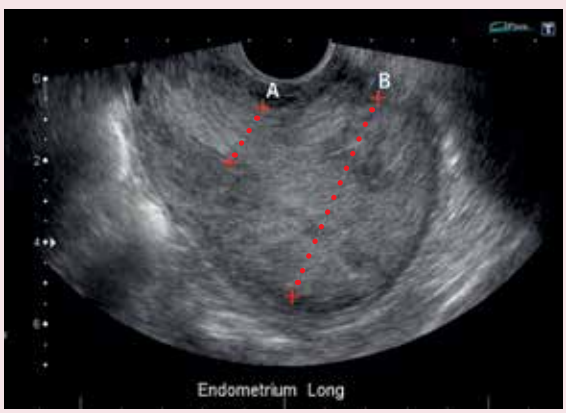


Figure 4. Sagittal ultrasound of the uterus showing irregular endometrial thickening caused by carcinoma. Thinning of the myometrium suggests myometrial invasion by carcinoma (A = lower uterine segment; B = uterine fundus).

Figure 5. Sagittal ultrasound image of the uterus showing a polypoid uterine carcinoma (outlined by callipers on the image).

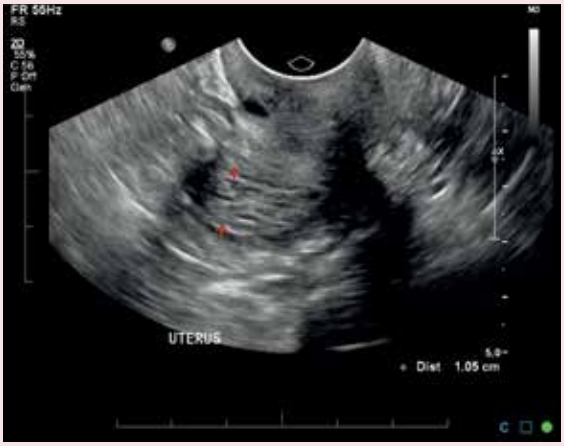


Figure 6. Sagittal ultrasound image of the uterus showing a thickened endometrium with multiple cystic spaces (between the callipers).

Endometrial biopsy is performed by a gynaecologist when there is diffuse endometrial thickening, focal endometrial thickening greater than 5 mm or an irregular endometrial mass. Definitive diagnosis of carcinoma can only be obtained by endometrial sampling, either with endometrial biopsy or dilatation and curettage.

Endometrial carcinoma spreads first by local invasion, followed by lymph node spread. Haematogenous spread is a late occurrence. CT scanning is of limited use in assessment of local invasion into the myometrium; it is used for assessment of distant metastases.[15]

MRI can be used to detect myometrial invasion, extra-uterine extension or pelvic lymphadenopathy.[16]

ANSWER 4

Tamoxifen is a selective oestrogen receptor modulator that has been used in the treatment of hormone receptor–positive breast cancer for over 30 years. It binds to oestrogen receptors, acting both as an oestrogen antagonist (in breast tissue) and an oestrogen agonist (in uterus and bone).[17] Tamoxifen increases the risk of developing endometrial carcinoma, with an increase in relative risk of 6.9 over 5 years of treatment.[18] Tamoxifen-associated carcinoma has an unfavourable prognosis compared with spontaneously developing endometrial carcinoma. Even taking this into account, the benefits of treatment with tamoxifen outweigh the risks, with the life expectancy of women with hormone receptor–positive breast cancer improved in those taking tamoxifen.[18]

Tamoxifen is also associated with an increased incidence of endometrial hyperplasia.[19] The endometrial hyperplasia associated with tamoxifen treatment typically has a cystic appearance on ultrasound (see *Figure 6*).

CASE 3

EMMA HAS WORSENING HEADACHES

Emma, aged 28 years, is an obese woman who presents to your clinic with worsening headaches over the past few months. Over the past week she has developed some mild blurred vision, but has not experienced any loss of vision. Emma has been married to Mark for 6 years. They have a daughter, Julie, aged 3 years.

QUESTION 1

In regard to headache presentations generally, what features on history and examination would raise a red flag and prompt early imaging investigation?

FURTHER INFORMATION

When you examine Emma, you find little of clinical significance except when you look at her fundus. Fundoscopy reveals papilloedema. You send Emma for an MRI, the results of which are shown in *Figures 7–9*.

Figure 7. Axial T2 fat suppressed orbits showing tortuous dilated optic nerve sheaths bulging into the back of the orbital globes – the imaging equivalent of papilloedema.

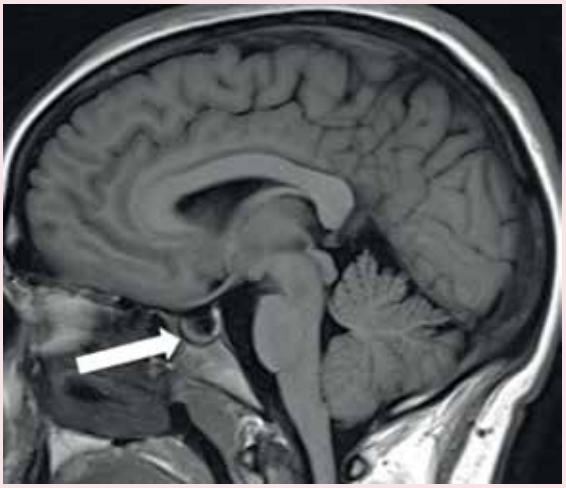


Figure 8. Sagittal T1 showing the pituitary gland flattened down, a feature known as 'partially empty sella'; this is a less specific sign of raised intracranial pressure.

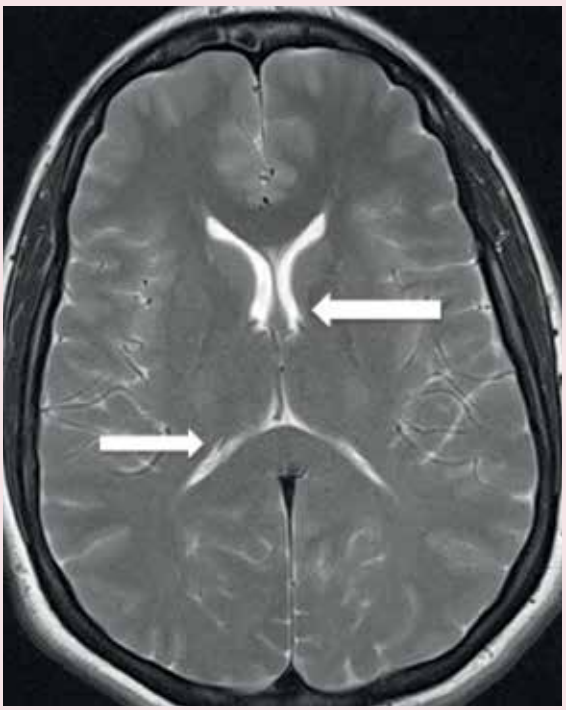


Figure 9. Axial T2 brain demonstrating normal or possibly reduced ventricle size, which excludes hydrocephalus as a cause of Emma's headaches.

QUESTION 2

What are the main differential diagnoses given these features?

QUESTION 3

How should you treat Emma?

QUESTION 4

What is the imaging investigation of choice for excluding cerebral venous sinus thrombosis (CVST)?

QUESTION 5

What imaging should be done in cases of suspected meningitis?

QUESTION 6

What initial imaging investigation is recommended for suspected giant cell arteritis?

CASE 3 ANSWERS

ANSWER 1

The features on history and examination that would raise red flags are outlined below.

- Headache type: thunderclap, different from usual migraine, positional or orthostatic headache or headache that is worsened by Valsalva, headache causing wakening, or headache that is progressively worsening
- Patient demographic: elderly, pregnant or postpartum, history of malignancy, trauma or immunocompromise
- Other signs: fever, neck stiffness, mental state alteration (personality or conscious state), neurological deficit, first episode of seizure, papilloedema
- Drug ingestion: anticoagulants, illicit drugs, immunosuppressants.[20,21]

ANSWER 2

There are two differential diagnoses for Emma's clinical and MRI findings.

1. Venous obstruction or CVST.
2. Idiopathic intracranial hypertension (IIH), which is also known as pseudotumour cerebri, and benign intracranial hypertension (BIH).

Emma's MRI shows dilated optic nerve sheaths with tortuosity bulging into the back of the orbital globes consistent with fundoscopic findings of papilloedema/raised intracranial pressure. There is an absence of venous obstruction/thrombosis or increased mass effect such as from brain oedema, hydrocephalus or tumour.

Thus, Emma's diagnosis is consistent with IIH, which is a diagnosis of exclusion.

Features of IIH include the following.

- Dural venous stenosis is a frequent finding and may be part of the underlying cause.
- It is most commonly seen in obese women of childbearing age. The other group that is affected is older men (usually non-obese), who are at twice the risk of vision loss.
- The incidence is approximately 1 per 100 000 per year; however, in women aged between 20 and 44 years who are 10% above ideal body weight, the incidence is 13 per 100 000 and in those 20% above ideal body weight, it is 19 per 100 000. Prevalence rates are higher, reflecting the chronic nature of the condition in many cases.[22]

ANSWER 3

Emma will need urgent initial assessment by an ophthalmologist to accurately determine whether she has significant alteration in her vision. As Emma has had only minimal blurred vision, her management is likely to consist of weight loss and medication such as acetazolamide to reduce cerebrospinal fluid (CSF) production.

Emma will need ongoing regular ophthalmology reviews. If her vision deteriorates, more aggressive measures such as shunt placement, venous sinus stenting or fenestration of the optic nerve sheaths may be indicated.

ANSWER 4

A combination of MRI and MR venogram (MRV) is the test of choice to exclude CVST for the following reasons. (See *Figures 10–12* for images of a patient with CVST with cerebral oedema secondary to venous hypertension.)

- MRI, in general, is more sensitive for detection of CVST at each stage after thrombosis; however, without the venographic component the diagnosis might not be excluded.
- While a CT scan is often the initial imaging choice by a GP or in an emergency department, non-contrast CT is only abnormal in approximately 30% of CVST cases.[23]
- Cerebral oedema as a complication of venous hypertension is much better appreciated on MRI than CT, and is often evident.
- MRV has the added benefits of not requiring contrast and of having no ionising radiation.
- If MRI is not available, venographic study using CT would be recommended.
- CVST accounts for 0.5–1% of strokes.[24]

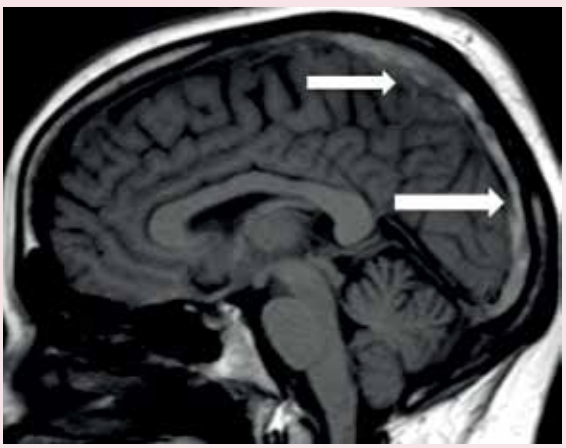


Figure 10. Sagittal T1 showing high signal within the superior sagittal sinus, which is confirmed on MRV in *Figure 15*.

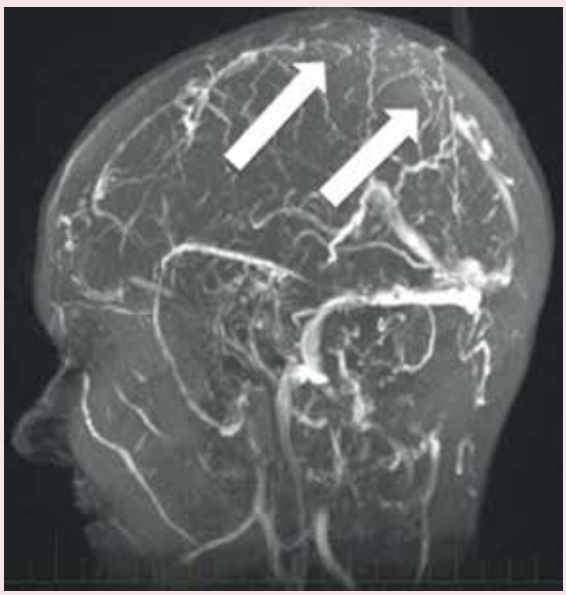


Figure 11. Sagittal MRV time of flight of venous sinus thrombosis with the thrombosed superior sagittal sinus seen as a defect/loss of continuity in the sinus (arrows).

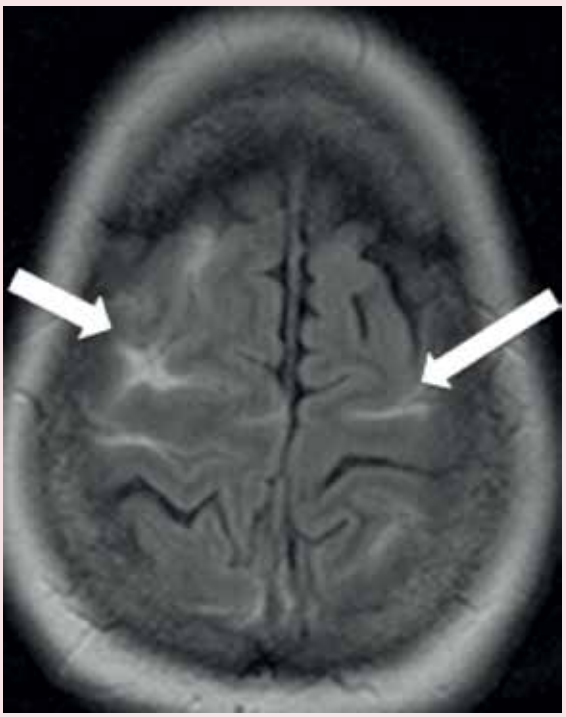


Figure 12. Axial flair – the bright areas represent brain oedema secondary to venous hypertension.

ANSWER 5

If meningitis is suspected, the taking of blood cultures and commencement of antibiotics should not be delayed by the use of imaging.[25]

Lumbar puncture should be deferred for 1 to 2 days in patients who are clinically very ill or likely to have raised intracranial pressure (e.g. altered conscious state, focal neurological deficit, papilloedema). Brain herniation from lumbar puncture may occur in the presence of a normal CT scan of the brain.[26]

MRI brain may be indicated (without delaying initial treatment) at some point if complications are suspected (e.g. CVST, intracranial abscess, immunocompromised patient).

ANSWER 6

Imaging is generally not indicated for patients with suspected giant cell arteritis. Diagnosis is based on a combination of clinical suspicion, inflammatory markers and temporal artery biopsy. Both MRI and ultrasound can be useful in equivocal or challenging cases, and can be used to help guide biopsy.

CASE 4

MATTHEW HAS A SORE KNEE

Matthew, aged 14 years, presents to your surgery with a 4-month history of pain in his left knee. He is very active and does some form of exercise on most days. He is a keen swimmer and plays water polo and cricket at school. He is otherwise well. He occasionally gets hay fever and had eczema as a child. He can't remember any injury to his knee. He has no history of knee pain prior to this episode. Matthew describes the pain as being made worse by activity. Occasionally the pain wakes him from sleep. He describes the pain as being 'all over' his knee and he is unable to pinpoint one discrete area of tenderness.

On examination, Matthew is afebrile. His BMI is 21. The skin over his left knee is normal and there is no discolouration. There is possibly some mild swelling anterior to the left tibial tuberosity, which is a little painful to palpation. Matthew tells you that the pain is not localised to just that spot and seems to be all over his knee. There is no mass palpable and his knee movements are slightly restricted.

QUESTION 1

What are the possible causes of Matthew's knee pain?

QUESTION 2

What investigations, if any, would you suggest? What are the advantages and disadvantages of the different imaging modalities?

QUESTION 3

What does the plain X-ray film in *Figure 13* show?

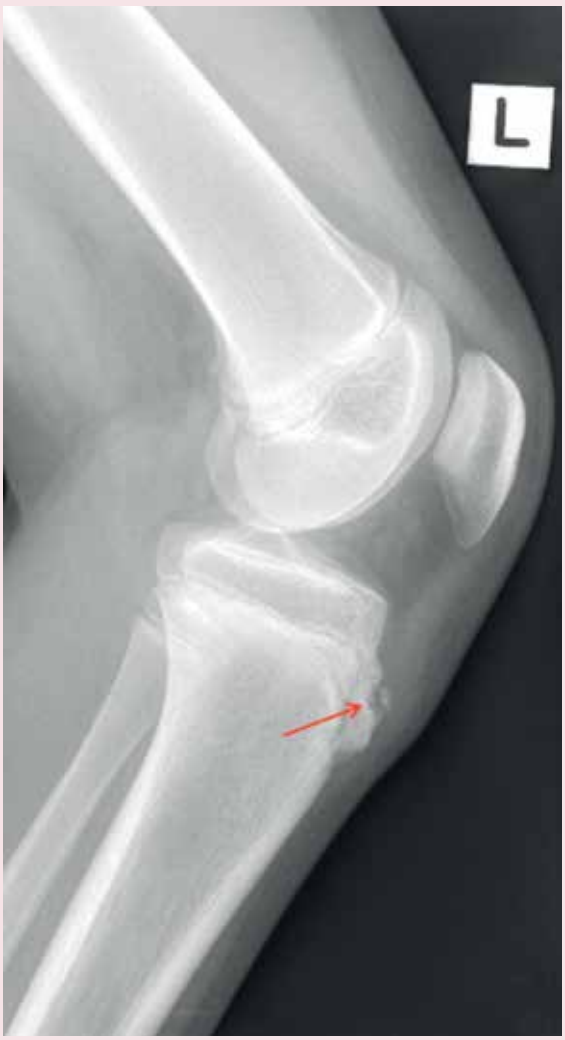


Figure 13. X-ray showing a lateral view of Matthew's left knee.

FURTHER INFORMATION

Matthew returns 6 months later with persistent, hard-to-localise knee pain.

QUESTION 4

What imaging, if any, would you recommend?

FURTHER INFORMATION

Matthew decreased his activity level as well as having an arthroscopic drilling procedure to stimulate healing. One year later his pain had resolved and a follow-up MRI (see *Figure 14*) showed almost complete healing.

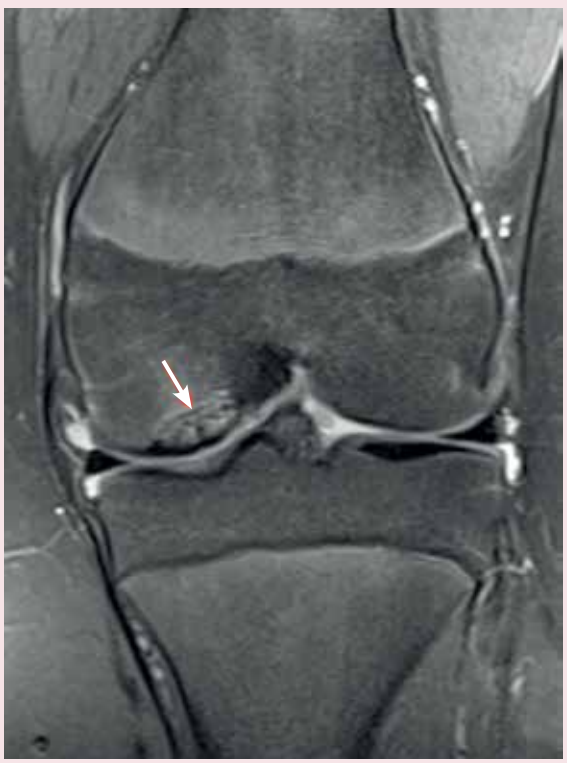


Figure 14. Resolution of osteochondral defect on MRI imaging.

CASE 4 ANSWERS

ANSWER 1

The causes of non-traumatic knee pain in a young adolescent are:[27]

- Osgood–Schlatter disease/Sinding–Larsen–Johansson disease
- osteochondral defect (osteochondritis dissecans)
- meniscal tear
- patellofemoral instability or maltracking
- bipartite patella
- synovitis – mostly post-viral
- aggressive causes such as osteomyelitis and osteosarcoma (rare).

ANSWER 2

Matthew warrants further investigation because he presents with a history of night pain and slight limitation of movement, which are not usually present in Osgood–Schlatter disease. While Osgood–Schlatter disease is the most common cause of knee pain in this age group it is important to exclude other pathology.

A plain X-ray film (anterior posterior (AP) and lateral) of the knee will provide bone detail and exclude tumour and fracture, but is unable to show causes of internal derangement of the knee.

Ultrasound does not have a role as it can only demonstrate a knee effusion. It cannot show other pathology within the knee, apart from further definition of a palpable mass if one is present.

CT is inappropriate as it only gives bone detail and does not show the muscles, tendons, ligaments, cartilage or menisci. It gives no detail of bone marrow oedema and exposes the patient to unnecessary radiation.

MRI will show articular cartilage, ligaments and tendons as well as bone detail, including the presence or absence of bone marrow oedema. It can be used for growth plate assessment and demonstrating synovial pathology within the joint, such as a knee joint effusion or synovial thickening.

ANSWER 3

The lateral view of the knee on plain X-ray in *Figure 13* shows some fragmentation of the tibial tuberosity, which is characteristic of Osgood–Schlatter disease.[28] Osgood–Schlatter disease is an osteochondrosis of the patellar tendon insertion onto the tibial tubercle. It affects patients aged 10–15 years, with up to 50% of cases being bilateral. It is likely to be secondary to chronic avulsive injury of the patellar tendon on the tibial tubercle. The chondro-osseous junction is considered the weakest component in the immature skeleton and injury often occurs here, rather than disrupting the patellar tendon itself.[27,29]

Osgood–Schlatter disease is a clinical diagnosis and doesn't require imaging unless there are unusual features such as night pain or limitation of movement. If MRI is performed it often shows a thickened patellar tendon, oedema in the fat surrounding the patellar tendon and also fragmentation of the tibial tuberosity.

Osgood–Schlatter disease is treated conservatively with advice to reduce high-level activities. It is a self-limiting condition that resolves spontaneously around the time of growth plate fusion.

ANSWER 4

The initial investigation is a repeat X-ray (see *Figure 15*) to assess bone detail and check for any changes. An irregularity of the medial femoral condyle is shown, probably an osteochondral defect.

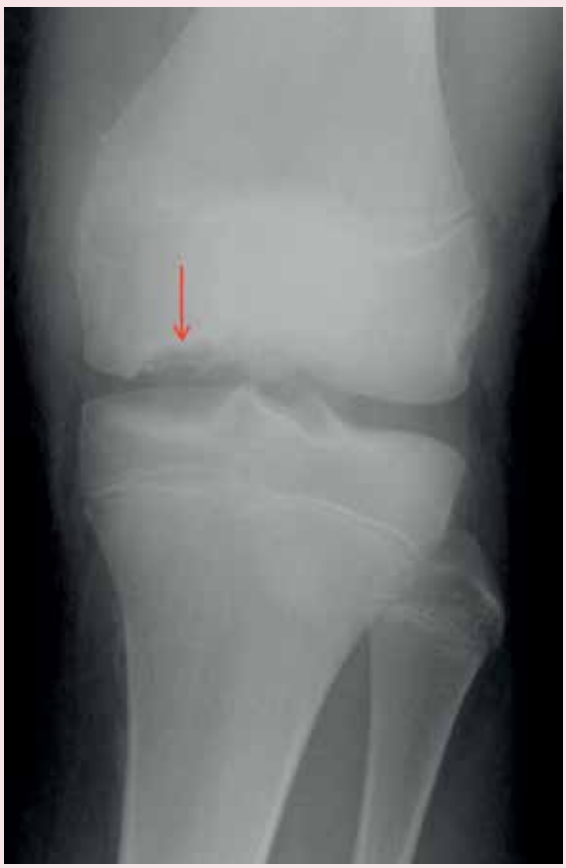


Figure 15. AP X-ray film of the knee demonstrates a lucency (arrow) in the medial femoral condyle that is consistent with an osteochondral defect.

MRI is now indicated as there are ongoing symptoms that are not consistent with Osgood–Schlatter disease.

MRI (*Figure 16*) shows a focus of abnormality of the medial femoral condyle with bone marrow oedema radiating away from it into the underlying bone. The overlying articular cartilage is intact, however the lesion has some findings of developing instability on MRI criteria.[30]

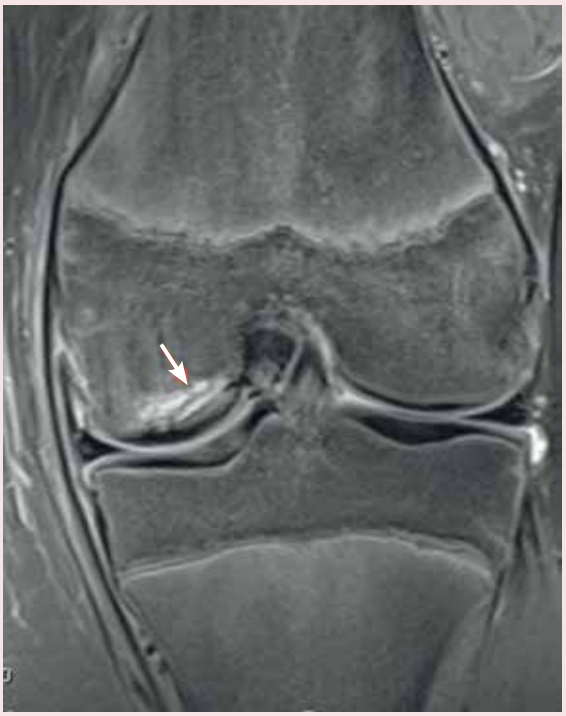


Figure 16. Coronal proton density (PD) MRI scan demonstrating that the plain X-ray film lucency in *Figure 14* corresponds to the bright focus within the medial femoral condyle in keeping with an ostechondral defect. There is a high signal cleft deep to the lesion and a few cysts (arrow), which are features of lesion instability.

Osteochondral defects occur most commonly in patients aged between 10 and 20 years and are more common in males. They often appear to be related to chronic traumatic injury, and less commonly to acute trauma.[31,32] Children with this condition often present with poorly localised knee pain that continues for more than a year. Typically, the pain increases with exercise.[33]

An osteochondral defect involves the lateral aspect of the medial femoral condyle in 75% of patients, and is bilateral in one-third of cases.[27] These subchondral bone defects or fragments may be only partially attached so they are unstable and prone to detachment, or they may be attached with fibrous tissue. MRI is useful in identifying lesion instability by showing a high signal interface between the defect and the femur.[30] A high T2 signal cleft deep to the lesion and cyst formation are used as signs of developing instability.[34] Lesion stability is important when deciding on treatment options, and this information cannot be obtained with a plain X-ray. Gadolinium enhanced studies have been used and can possibly differentiate between instability and stability,[35] but are not routinely used as usually the findings are clear on non-gadolinium studies.

Treatment of stable osteochondral lesions on MRI criteria includes an initial period of non-weight-bearing followed by a graded return to normal activity. Follow-up MRI and plain X-ray are used to monitor progress. The patient often has continued low-grade discomfort that eventually resolves. If the osteochondral defect is unstable there are a number of treatment options available. These include arthroscopic drilling to promote healing, in situ pinning of the bone fragment or chondrocyte reimplantation.[33,36]

CASE 5

SIMONE HAS CHEST PAIN

Simone, aged 21 years, is a bookkeeper who presents to your surgery with a sudden onset of chest pain when she breathes in. She tells you that she has been coughing up some blood. She also says that her right leg has been painful, swollen and mildly red for 1 week leading up to her current presentation. She lives at home with her parents and is currently not on any medication. She is allergic to penicillin. She recently travelled to the United States for a holiday.

On examination she is afebrile. Her BP is 110/70 mmHg, her heart rate is 80 beats per minute and regular, and her respiratory rate is 20 breaths per minute. Her right calf is slightly swollen with mild erythema and is tender to gentle palpation. Her chest is clear on auscultation. A pulse oximeter shows that she is mildly hypoxic with an oxygen saturation of 91% on room air.

QUESTION 1

What is the most likely diagnosis? What differential diagnoses need to be considered?

QUESTION 2

What are the risk factors for developing pulmonary embolism (PE) in a young patient? What importance should be placed on overall pre-test probability before performing imaging?

QUESTION 3

What is the most appropriate investigation for Simone?

QUESTION 4

Which tests would be the most appropriate if Simone told you that she was pregnant?

QUESTION 5

What are some newer imaging modalities in the diagnosis of PE?

CASE 5 ANSWERS

ANSWER 1

The most important diagnosis to exclude is PE. It is important to rapidly diagnose PE as it is associated with a high mortality rate. Of patients diagnosed with PE, 17.4% will die within 3 months of diagnosis[37] and delayed treatment may lead to significant morbidity as well as the development of secondary pulmonary hypertension.[38]

Other causes of pleuritic chest pain in a young adult include pneumothorax, pneumonia, pericarditis, as well as infective, inflammatory and musculoskeletal causes.

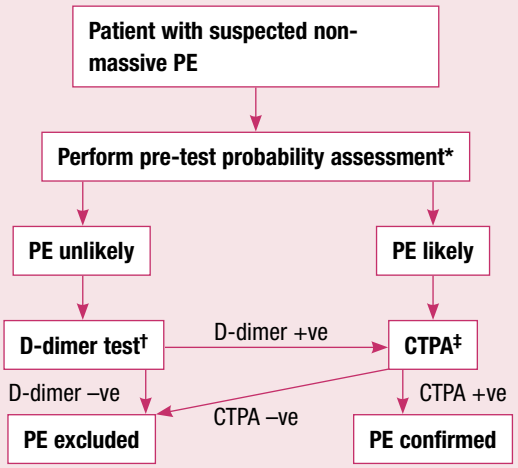
ANSWER 2

Risk factors for PE in a young patient include recent travel, pregnancy and use of the oral contraceptive pill. Other risk factors include recent surgery, malignancy and coagulation disorders.

Assessment of pre-test probability such as the Wells Pulmonary Embolism Score (see *Table 1*) in combination with biochemical testing with D-dimer (see *Figure 17*) is important in determining the most appropriate imaging pathway. The investigation of PE is based on recommendations from the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) II trial.[39]

Table 1. Simplifed Wells Pulmonary Embolism Score

Variable	Points
Clinical signs and symptoms of deep vein thrombosis (minimum of leg swelling and pain on palpation of the deep veins)	3.0
Alternative diagnosis less likely than pulmonary embolism	3.0
Heart rate >100 beats per minute	1.5
Immobilisation (>3 days) or surgery within the previous 4 weeks	1.5
Previous pulmonary embolism or deep vein thrombosis	1.5
Haemoptysis	1.0
Malignancy (receiving treatment, treated in last 6 months or palliative)	1.0
Clinical probability of pulmonary embolus unlikely: score ≤4 points Clinical probability of pulmonary embolus likely: score >4 points This table originally appeared in: McRae S. Pulmonary embolism. Aust Fam Physician 2010;39(7):462–6.	



* Assess pre-test probability according to simplifed Wells score (*Table 1*)

† Algorithm applies if D-dimer test used is a high sensitivity test (e.g. sensitivity >95%)

‡ Ventilation/Perfusion (V/Q) scanning can be used as an alternative imaging test in patients with renal impairment, or where there are concerns regarding radiation exposure (e.g. young women, particularly during pregnancy). If CT pulmonary angiography (CTPA) is technically inadequate, V/Q scanning can also be performed.

Figure 17. Algorithm for the diagnosis of PE. This chart orignally appeared in: McRae S. Pulmonary embolism. Aust Fam Physician 2010;39(7):462–6.

ANSWER 3

The most appropriate investigation for Simone depends on her pre-test probability, her clinical stability, imaging availability and potential risks of radiation exposure and/or iodinated contrast material.[39]

Current suggested imaging pathways are based on recommendations from the PIOPED II trial.[40] Following risk evaluation using the Wells or Geneva scoring system, a rapid enzyme linked immunosorbent assay (ELISA) D-dimer is performed. For patients with low or moderate pre-test probability with a negative D-dimer, no further investigation or treatment is necessary.[39] For patients with a positive D-dimer, further imaging investigation should be performed. In the high pre-test probability group no D-dimer testing needs to be performed – imaging investigation is suggested as first-line evaluation.[39]

The most easily assessable imaging investigations include chest X-ray, venous Doppler ultrasound, V/Q lung scan (see *Figure 18*) and CTPA (see *Figures 19*, *20* and *21*). Older tests such as catheter pulmonary angiography are invasive, associated with high radiation dose[39] and carry a risk of severe complications,[41] and are, therefore, not commonly performed.

Chest X-ray carries low sensitivity and specificity for the detection of PE, with the classic Westermark sign (oligaemia of the affected region of the lungs with decreased vessel diameter and density) seen in only 2% of cases.[42] Chest X-ray is useful in identifying other causes of pleuritic chest pain.

Venous Doppler ultrasound carries high sensitivity for the detection of symptomatic deep vein thrombosis,[43] but sensitivity is lower in the setting of PE for detection of above-knee thrombosis. The detection of a thrombus does not necessarily prove PE, and it is hence limited as a stand-alone test.[43]

The PIOPED II recommendations favour the use of CTPA over V/Q scanning for all pre-test probabilities where further investigation is required.[39] However, CTPA requires good renal function and the use of iodinated contrast material; in some situations V/Q scanning may be more appropriate.

CTPA should not be used with patients who have an allergy to iodinated contrast material and in those with impaired renal function. In these scenarios, venous Doppler ultrasound and V/Q scanning are recommended.

While the PIOPED II study recommends CTPA over V/Q scanning for women of reproductive age, the latter may be appropriate in patients suspected of having PE in the setting of a normal chest X-ray.[44] This may, therefore, be an appropriate investigation for Simone as it is associated with a lower radiation dose.[39]

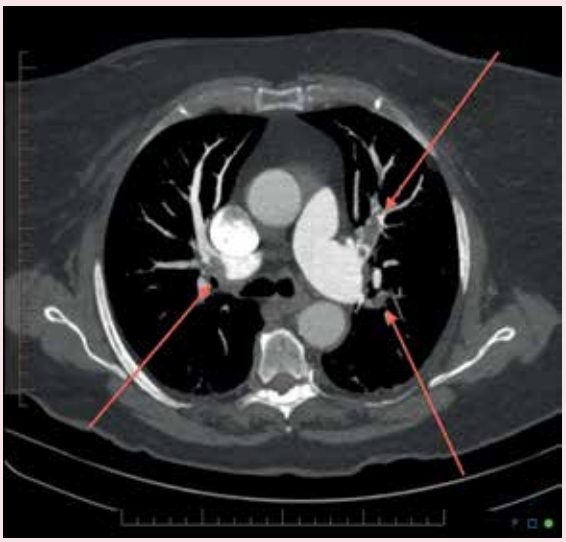


Figure 19. Axial CTPA demonstrates multiple large filling defects in major proximal pulmonary arterial branches, in keeping with acute PE.

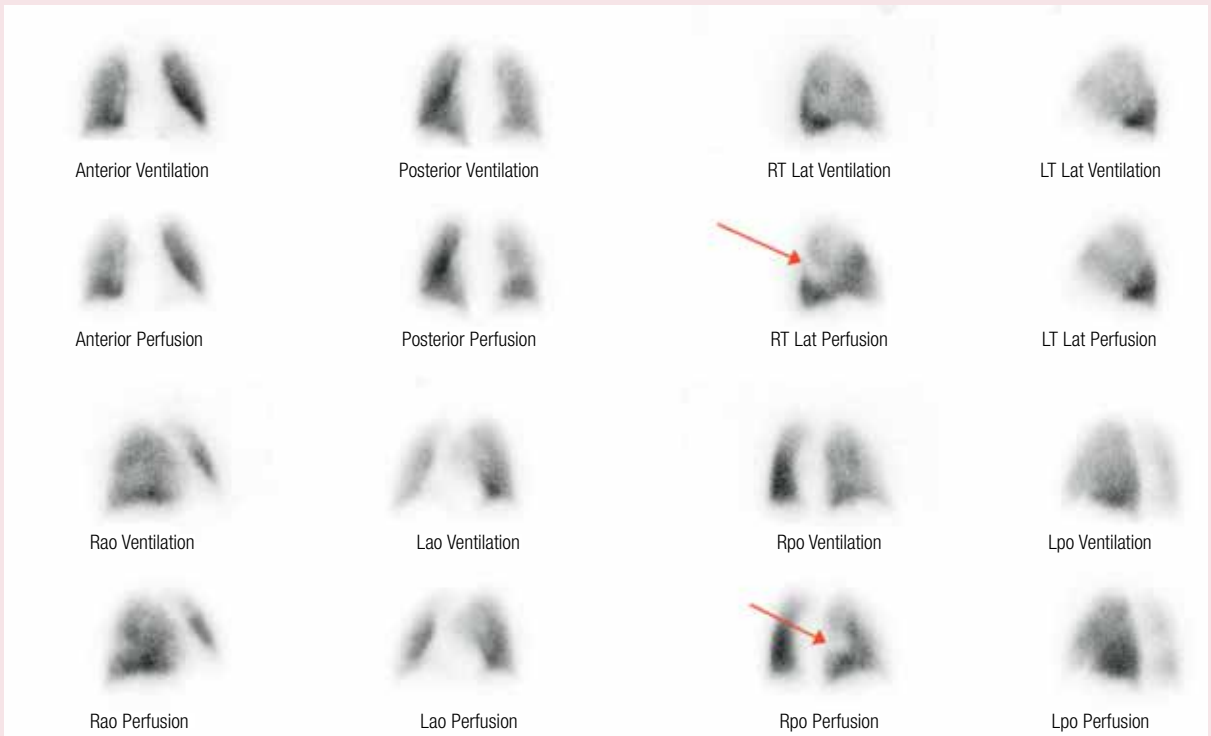


Figure 18. V/Q scanning demonstrates multiple unmatched segmental defects in keeping with PE. The largest perfusion defect is in the right mid zone.

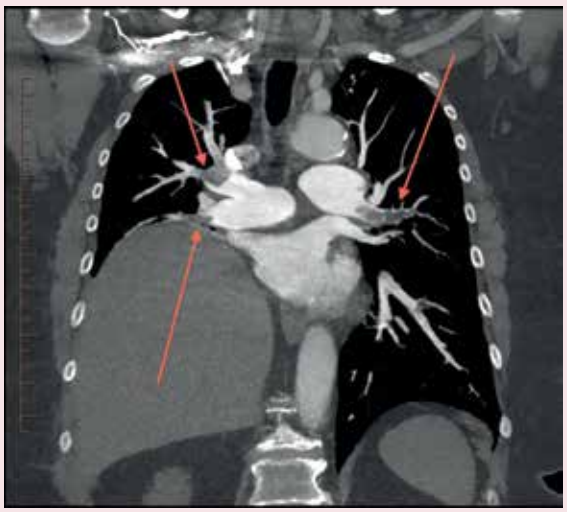


Figure 20. Coronal CTPA once again demonstrates multiple large filling defects in keeping with large PE.

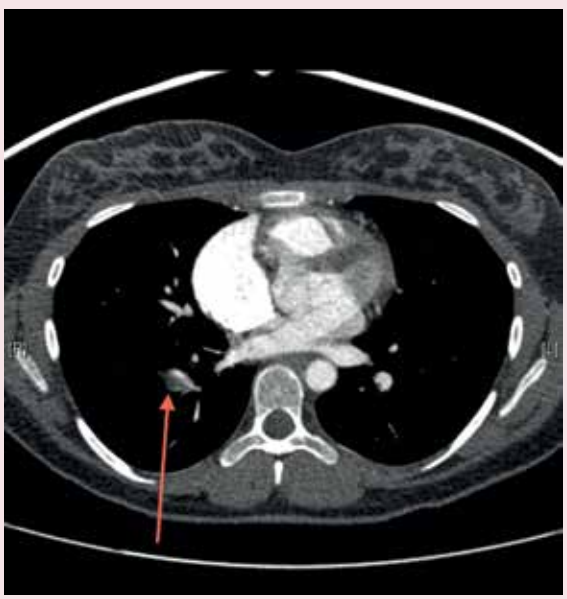


Figure 21. CTPA demonstrates a filling defect to the right lower lobe in keeping with acute PE.

ANSWER 4

The PIOPED II study recommends an initial D-dimer test for pregnant patients. If this is positive then a venous Doppler ultrasound is recommended.[39] If further imaging is required, V/Q is usually preferred over CTPA[39] because it results in a lower level of whole-body radiation. The overall foetal dose may be similar.[45] This recommendation is controversial, as a recent study showed that foetal dose may be lower with CTPA than with V/Q scanning.[46]

It is worth performing a D-dimer test during pregnancy[47] as this may avoid further investigation if it is negative. D-dimer is usually normal in the first trimester and rises in the second and third trimesters before falling to normal levels in the postpartum period.[48]

Venous Doppler ultrasound is a useful initial test due to the absence of ionising radiation. In the pregnant patient, ultrasound will detect thrombus in 13–15% of those suspected of having PE[49,50] and in 29% who are later found to have PE.

ANSWER 5

Newer imaging modalities, such as dual energy CT (*Figure 22)* and MR pulmonary angiography, are emerging and may play a greater role in the detection of PE in the future.

In addition to standard CTPA images, iodine perfusion maps can be created with dual energy CT, thereby increasing the ability to detect smaller subsegmental PE,[51] which has been difficult with standard CTPA.

Gadolinium enhanced MR angiography is an emerging technique evaluated in the recent PIOPED III trial, where it showed promising results. The advantage of using MR over CTPA or V/Q scanning is the absence of ionising radiation. MR may play a greater role in the diagnosis of PE in the future.

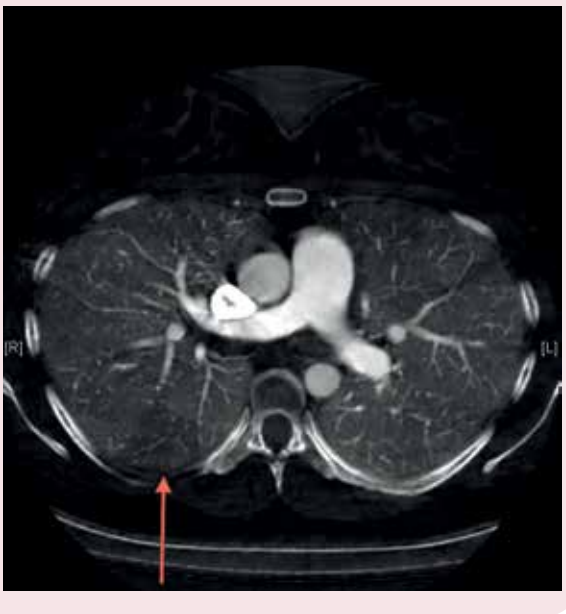


Figure 22. Approximately 2 years later, a follow-up to the CTPA shown in *Figure 21* was undertaken using dual energy CT with iodine perfusion mapping, showing a persistent wedge-shaped perfusion defect. This represents an area of hypoperfusion that may represent segmental infarction.

CASE 6

ERIN HAS PAIN IN HER HIP

Erin, aged 60 years, presents to your surgery with pain in her right hip. She has had the pain for several months now and she has noticed that it seems to be worse when she lies on her right side. She had a rotator cuff injury in her left shoulder 2 years ago that resolved with physiotherapy. She works as a receptionist at a nearby hospital and is usually fit and well. She walks on most days, is a non-smoker and only has the occasional glass of wine. On examination she has a full range of hip movement but she has focal tenderness over the greater trochanter. She has a good range of movement in her back and straight leg raising is normal on both sides. You make a clinical diagnosis of greater trochanteric pain syndrome (GTPS).

QUESTION 1

List three possible causes of GTPS.

QUESTION 2

Which patient group is most commonly affected by GTPS?

QUESTION 3

Which imaging modalities are most useful in the investigation of GTPS?

FURTHER INFORMATION

You refer Erin for an MRI to determine the cause of her chronic right hip pain. The results of her MRI are shown in *Figures 23* and *24*. You confirm the diagnosis of GTPS.

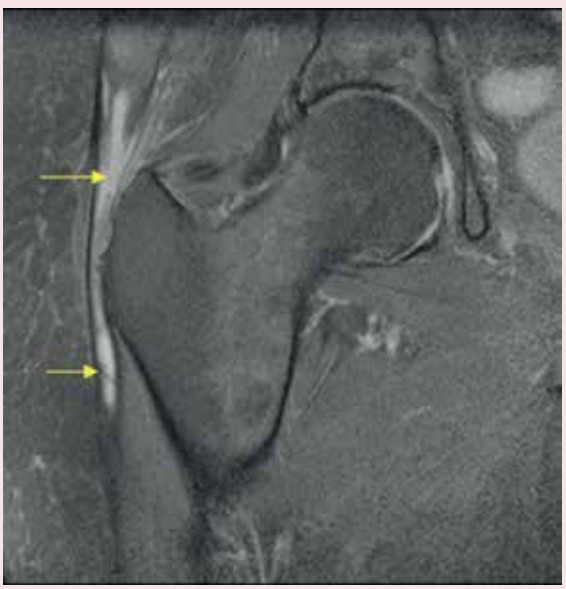


Figure 23. Coronal PD weighted MRI image with fat saturation of the right hip demonstrating bright fluid distending the greater trochanteric bursa (arrows).

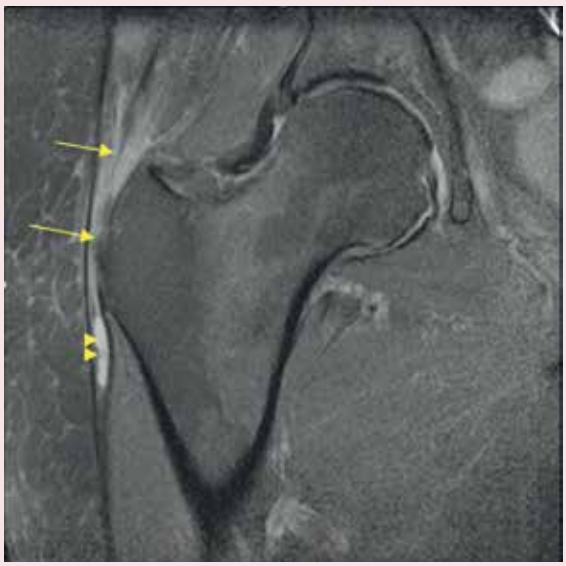


Figure 24. Coronal PD weighted MRI of the right hip with fat saturation demonstrating thickening and increased signal in the gluteus medius tendon at its insertion with focal discontinuity of the tendon fibres (arrows) indicative of tendinopathy and a partial thickness tear at the insertion. Bright fluid again demonstrated in the bursa (arrow heads).

QUESTION 4

What treatment options are available for Erin in the management of her GTPS? Does imaging have a role in the treatment of GTPS?

CASE 6 ANSWERS

ANSWER 1

GTPS has recently replaced the term 'trochanteric bursitis' to describe patients with chronic pain accompanied by reproducible tenderness in the region of the greater trochanter, buttock and lateral thigh. The reason for this is that the pain and tenderness can have many different causes including, but not confined to:

- trochanteric bursitis
- tendinopathy and tears of the gluteus medius and minimus tendons
- iliotibial band (ITB) disorders
- general or localised pathology in the surrounding tissues.[52]

With the advent of high resolution ultrasound and MRI it is becoming more apparent that tears of the gluteus medius and minimus tendons may be the leading cause of GTPS[53] and that trochanteric bursitis is commonly secondary to pathologies in the gluteus medius and minimus tendons.[54] Isolated distension of the trochanteric bursae is uncommon, and does not usually occur in the absence of gluteus medius pathology.[53]

FEEDBACK

GTPS is an important diagnosis to make, as the pain in this syndrome can refer down the lateral thigh, anterior groin and buttock, and can thus mimic the pain of nerve root compression in the lumbar spine or osteoarthritis (OA) of the hip.[52,54]

To complicate matters, patients with pre-existing low back pain and OA of the hip and knee are predisposed to developing GTPS because of altered lower limb biomechanics and abnormal force vectors across the hip joint.[55] Failure to make the diagnosis can result in costly patient referrals, inappropriate diagnostic testing and surgery that may not resolve the pain.[52]

GTPS is associated with significant impairment to the activities of daily living,[56] so an accurate diagnosis and treatment is of great benefit to the patient.

The anatomy around the greater trochanter has been likened to the 'rotator cuff of the hip'.[57] The gluteus medius and minimus tendons insert onto the greater trochanter while the adjacent trochanteric bursae, which vary in size, location and number, provide cushioning for the gluteal tendons, ITB, and the tensor fascia lata, with tension in the ITB causing repetitive friction.[54] This results in a similar spectrum of pathologies affecting the tendons and bursae as seen in the rotator cuff of the shoulder. The subgluteus maximus bursa is located lateral to the greater trochanter, between the gluteus medius tendon and the gluteus maximus muscle, and is the most frequently incriminated bursa in GTPS.[58]

Tears of the gluteal tendons and injury to the bursa can also occur with trauma.[54] In a setting of acute trauma it is important to exclude femoral fracture as a cause of pain.

Bursitis and tendon pathology can be caused by infection, especially tuberculosis, as well as crystal deposition diseases and inflammatory arthropathies such as rheumatoid arthritis.[59]

ANSWER 2

GTPS is more common in middle-aged and elderly women.[52] It has been found to be more common in patients with coexisting low back pain, knee OA, ITB tenderness and obesity. Again, an alteration in gait and altered biomechanics may be a contributing factor.[55]

GTPS is a common condition with an incidence in primary care of 1.8 patients per 1000 per year.[56]

In the younger population GTPS can also occur in athletes, particularly in runners and those performing step aerobics.[60,61]

ANSWER 3

Ultrasound and MRI are the most useful imaging modalities in the investigation of GTPS.

Ultrasound is well suited to assessing the structures around the greater trochanter due to their superficial nature. It is readily available and safe. Tendinopathy of the gluteal tendons is characterised by thickening, decreased echogenicity and sometimes increased vascularity within the tendons. Tears, which can be partial or full thickness, appear as focal discontinuities of the tendon fibres.

Trochanteric bursitis may demonstrate thickening of the bursa and/or fluid within the bursa. The ITB can be assessed for thickening and for fluid deep to it. As ultrasound is performed in real time, the exact anatomical site of the patient's focal tenderness can be determined, enabling confirmation of the clinical diagnosis of GTPS.

The hip joint can also be assessed for the presence of an effusion.

Ultrasound can be used to guide aspiration of fluid in the bursa and to inject local anaesthetic and corticosteroids into the bursa.

MRI can also be used to image the structures around the greater trochanter. A recent review determined that it had the highest correlation with surgical and clinical findings in patients with GTPS.[62]

On PD weighted images, normal tendons appear low in signal (i.e. black). Tendinopathy is characterised by thickening and increased signal (i.e. whiter and brighter) in the tendon. Tendon tears appear as focal loss of continuity of the tendon fibres with a bright fluid signal in the defect. Bursitis demonstrates thickening of the wall of the bursa and may demonstrate bright fluid distending the bursa. These pathological tissue changes and fluid collections characterised by increased signal on the PD images can be made more conspicuous by applying a fat saturation pulse, which removes the bright signal from adjacent fat.

MRI scanning will also enable an accurate assessment of the hip joint and assist in excluding other conditions such as stress fractures and avascular necrosis of the femoral neck. It is important to diagnose these conditions, especially in patients with risk factors for them, to avoid a misdiagnosis of GTPS.

ANSWER 4

Erin can be treated conservatively with non-steroidal anti-inflammatory drugs (NSAIDs), ice, weight loss and physical therapy.[58]

Erin has a 6-week course of physiotherapy and applies ice regularly. She is not keen to use NSAIDs as she developed epigastric discomfort 2 years ago when they were prescribed for her shoulder injury. She wants to know if there is any other treatment available. You suggest that she could have an ultrasound guided injection into the greater trochanteric bursa. There is evidence to support the use of a steroid injection into the bursa, with a three-fold increase in recovery at 5 years.[56] Landmark guided injections are effective in 77% of patients 1 week after injection and 61% of patients 6 months post-procedure.[63]

The technique involves placing the patient on the examination table on their side in the lateral decubitus position with the affected side uppermost. As the bursae are relatively superficial structures, they are readily accessible. Using a sterile technique the needle tip is inserted into the affected bursa under ultrasound guidance. The bursa is injected with corticosteroid and a local anaesthetic. Surgery may be considered for patients with intractable pain.[60]

FEEDBACK

Erin undergoes a trochanteric bursa injection under ultrasound guidance. An image of her trochanteric bursa before injection is shown in *Figure 25*.

Erin comes to see you 6 weeks after the procedure. She tells you she has had a significant decrease in her pain. Six months later, Erin is pain free (*Figure 26*).

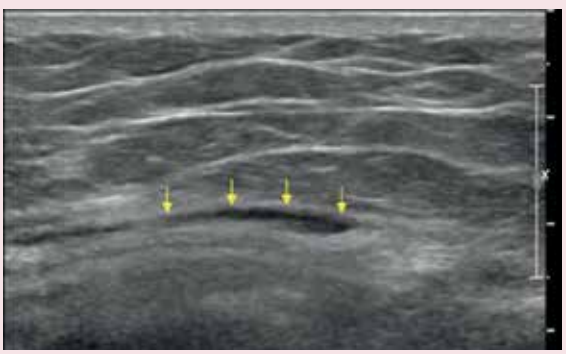


Figure 25. Ultrasound image demonstrates fluid distending the trochanteric bursa (arrows).

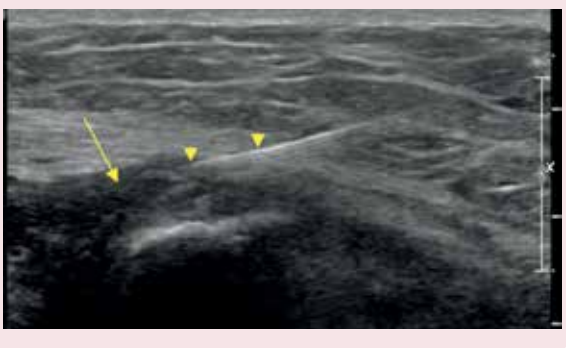


Figure 26. Ultrasound image demonstrates thickened hypoechoic gluteus medius tendon (arrow) with needle tip (arrow heads) in the trochanteric bursa.

CASE 7

MICHAEL HAS SHOULDER PAIN

Michael, aged 40 years, is an administrative assistant who presents with a sore right shoulder. He has a desk job but is a keen amateur sportsman and has recently started training for an upcoming 'pier to pub' swim. His pain is preventing him swimming freestyle and reaching out for objects on his bedside table. He is eager to have the condition diagnosed and treated because he would like to continue his training.

On examination his BP is 145/80 mmHg and his BMI is 23.5. He is afebrile. He has a painful arc of abduction between 25° and 70° but is able to 'push through' the pain and is pain free when he fully abducts his right shoulder. There is no weakness in his rotator cuff muscles and he has a good range of movement in his cervical spine.

QUESTION 1

What is your provisional diagnosis?

FURTHER INFORMATION

Michael has a painful arc of abduction and a positive Hawkins test[64] (pain on axial rotation of an already 90° abducted shoulder) as well as a positive Neer test[65] (pain with forced forward elevation against the acromion).

QUESTION 2

Do you need to do any imaging at this stage?

QUESTION 3

How does shoulder impingement occur?

FURTHER INFORMATION

Michael has completed a course of anti-inflammatory tablets and is doing physiotherapy, but when he comes to see you for a follow-up appointment, he complains of gastric symptoms and says physiotherapy is making his pain worse.

QUESTION 4

Given Michael's feedback, you decide to organise imaging. What tests would you order?

QUESTION 5

What information will they give you?

FURTHER INFORMATION

An ultrasound demonstrates a painful arc of abduction as the supraspinatus (SS) tendon passes beneath the coracoacromial ligament and mild SS tendinopathy. You decide that a bursal injection of cortisone is likely to help reduce Michael's pain and enable him to do his physiotherapy exercises properly. Ultrasound guided injection is a simple, safe and effective technique for getting the steroid into the right place (see *Figure 27*).

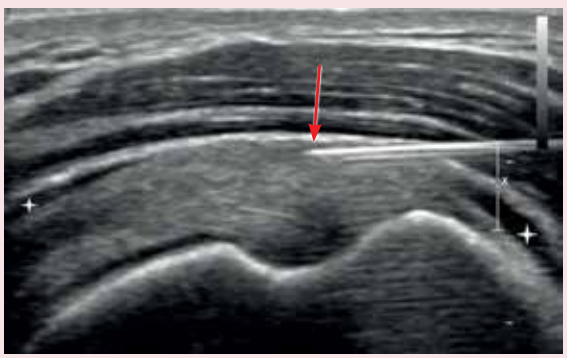


Figure 27. Needle (arrow) injecting fluid into the subacromial bursa. The injected fluid appears black (stars).

QUESTION 6

What are the potential side effects of cortisone injections that you need to warn Michael about?

FURTHER INFORMATION

At Michael's follow-up appointment, the radiology report states, and Michael confirms, that his painful arc of abduction was relieved by the anaesthetic component of the injection. This helps confirm the diagnosis of impingement.

QUESTION 7

Suppose Michael's ultrasound had shown a 15 mm focus of soft calcification within the SS tendon and he called you the afternoon following the ultrasound describing sudden onset of unrelenting pain in his shoulder when he was lifting shopping out of the car. What would you think had happened?

CASE 7 ANSWERS

ANSWER 1

The most likely diagnosis is rotator cuff tendinopathy or impingement syndrome involving his subacromial bursa and the rotator cuff tendons. If Michael has had long-standing niggling shoulder pain that has been exacerbated by the new training regimen, the possibility of calcific tendinopathy or bursitis should be considered.

In this age group a rotator cuff tear or glenohumeral arthritis are unlikely without a specific traumatic event, but Michael could have strained his acromioclavicular (AC) joint. His history is not typical of a labral tear.

ANSWER 2

If your clinical evaluation confirms the diagnosis of impingement or rotator cuff tendinopathy then Michael does not need imaging unless he fails to respond to treatment.

ANSWER 3

Shoulder impingement is caused by the rotator cuff tendons and bursa rubbing as they pass below the subacromial arch during shoulder abduction.[65] This usually occurs secondary to subtle imbalance of the glenohumeral joint with the head moving forward in the socket on abduction to trap these structures. This is why appropriate physiotherapy exercises are vital to realign the joint and make more space for the tendons to pass beneath the coracoacromial arch. Impingement is made worse if the tendons and bursa become enlarged because of this rubbing, or if a focus of calcification develops and expands the tendons. Impingement may develop if there is not enough space for the tendons to fit through the arch due to an acromial bone spur or due to expansion of the AC joint secondary to arthritis.

ANSWER 4

You would order a shoulder X-ray and ultrasound. MRI is not the first-line modality for investigation of impingement. Note that benefits for shoulder ultrasound are only payable when referral is based on the clinical indicators outlined in the Medicare Benefits Schedule item descriptions. No benefits are payable if the referrable is for non-specific shoulder pain.

ANSWER 5

While an X-ray is usually normal in this age group, it is able to show the shape of the acromion, subacromial bone spurs (see *Figure 28*), soft tissue calcification, AC joint OA and misalignment. It may also reveal the unexpected, such as glenohumeral arthritis or a bone tumour.

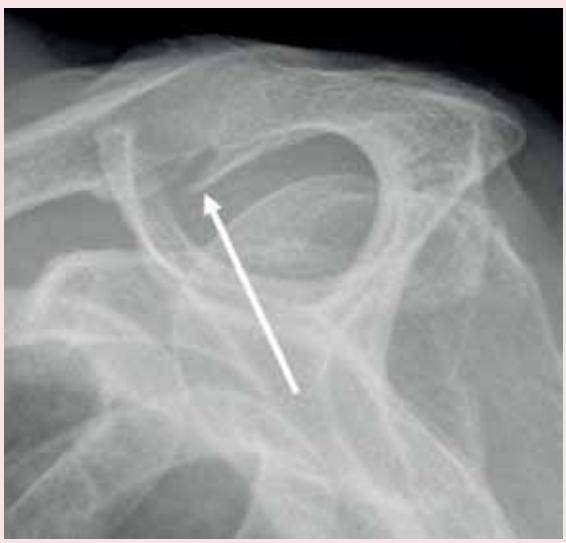


Figure 28. Small subacromial bone spur (arrow) that may rub the SS tendon. Consider surgical referral if symptoms are non-responsive to conservative therapy.

Dynamic ultrasound evaluation is the mainstay of the diagnosis of shoulder impingement. The principle criterion is observation of mechanical compression of the SS tendon and/or bursa beneath the coracoacromial arch on abduction combined with the clinical observation that this elicits patient pain.[66] Other signs include bunching or fluid distension of the bursa[67] and bunching or crowding of the SS tendon as it tries to pass beneath the coracoacromial arch.[68] Dynamic ultrasound can confirm, but not exclude, the diagnosis of impingement as false negative results of up to 18% are reported.[69] This may occur because the impingement is inactive at the time of examination or masked by anti-inflammatory or pain relief medication.

Ultrasound will demonstrate the cause of impingement such as tendinopathy, calcification in the tendon, tears of the tendon and bursitis (see *Figure 29*). A thick bursa or painless bursal bunching should never be interpreted as a sign of impingement as they can be present in up to 30% of asymptomatic shoulders (see *Figure 30*).

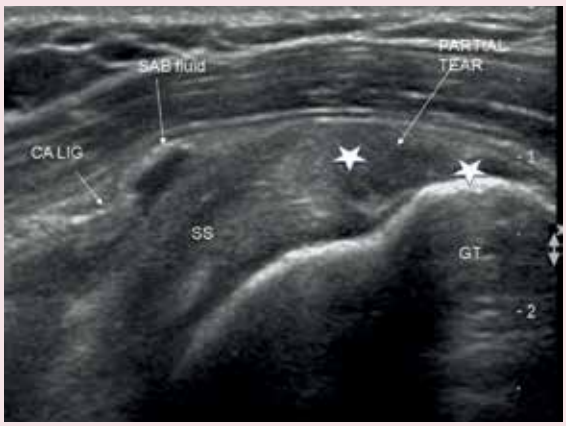


Figure 29. Fluid distending the bursa and bulging above the coracoacromial ligament as Michael starts to abduct. Note thickened, mildly hypoechoic SS tendon and the partial tear.

Figure 30. A thick but well-defined bursa in an asymptomatic man. This should not be mistaken for impingement.

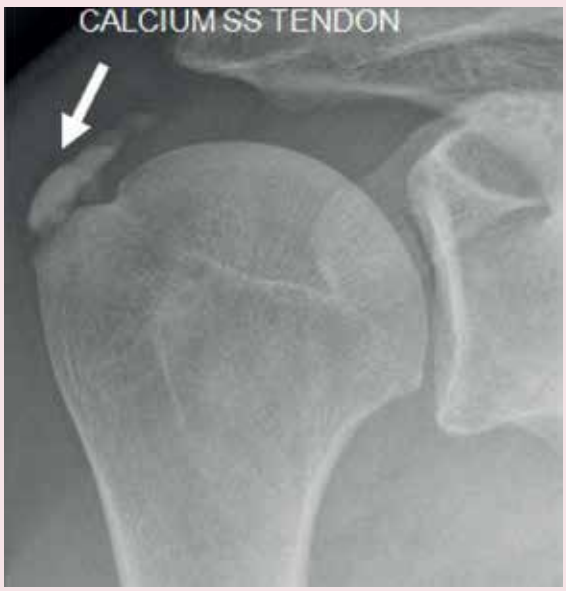


Figure 31. Calcium in the SS tendon.

ANSWER 6

The only serious but rare side effect of a cortisone injection is infection (usually staphylococcus). Other side effects reported by patients are a red skin flush usually around the head and neck, which is sometimes associated with feeling a bit under the weather with flu-like symptoms. Patients may get a flare of pain in the injected area for 1–3 days after the anaesthetic has worn off.[70] If Michael is diabetic he is likely to see a rise in his blood glucose levels for about a week, so he may need to check his blood sugars more frequently and adjust his medication.

ANSWER 7

In this scenario, some of the calcium from the tendon has extruded into the bursa setting up a severe bursitis, which is an extremely painful condition (see *Figures 31*, *32* and *33*). It is usually a self-limiting condition within a few weeks, but most patients will elect to have ultrasound guided aspiration of the calcium (see *Figure 34*) followed by steroid injection, which is an extremely effective method of pain control, usually working within days.[71] This technique is also effective for pain control of calcific tendonitis and will reduce the bulk of the tendon, making it easier for the tendon to fit through the coracoacromial arch on abduction.

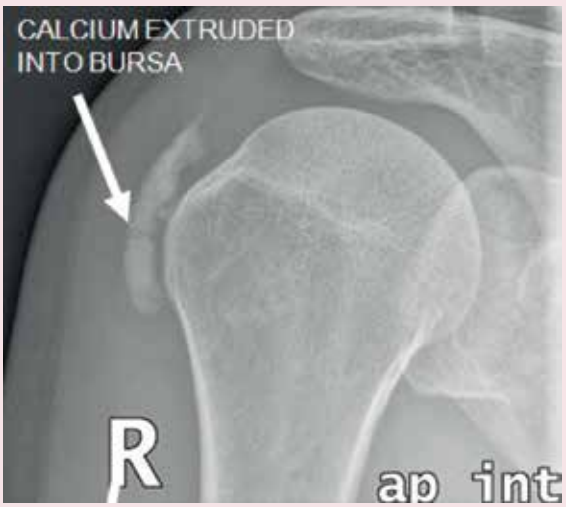


Figure 32. The calcium has extruded into the subacromial bursa, resulting in a painful bursitis.

Figure 33. Ultrasound image showing the extruded calcium in the bursa.

Figure 34. Calcium that has been aspirated from the bursa.

1. National Breast Cancer Centre. Clinical practice guidelines for the management and support of younger women with breast cancer. 2004 Camperdown: National Breast Cancer Centre. Available from http://nbcc.org.au
2. National Radiological Protection Board. X-rays: how safe are they? Chilton: National Radiological Protection Board, 2001. Available at www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1259151968131 [accessed 22 April 2013].
3. National Breast Cancer Centre. Breast imaging: a guide for practice. Camperdown, NSW: National Breast Cancer Centre, 2002.
4. Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice, 8th edn. East Melbourne: Royal Australian College of General Practitioners, 2012, p.65–6.
5. Davidson KG, Dubinsky TJ. Ultrasonographic evaluation of the endometrium in postmenopausal vaginal bleeding. Radiol Clin North Am 2003;41(4):769–80.
6. Lidor A, Ismajovich B, Confino E, David, MP. Histopathological findings in 226 women with post-menopausal uterine bleeding. Acta Obstet Gynecol Scand 1986;65(1):41–3.
7. Sahdev A. Imaging the endometrium in postmenopausal bleeding. BMJ 2007;334(7594):635–6.
8. Soliman PT, Oh JC, Schmeler KM, Sun CC, Slomowitz, BM, Gershenson, DM, et al. Risk factors for young premenopausal women with endometrial cancer. Obstet Gynecol 2005;105(3):575–80.
9. Nayfield SG, Karp JE, Ford LG, Dorr FA, Kramar BS. Potential role of tamoxifen in prevention of breast cancer. J Natl Cancer Inst 1991;83(20):1450–9.
10. Cancer Council Australia. Clinical practice guidelines for the treatment and management of endometrial cancer. Available at www.canceraustralia.gov.au/sites/default/files/publications/ncgc-vaginal-bleeding-flowcharts-march-20111_504af02038614.pdf
11. Gerber B, Krause A, Muller H, Reimer T, Kulz T, Makovitsky A, et al. Effects of adjuvant tamoxifen on the endometrium in postmenopausal women with breast cancer: a prospective long-term study using transvaginal ultrasound. J Clin Oncol 2000;18(20):3464–70.
12. Erdem M, Bilgin U, Bozkurt N, Erdem Al. Comparison of transvaginal ultrasonography and saline infusion sonohysterography in evaluating the endometrial cavity in pre- and postmenopausal women with abnormal uterine bleeding. Menopause 2007;14(5):846–52.
13. Bradley LD, Falcone T, Magen AB. Radiographic imaging techniques for the diagnosis of abnormal uterine bleeding. Obstet Gynecol Clin North Am 2000;27(2):245–76.
14. Nalaboff KM, Pellerito JS, Ben-Levi E. Imaging the endometrium: disease and normal variants. Radiographics 2001;21(6):1409–24.
15. Hardesty LA, Sumkin JH, Hakim C, Johns C, Nath M The ability of helical CT to preoperatively stage endometrial carcinoma. Am J Roentgenol 2001;176(3):603–6.
16. Manfredi R, Mirk P, Maresca G, et al. Local-regional staging of endometrial carcinoma: role of MR imaging in surgical planning. Radiology 2004;231(2):372–8.
17. Fisher B, Costantino JP, Wickerham DL, et al. Tamoxifen for prevention of breast cancer: report of the National Surgical Adjuvant Breast and Bowel Project P-1 Study. J Natl Cancer Inst 1998;90(18):1371–88.
18. Bergman L, Beelen ML, Gallee MP, Hollema H, Benraadt J, van Leeuwen FE. Risk and prognosis of endometrial cancer after tamoxifen for breast cancer. Lancet 2000;356(9233):881–7.
19. Kedar RP, Bourne TH, Powles TJ, et al. Effects of tamoxifen on uterus and ovaries of postmenopausal women in a randomised breast cancer prevention trial. Lancet 1994;343(8909):1318–21.
20. Neff MJ. Practice guidelines. Evidence-based guidelines for neuroimaging in patients with nonacute headache. Am Fam Physician 2005;71(6):1219–22.
21. Government of Western Australia Department of Health. A clinical decision support tool and educational resource for diagnostic imaging. Available at www.imagingpathways.health.wa.gov.au/includes/index.html [accessed 22 April 2013].
22. Acheson J. Idiopathic intracranial hypertension and visual function. Br Med Bull 2006;79–80(1):233–44.
23. Saposnik G, Barinagarrementeria F, Brown RD, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011;42(4):1158–92.
24. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. Lancet Neurol 2007;6(2):162–70.
25. Hasbun R, Abrahams J, Jekel J, Quagliarello VJ. Computed tomography of the head before lumbar puncture in adults with suspected meningitis. N Engl J Med 2001;345(24):1727–33.
26. The Royal Children's Hospital. Lumbar puncture guidelines. Available at www.rch.org.au/clinicalguide/guideline_index/lumbar_puncture_guideline [accessed 22 April 2013].
27. Sanchez R, Strouse PJ. The knee: MR imaging of uniquely pediatric disorders. Magn Reson Imaging Clin N Am 2009;17(3):521–37.
28. Stevens MA, El-Khoury GY, Kathol MH, Brandser EA, Chow S. Imaging features of avulsion injuries. Radiographics 1999;19(3):655–72.
29. Harcke HT, Mandell GA, Maxfield BA. Trauma to the growing skeleton. In: Kuhn JP, Slovis TL, Haller JO (eds). Caffey's pediatric diagnostic imaging. 10th edn. Philadelphia: Mosby, 2003, 2269–303.
30. De Smet AA, Fisher DR, Graf BK, Lange RH. Osteochondritis dissecans of the knee: value of MR imaging in determining lesion stability and the presence of articular cartilage defects. Am J Roentgenol 1990;155(3):549–53.
31. Wall E, Von Stein D. Juvenile osteochondritis dissecans. Orthop Clin North Am 2003;34(3):341–53.
32. Polousky JD. Juvenile osteochondritis dissecans. Sports Med Arthrosc 2011;19(1):56–63.
33. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. Am J Sports Med 2006;34(7):1181–91.
34. Bohndorf K. Osteochondritis (osteochondrosis) dissecans: a review and new MRI classification. Eur Radiol 1998;8(1):103–12.
35. Kramer J, Stiglbauer R, Engel A, Prayer L, Imhof H. MR contrast arthrography (MRA) in osteochondrosis dissecans. J Comput Assist Tomogr 1992;16(2):254–60.
36. Herring JA. Disorders of the knee. In: Herring JA ed. Tachdjian's pediatric orthopedics. 3rd edn. Philadelphia: WB Saunders Co, 2001, 789–838.
37. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999;353(9162):1386–9.
38. Pengo V, Lensing AW, Prins MH, et al. Incidence of chronic thromboembolic pulmonary hypertension after pulmonary embolism. N Engl J Med 2004;350(22):2257–64.
39. Stein PD, Woodard PK, Weg JG, et al. Diagnostic pathways in acute pulmonary embolism: recommendations of the PIOPED II investigators. Radiology 2007;242(1):15–21.
40. Stein PD, Fowler SE, Goodman LR, et al. Multidetector computed tomography for acute pulmonary embolism. N Engl J Med 2006;354(22):2317–27.
41. Mills SR, Jackson DC, Older RA, Heaston DK, Moore AV. The incidence, etiologies, and avoidance of complications of pulmonary angiography in a large series. Radiology 1980;136(2):295–9.
42. Worsley DF, Alavi A, Aronchick JM, Chen JT, Greenspan RH, Ravin CE. Chest radiographic findings in patients with acute pulmonary embolism: observations from the PIOPED study. Radiology 1993;189(1):133–6.

43. Zierler BK. Ultrasonography and diagnosis of venous thromboembolism. Circulation 2004;109:9–14.
44. Forbes KP, Reid JH, Murchison JT. Do preliminary chest X-ray findings define the optimum role of pulmonary scintigraphy in suspected pulmonary embolism? Clin Radiol 2001;56(5):397–400.
45. Hurwitz LM, Yoshizumi T, Reiman RE, et al. Radiation dose to the fetus from body MDCT during early gestation. Am J Roentgenol 2006;186(3):871–6.
46. Cook JV, Kyriou J. Radiation from CT and perfusion scanning in pregnancy. BMJ 2005;331(7512):350.
47. Eichinger S. D-dimer testing in pregnancy. Pathophysiol Haemost Thromb 2003;33(5–6):327–9.
48. Francalanci I, Comeglio P, Alessandrello Liotta A, et al. D-dimer plasma levels during normal pregnancy measured by specific ELISA. Int J Clin Lab Res 1997;27(1):65–7.
49. Perrier A, Roy PM, Aujesky D, et al. Diagnosing pulmonary embolism in outpatients with clinical assessment, D-dimer measurement, venous ultrasound, and helical computed tomography: a multicenter management study. Am J Med 2004;116(5):291–9.
50. Turkstra F, Kuijer PM, van Beek EJ, Brandjes DP, ten Cate JW, Buller HR Diagnostic utility of ultrasonography of leg veins in patients suspected of having pulmonary embolism. Ann Intern Med 1997;126(10):775–81.
51. Kang MJ, Park CM, Lee CH, Goo JM, Lee HJ. Dual-energy CT: clinical applications in various pulmonary diseases. Radiographics 2010;30(3):685–98.
52. Tortolani PJ, Carbone JJ, Quartararo LG. Greater trochanteric pain syndrome in patients referred to orthopedic spine specialists. Spine J 2002:2(4):251–4.
53. Bird PA, Oakley SP, Schnier R, Kirkham BW. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. Arthritis Rheum 2001;44(9):2138–45.
54. Kingzett-Taylor A, Tirman PF, Feller J, et al. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. Am J Roentgenol 1999:173(4):1123–6.
55. Segal NA, Felson DT, Torner JC, et al. Greater trochanteric pain syndrome: epidemiology and associated factors. Arch Phys Med Rehabil 2007;88(8):988–92.
56. Lievense A, Bierma-Zeinstra S, Schouten B, Bohnen A, Verhaar J, Koes B. Prognosis of trochanteric pain in primary care. BRJ Gen Pract 2005;55(512):199–204.
57. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Joint Surg Br 1997;79(4):618–620.
58. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. Anesth Analg 2009;108(5):1662–70.
59. Tanaka H, Kido K, Wakisaka A, Mine T Kawai S. Trochanteric bursitis in rheumatoid arthritis. J Rheumatol 2002;29(6):1340–1.
60. Slawski DP, Howard RF. Surgical management of refractory trochanteric bursitis. Am J Sports Med 1997;25(1):86–9.
61. Clancy WG. Runners' injuries. Part two. Evaluation and treatment of specific injuries. Am J Sports Med 1980;8(4):287–9.
62. McMahon SE, Smith TO, Hing CB. A systematic review of imaging modalities in the diagnosis of greater trochanteric pain syndrome. Musculoskeletal Care 2012;10(4):232–9.
63. Shbeeb MI, O'Duffy JD, Michet CJ Jr, O'Fallon WM, Matteson ELI. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol 1996;23(12):2104–6.
64. Hegedus EJ, Goode A, Campbell S, et al. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med 2008;42(2):80–92.
65. Neer CS 2nd. Impingement lesions. Clin Orthop Relat Res 1983;173: 70–7.
66. Read JW, Perko M. Ultrasound diagnosis of subacromial impingement for lesions of the rotator cuff. Australasian J Ultrasound Med 2010;13(2): 11–15.
67. Farin PU, Jaroma H, Harju A, Soimakillio SI. Shoulder impingement syndrome: sonographic evaluation. Radiology 1990;176(3):845–9.
68. Bureau NJ, Beauchamp M, Cardinal E, et al. Dynamic sonography evaluation of shoulder impingement syndrome. Am J Roentgenol 2006;187(1):216–20.
69. Read JW, Perko M. Shoulder ultrasound: diagnostic accuracy for impingement syndrome, rotator cuff tear, and biceps tendon pathology. J Shoulder Elbow Surg 1998;7(3):264–71.
70. Brinks A, Koes BW, Volkers AC, Verhaar JA, Bierma-Zeinstra SM. Adverse effects of extra-articular corticosteroid injections: a systematic review. BMC Musculoskelet Disord 2010;11:206.
71. Farin PU, Rasanen H, Jaroma H, Harju A. Rotator cuff calcifications: treatment with ultrasound-guided percutaneous needle aspiration and lavage. Skeletal Radiol 1996;25(6):551–4.

RESOURCES FOR DOCTORS

- The National Breast Cancer Centre's publication *Breast imaging: a guide for practice* is available for free download from Cancer Australia's website, http://canceraustralia.gov.au/publications-resources/cancer-australia-publications/breast-imaging-guide-practice [accessed 22 April 2013].
- The UK National Radiological Protection Board, with the College of Radiographers, the Royal College of Radiologists and the Royal College of General Practitioners, has produced a helpful, plain-language pamphlet called 'X-rays: how safe are they?' It is available for free download at www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1259151968131 [accessed 22 April 2013].
- Debra Ikeda's *Breast imaging: the requisites* provides a wealth of knowledge about diagnostic imaging of the breast.
- The American College of Radiology has developed ACR Appropriateness Criteria®, which can be found at www.acr.org/Quality-Safety/Appropriateness-Criteria [accessed 22 April 2013]. These criteria help referring physicians to make the most appropriate imaging decision for their patients.
- The Scottish Intercollegiate Guidelines Network guideline for investigation of post-menopausal bleeding can be found at www.sign.ac.uk/guidelines/fulltext/61/index.html [accessed 22 April 2013].
- Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice, 8th edn. East Melbourne: Royal Australian College of General Practitioners, 2012.
- National Breast Cancer Centre. Clinical practice guidelines for the management and support of younger women with breast cancer. 2004 Camperdown: National Breast Cancer Centre. Available from http://nbcc.org.au
- Cancer Council Australia. Clinical practice guidelines for the treatment and management of endometrial cancer. Available at www.canceraustralia.gov.au/sites/default/files/publications/ncgc-vaginal-bleeding-flowcharts-march-20111_504af02038614.pdf
- The Royal Australian and New Zealand College of Radiologists has developed an excellent website for consumers and referring doctors about a range of imaging procedures. It is available at www.insideradiology.com.au [accessed 22 April 2013].

RESOURCES FOR PATIENTS

- The UK National Radiological Protection Board, with the College of Radiographers, the Royal College of Radiologists and the Royal College of General Practitioners, has produced a helpful, plain-language pamphlet called 'X-rays: how safe are they?' It is available for free download at www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1259151968131 [accessed 22 April 2013].
- The Department of Health and Ageing has developed a helpful website (www.cancerscreening.gov.au) that provides information about a range of cancer screening programs.
- Cancer Council Australia (www.cancer.org.au) provides information about different types of cancer, including breast cancer.
- The Royal Australian and New Zealand College of Radiologists has developed an excellent website for consumers and referring doctors about a range of imaging procedures. It is available at www.insideradiology.com.au [accessed 22 April 2013].
- An information sheet for patients about cortisone injections is available at www.vhmi.com.au/upload/Cortisone%20Injections1.pdf [accessed 22 April 2013].

Imaging

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and
- log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au.

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.
FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

QUESTION 1

Adam is a 19-year-old university student who presents with persistent knee pain. He jogs and cycles and plays competitive basketball two nights per week. He has noticed that his pain is worse after exercise. Imaging reveals that he has a left osteochondral defect in the medial femoral condyle.

When is treatment such as arthroscopic drilling or chondrocyte reimplantation recommended?

A. If pain persists after 6 weeks of non-weight-bearing
B. If the lesion is unstable
C. If the lesion is stable
D. If he has persistent pain for more than 3 months
E. If he has irregularity of the medial condyle on X-ray.

QUESTION 2

Which of the following is true of tamoxifen?

A. Tamoxifen can act as both an oestrogen agonist and an oestrogen antagonist.
B. Tamoxifen increases the risk of cervical cancer.
C. Tamoxifen is used for the treatment of receptor-negative breast cancer.
D. Tamoxifen is usually prescribed to be taken for 4 years following a diagnosis of breast cancer.
E. Tamoxifen decreases the risk of endometrial hyperplasia.

QUESTION 3

Dot, aged 65 years, is brought to your clinic by her daughter June. Dot has had intermittent vaginal bleeding for 2 months. The bleeding is not heavy, but has occurred on most days. Dot is married to Bill; June is their only child. You order a transvaginal ultrasound, which shows a smooth endometrium of approximately 3 mm. Which of the following is the most likely diagnosis?

A. Endometrial atrophy
B. Benign endometrial hyperplasia
C. Endometrial carcinoma
D. Endometrial polyp
E. Uterine fibroid.

QUESTION 4

Mariko presents to your clinic with a severe headache of 3 hours duration. The headache started suddenly and is severe and unremitting. It is generalised. Mariko also complains of photophobia. On examination, she has a temperature of 37.5°C and some slight neck stiffness. You make a provisional diagnosis of meningitis. What imaging would you have performed immediately?

A. Skull X-ray
B. CT scan
C. MRI
D. MRV
E. None of the above.

QUESTION 5

Faraza, aged 30 years, comes to see you because she has felt a lump in her right breast. On examination you can feel a swelling in the UOQ of the breast. What is the most appropriate initial investigation?

A. Mammogram
B. Ultrasound
C. Mammogram and ultrasound
D. Referral to BreastScreen
E. Biopsy.

QUESTION 6

Janet, aged 53 years, presents with a 2-month history of pain in her right hip. She walks and swims regularly and has recently started attending a gym. She noticed the pain in her right hip shortly after starting a new exercise regimen that includes lunges and squats. These exercises seem to aggravate the pain. She has tenderness to palpation over the right greater trochanter and right buttock. What is the most likely cause of her pain?

A. OA of the hip
B. Gluteus medius tear
C. Torn acetabular labrum
D. Polymyalgia rheumatica
E. OA of the spine.

QUESTION 7

Alison, aged 37 years, presents with shortness of breath and some chest discomfort. She recently returned from a trip to Sweden where she was on holiday with friends. She is afebrile and her pulse rate is 100 beats per minute. Her chest is clear and she has no calf tenderness. You suspect she could have a PE. Along with the Wells Pulmonary Embolism Score, which test can be used to determine the most appropriate imaging pathway in Alison's case?

A. D-dimer
B. ECG
C. FBE
D. Coagulation profile
E. Spirometry.

QUESTION 8

Alison (see *Question 7*) is admitted to hospital with suspected PE. While she is having blood tests and an ECG, she tells staff that her period is late and there is a possibility she could be pregnant. What is a useful first test to perform if Alison is pregnant?

A. Pulmonary angiography
B. Venous Doppler ultrasound
C. MRI
D. CTPA
E. V/Q scan.

QUESTION 9

Kyle, aged 14 years, presents to your surgery with a 6-week history of pain in his left knee. He is a keen athlete and plays sport on most days. He is otherwise well. On examination he is afebrile. He has a full range of movement in his knee but has mild tenderness on palpation of his left tibial tuberosity. What is the most likely diagnosis?

A. Viral synovitis
B. Patellofemoral instability
C. Osteochondritis dissecans
D. Osgood–Schlatter disease
E. Left meniscal tear.

QUESTION 10

Walid, aged 57 years, presents with pain in his right shoulder. He has had the pain for 2 months. He is a keen swimmer, but the pain makes it difficult for him to swim more than 20 laps. On examination he has a painful arc and internal rotation on the right causes pain. Which imaging technique will give the most information to help you make a diagnosis of impingement syndrome?

A. X-ray
B. Ultrasound
C. Dynamic ultrasound
D. MRI
E. CT scan.

check

Independent learning program for GPs