

# check

Independent learning program for GPs



Unit 507 July 2014

## Renal problems

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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

## ABOUT THIS ACTIVITY

About 500,000 Australians present with kidney disease and urinary tract infections each year.<sup>1</sup> Kidney problems arise from acute illnesses (eg severe infections) or progressive damage due to conditions such as untreated diabetes or hypertension. Chronic kidney disease (CKD) is the progressive loss of renal function over time. Symptoms of worsening kidney function are non-specific and up to 90% of kidney function may be lost before symptoms appear.<sup>1</sup> CKD is highlighted as one of several chronic conditions that increase the burden of disease in Australia.<sup>2</sup> Early detection and management of CKD reduces deterioration of kidney function.<sup>3</sup> GPs have an important role in screening people at high risk of kidney disease.<sup>4</sup> This edition of *check* will consider renal scenarios of relevance to general practice in Australia.

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**LEARNING OUTCOMES**

At the end of this activity, participants will be able to:

- outline current management options for pregnant women with a urinary tract infection
- describe problems that may arise during end-stage renal failure in palliative care settings and how these can be managed
- outline appropriate screening options for people with suspected kidney disease
- describe the differential diagnosis of nephrotic syndrome, including possible secondary causes
- define resistant hypertension and outline its management.

**REFERENCES**

1. Kidney Health Australia: Fast facts on CKD in Australia. Available at [www.kidney.org.au/KidneyDisease/FastFactsonCKD/tabid/589/Default.aspx](http://www.kidney.org.au/KidneyDisease/FastFactsonCKD/tabid/589/Default.aspx) [Accessed 23 April 2014].
2. Royal Australasian College of General Practitioners. RACGP Curriculum for Australian General Practice. East Melbourne: RACGP; 2011. Available at [www.curriculum.racgp.org.au/](http://www.curriculum.racgp.org.au/) [Accessed 8 April 2014].
3. Stumpers S, Thomson N. Review of kidney disease among Indigenous people. Aust Indigenous Health Rev 2013. Available at [www.healthinfonet.ecu.edu.au/kidney\\_review](http://www.healthinfonet.ecu.edu.au/kidney_review) [Accessed 11 June 2014].
4. Royal Australasian College of General Practitioners. Guidelines for preventive activities in general practice 8th edition (the Red Book). East Melbourne: RACGP 2012. Available at [www.curriculum.racgp.org.au/](http://www.curriculum.racgp.org.au/) [Accessed 23 April 2014].

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

ACD	advance care directive	CVD	cardiovascular disease	MSU	midstream urine
ACEI	angiotensin converting enzyme inhibitor	DKD	diabetic kidney disease	NGSP	National Glycohemoglobin Standardization Program
ADL	activity of daily living	dsDNA	double-stranded DNA antibody	NSAID	non-steroidal anti-inflammatory drug
ADP	adenosine diphosphate	eGFR	estimated glomerular filtration rate	UACR	urinary albumin-to-creatinine ratio
AMI	acute myocardial infarct	ENA	extractable nuclear antigens	UEC	urea, electrolytes and creatinine
ANA	antinuclear antibodies	EPO	erythropoietin	UTI	urinary tract infection
ANCA	anti-neutrophil cytoplasmic antibodies	ESKD	end-stage kidney disease		
ARB	angiotensin receptor blocker	GORD	gastroesophageal reflux disease		
ATP	adenosine triphosphate	G6PD	glucose-6-phosphate dehydrogenase		
BMI	body mass index	IFCC	International Federation of Clinical Chemistry		
CCF	congestive cardiac failure	KHA-CARI	Kidney Health Australia-Caring for Australasians with Renal Insufficiency		
CKD	chronic kidney disease	KCAT	Kidney Check Australia Taskforce		
CPR	cardiopulmonary resuscitation	LDL	low density lipoprotein		
CrCl	creatinine clearance				
CTG	cardiotocography				

**CASE 1**

**JEFF IS WORRIED AS HIS MOTHER DIED ON DIALYSIS**

Jeff, a Caucasian man aged 65 years, presents to your practice after the death of his mother. His mother had end-stage kidney disease (ESKD) and recently died of a complication while on dialysis. Her chronic kidney disease (CKD) was a result of her diabetes and she had diabetic kidney disease (DKD). Jeff tells you that he has had type 2 diabetes for 11 years. He explains that he has been so busy caring for his mother and ageing father that he has not had time to care for himself. His last National Glycohemoglobin Standardization Program (NGSP) HbA1c was 10.3% (International Federation of Clinical Chemistry [IFCC] HbA1c is 89 mmol/mol). He has gained weight recently and has a body mass index (BMI) of 33 kg/m<sup>2</sup>. He is concerned that he now has DKD.

**QUESTION 1** 

What is concerning about Jeff's history? Do you think that his concerns about having DKD are appropriate?

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**QUESTION 2** 

What basic investigations (clinical and biochemical) would you consider for Jeff in order to best manage him at this stage?

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**FURTHER INFORMATION**

You begin to review Jeff's clinical course and past history and find that he had CKD stage 3a (eGFR 58 mL/min/1.73 m<sup>2</sup>) reported in his last evaluation, which was carried out by his previous GP 16 months ago. His type 2 diabetes is now insulin-requiring, he has been seeing a diabetic specialist and on evaluation today you find that he also has uncontrolled hypertension (blood pressure on repeated measurements is persistently >160/90 mmHg). In addition, he has ischaemic heart disease and had a coronary artery bypass graft 10 years ago. He also has dyslipidaemia, gout and asthma. A repeat eGFR is now 36 mL/min/1.73 m<sup>2</sup> and he has macroalbuminuria (UACR 70 mg/mmol). His NGSP HbA1c is now 7.2% (IFCC HbA1c 55 mmol/mol).

**QUESTION 3** 

What are your concerns following these findings? What would you do now?

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**QUESTION 4** 

Why does his diabetes seem to be better controlled? What are the risks with treatment now?

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**QUESTION 5** 

Should Jeff be referred to a nephrologist? If so, why?

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**FURTHER INFORMATION**

At Jeff's most recent review, his renal function has declined further and the eGFR is now 28 mL/min/1.73 m<sup>2</sup>. The continued decline in renal function is a major concern. There is some improvement in proteinuria but his renal function has continued to decline slightly. Blood pressure is also better at 149/58 mmHg, although his systolic blood pressure is not yet at target. Of note, his triglyceride and low density lipoprotein (LDL) levels are high despite the use of atorvastatin. His most recent relevant investigations include:

- albumin 35 g/L
- creatinine 172 μmol/L
- eGFR 35 mL/min/1.73 m<sup>2</sup> (previously 39, 43, 64 mL/min/1.73 m<sup>2</sup>)
- UACR 105 mg/mmol
- calcium 2.46 mmol/L
- phosphate 1.44 mmol/L
- HbA1c 7.36% (56)
- haemoglobin 109
- urate 0.42 mmol/L.

**QUESTION 6** 

Are there any remaining issues to consider?

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**CASE 1 ANSWERS**

**ANSWER 1**

In general, a doctor seeing a patient such as Jeff should be concerned about the likelihood of DKD, as there is a high association between diabetes and kidney disease. According to the Nefron Study,<sup>1</sup> every second patient with type 2 diabetes whom a doctor sees is likely to have CKD (47%). In the AusDiab study (2001)<sup>2</sup> and follow-on study (2005)<sup>3</sup> 16% of the 11,247 adults (>25 years) chosen by a census district Australian nation-representative sample had kidney damage, as evidenced by micro or macroalbuminuria (6.6%), an eGFR <60 mL/min/1.73 m<sup>2</sup> (5.8%) or haematuria (2.5%). Since 1998–2008, in most age groups, the rate of new patients with DKD per million people has been increasing. This is especially the case for people with Jeff's duration of diabetes (>10 years) and his age group (65–74 years).<sup>4</sup> His age group is most likely to be presenting with ESKD and requiring dialysis. Indeed, diabetes is the underlying cause of kidney failure that is largely driving up the increase in the number of dialysis patients in Australia.<sup>4</sup>

Apart from his diabetes, Jeff has a number of risks factors for CKD. He has a high likelihood of having CKD as he has a strong family history of CKD and, in particular, a first-degree relative, his mother, had CKD.<sup>5</sup> Among race and sex groups, 14.1% of Caucasian men, 14.6% of Caucasian women, 22.9% of African-American men and 23.9% of African-American women reported a first- or second-degree relative with ESKD (*P* = 0.001).<sup>5</sup> Another concern is his weight gain, which further increases his risk of CKD. Obesity (BMI ≥30 kg/m<sup>2</sup>) increases his relative risk to 1.78, compared with normal non-overweight (BMI <25 kg/m<sup>2</sup>) or overweight (BMI 25–29 kg/m<sup>2</sup>) individuals.<sup>6</sup> Those who are overweight or obese (BMI >30 kg/m<sup>2</sup>) are 40% and 80%, respectively, more likely to develop CKD, compared with those whose weight is in the normal range. Although not as powerful as diabetes or hypertension as a risk factor, obesity increases the risk of albuminuria and proteinuria. Obesity results in greater difficulty in achieving glycaemic and blood pressure control. Lastly, two-thirds of individuals in Jeff's age group are likely to have CKD. This means these individual have higher cardiovascular morbidity and mortality rate, compared with those without CKD, rather than a likelihood of requiring dialysis.<sup>7</sup>

**ANSWER 2**

As Jeff is at increased risk of DKD you need to investigate whether he has CKD and determine his stage and cardiovascular risk, as well as his likelihood of deterioration to ESKD or dialysis. He has at least five of eight established risk factors for CKD<sup>8</sup> (diabetes, hypertension, age >60 years, obesity and a family history of kidney failure). He does not smoke and is not an Aboriginal or Torres Strait Islander and you are not yet sure if he has established cardiovascular disease (CVD) or controlled hypertension. As per the Kidney Check Australia Taskforce (KCAT)

**Table 1. The new Australian CKD staging schema<sup>8</sup>**

GFR stage	GFR (mL/min/1.73 m <sup>2</sup> )	Albuminuria stage		
		Normal (urine ACR mg/mmol) Male: <2.5; Female: <3.5	Microalbuminuria (UACR mg/mmol) Male: 2.5–25; Female: 3.5–35	Macroalbuminuria (UACR mg/mmol) Male: >25; Female: >35
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present		
2	60–89			
3a	45–59			
3b	30–44			
4	15–29			
5	<15 or on dialysis			

Green = low; yellow = moderate; orange = high; red = very high

GP guidelines,<sup>8</sup> you would carry out a kidney health check on Jeff. This involves a blood test to measure his creatinine and therefore calculate his estimated glomerular filtration rate (eGFR), a urine test to assess urinary albumin-to-creatinine ratio (UACR) and so check for albuminuria, and finally a blood pressure check as this should be consistently below 130/80 mmHg for people with diabetes or albuminuria. Of those with type 2 diabetes, 20–40% develop nephropathy, which classically occurs in two stages: early nephropathy, which presents as microalbuminuria and normal–high eGFR, or overt nephropathy and macroalbuminuria with a progressive decline in eGFR.<sup>9,10</sup>

**ANSWER 3**

According to the KCAT guidelines Jeff has stage 3b CKD (eGFR 36 mL/min/1.73 m<sup>2</sup>) and macroalbuminuria, which is likely to be due to DKD (Table 1). The preferred measure of albuminuria should be done using UACR. Table 1 shows that he has significant proteinuria or macroalbuminuria. The approximate equivalents between UACR and other measures of albuminuria and proteinuria are shown in Table 2.<sup>11</sup>

**Table 2. Approximate equivalents between UACR and other measure of albuminuria and proteinuria<sup>11</sup>**

	UACR (mg/mmol)	24-hour urine albumin (mg/day)	Urine PCR (mg/mmol)	24-hour urine protein (mg/day)
Microalbuminuria	Male: 2.5–25	30–300	Male: 4–40	50–500
	Female: 3.5–35		Female: 6–60	
Macroalbuminuria	Male: >25	>300	Male: >40	>500
	Female: >35		Female: >60	

From the new Australian CKD staging schema (Table 1) he is at the highest risk for developing cardiovascular complications and

progression of his CKD. According to the KCAT guidelines<sup>8</sup> he fits into the 'Red Clinical Action Plan'.

What is further concerning is that he has uncontrolled hypertension, which is extremely common among those with type 2 diabetes and particularly in people with DKD. However, achieving blood pressure control is one of the most effective ways to delay the progression of kidney disease. In Jeff's case, therefore, particular effort should be made to control his blood pressure to a target consistently less than 130/85 mmHg. If Jeff's blood pressure is consistently below this target, the eGFR loss per year could be reduced by 80%<sup>12</sup> (Figure 1). Antihypertensive agents preferred for use by patients with diabetes are angiotensin converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARBs).<sup>13</sup> These agents may also slow the progression of CKD.<sup>14,15</sup> Other antihypertensive agents that lower blood pressure to target will also improve the patients' future CKD and CVD outlook.

Unfortunately, Jeff's absolute CVD risk is at the highest level. Absolute CVD risk assessment is not required for adults with any of the following conditions because they are already known to be at high risk of CVD:<sup>16</sup>

- diabetes and age >60 years
- diabetes with microalbuminuria
- moderate or severe CKD (eGFR <45 mL/min/1.73 m<sup>2</sup>)
- previous diagnosis of familial hypercholesterolaemia
- systolic BP ≥180 mmHg or diastolic BP ≥110 mmHg
- serum total cholesterol >7.5 mmol/L
- Aboriginal and Torres Strait Islander adults >74 years.<sup>16</sup>

In light of these findings, Jeff's risk factors should be aggressively controlled. Treating Jeff's diabetes aggressively will also slow progression of renal failure.<sup>17</sup> The most common recommended target in ordinary adult patients with diabetes is an HbA1c <6.5–7% (48–53 mmol/mol). For some patients, tighter control is the goal and is possible. One needs, therefore, to consider lifestyle modification, pharmacotherapy to treat his hypertension and monitoring every 6–12 weeks until sufficient improvements are achieved.<sup>18</sup>

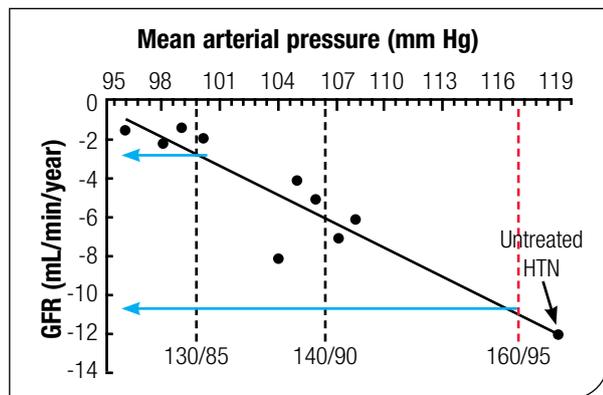


Figure 1. Adequate blood pressure management delays the progression of CKD<sup>12</sup>

#### ANSWER 4

His diabetes now seems better controlled as he has established renal failure. One has to now be careful not to cause hypoglycaemia with treatment. He may not require intensive control, especially if there is declining renal function. CKD increases the risk of hypoglycaemia for the following reasons:

- decreased renal glucose production
- reduced gluconeogenesis from alanine, pyruvate, glycerol, and decreased mitochondrial adenosine triphosphate/diphosphate (ATP/ADP) ratio in the liver
- decreased glycogen reserve
- reduced systemic response to adrenaline and glucagon
- decreased insulin degradation (normal kidney extracts 40% insulin), which intensifies and prolongs insulin actions
- reduced renal clearance of oral hypoglycaemic agents and their active metabolites.<sup>19</sup>

#### ANSWER 5

Referral to a nephrologist is recommended if any of the following are present:

- eGFR <30 mL/min/1.73 m<sup>2</sup>
- persistent significant albuminuria (UACR ≥30 mg/mmol)
- rapidly declining eGFR from a baseline of <60 mL/min/1.73 m<sup>2</sup> (a decline of >5mL/min/1.73 m<sup>2</sup> over a 6-month period, which is confirmed on at least three separate readings)
- CKD and hypertension that is difficult to get to target despite at least three anti-hypertensive agents
- haematuria with macroalbuminuria.<sup>8</sup>

Jeff's UACR result was 70 mg/mmol and if this persists on at least two repeat urine specimens, a referral is recommended.

Appropriate referral is associated with reduced rates of progression to ESKD, decreased rates of morbidity and mortality, decreased need for and duration of hospitalisation, increased likelihood of

permanent dialysis access created prior to dialysis onset, increased likelihood of kidney transplantation and reduced initial costs of care following the commencement of dialysis.<sup>20</sup>

#### ANSWER 6

Jeff's ongoing decline is probably related to the inability to control some of his risk factors. However, it may also be related to his still significant proteinuria and probable diabetic nephropathy. It is always important to consider an ultrasound to exclude obstruction as a cause for decline in renal function and to assess kidney morphology and size. Additional control is required to improve Jeff's blood pressure, which is still high. The use of a calcium channel blocker or low-dose thiazide diuretic would be appropriate for his now isolated systolic hypertension.<sup>21</sup>

For Jeff, the risk of death due to CVD is 20 times greater than the likelihood of surviving to the stage of needing dialysis or renal transplant. CKD is one of the most potent known independent risk factors for cardiovascular disease. Individuals with CKD have a 2–3-fold greater risk of cardiac death than individuals without CKD.<sup>22</sup> Therefore, ongoing aggressive measures to reduce morbidity and mortality due to cardiovascular disease are important. For this reason, Jeff's dyslipidaemia needs to be managed. There is some evidence that a combination of anti-lipid medication, such as simvastatin plus ezetimibe, has an advantage, especially for the reduction in cardiovascular risk. The results of the Study of Heart And Renal Protection (SHARP) showed a 17% reduction in major atherosclerotic events when comparing a placebo versus combination anti-lipid medication. Major atherosclerotic events included coronary death, myocardial infarction, non-haemorrhagic stroke or any revascularisation.<sup>23</sup> However, it is important to be aware that statins decrease mortality and cardiovascular events in people with early-stage CKD, have little or no effect in people receiving dialysis and have uncertain effects in kidney transplant recipients.<sup>24</sup>

#### REFERENCES

1. Thomas MC, Weekes AJ, Broadley O J, Cooper ME, Mathew TH. The burden of chronic kidney disease in Australian patients with type 2 diabetes (the NEFRON study). *Med J Aust* 2006;185:140–44.
2. Chadban SJ, Briganti EM, et al. Prevalence of kidney damage in Australian adults: the AusDiab kidney study. *J Am Soc Nephrol* 2003;14(7 Suppl 2):S131–38.
3. Barr EL, Wong TY, Tapp RJ, et al. Is peripheral neuropathy associated with retinopathy and albuminuria in individuals with impaired glucose metabolism? The 1999–2000 AusDiab. *Diabetes Care* 2006;29:1114–16.
4. Grace B, Hurst K, McDonald S. Australia and New Zealand Dialysis and Transplant Registry. New Patients. Adelaide: South Australia; 2012.
5. Freedman BI, Soucie JM, McClellan WM. Family history of end-stage renal disease among incident dialysis patients. *J Am Soc Nephrol* 1997;8:1942–45.
6. Hallan S I, Coresh J, Astor BC A, et al. International comparison of the relationship of chronic kidney disease prevalence and ESRD risk. *J Am Soc Nephrol* 2006;17:2275–84.

7. Mathew TH, Johnson DW, Jones GR. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: revised recommendations. *Med J Aust* 2007;187: 459–63.
8. Kidney Health Australia. Chronic Kidney Disease (CKD) Management in General Practice. Melbourne: Kidney Health Australia; 2012.
9. Tapp RJ, Shaw JE, Zimmet PZ, et al. Albuminuria is evident in the early stages of diabetes onset: results from the Australian Diabetes, Obesity, and Lifestyle Study (AusDiab). *Am J Kidney Dis* 2004;44:792–98.
10. Agarwal SK. Renal damage should be unquestionable when defining early chronic kidney disease. *Nephrol Dial Transplant* 2011;26:1110–11.
11. Johnson DW, Jones GR, Mathew TH, et al. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: new developments and revised recommendations. *Med J Aust* 2012;197:224–25.
12. Bakris GL, Williams M, Dworkin L, et al. Preserving renal function in adults with hypertension and diabetes: a consensus approach. National Kidney Foundation Hypertension and Diabetes Executive Committees Working Group. *Am J Kidney Dis* 2000;36: 646–61.
13. Harris P, Mann L, Phillips P, Bolger-Harris H, Webster C. Factors Complicating Management. In: *Diabetes Management in General Practice*. 17 ed. Canberra: Diabetes Australia; 2012. Available at [www.racgp.org.au/download/documents/Guidelines/Diabetes/201107diabetesmanagementgeneralpractice.pdf](http://www.racgp.org.au/download/documents/Guidelines/Diabetes/201107diabetesmanagementgeneralpractice.pdf) [Accessed 3 June 2014].
14. Barnett AH, Bain SC, Bouter P, et al. Angiotensin-receptor blockade versus converting-enzyme inhibition in type 2 diabetes and nephropathy. *N Engl J Med* 2004;351:1952–61.
15. Strippoli GF, Craig M, Deeks JJ, Schena FP, Craig JC. Effects of angiotensin converting enzyme inhibitors and angiotensin II receptor antagonists on mortality and renal outcomes in diabetic nephropathy: systematic review. *BMJ* 2004;329:828.
16. Ludlow M, Mathew T. Changes in the detection and management of kidney disease. *Diabetes Management* 2012;39:6–17,20–21
17. UK Prospective Diabetes Study Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 1998;352:837–53.
18. National Blood Pressure and Vascular Disease Advisory Committee. Guide to management of hypertension. Canberra: National Heart Foundation of Australia; 2008 (updated 2010). Available at [www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf) [Accessed 3 June 2014].
19. Moen MF, Zhan M, Hsu VD, et al. Frequency of hypoglycaemia and its significance in chronic kidney disease. *Clin Am Soc Nephrol* 2009;4:1121–27.
20. Huisman RM. The deadly risk of late referral. *Nephrol Dial Transplant* 2004;19:2175–80.
21. van Zwieten PA. Drug treatment of isolated systolic hypertension. *Nephrol Dial Transplant* 2001;16:1095–97.
22. Go A, Chertow G, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events and hospitalization. *N Engl J Med* 2004;351:1296–1305.
23. Baigent C, Landray MJ, Reith C, et al. The effects of lowering LDL cholesterol with simvastatin plus ezetimibe in patients with chronic kidney disease (Study of Heart and Renal Protection): a randomised placebo-controlled trial. *Lancet* 2011;377:2181–92.
24. Palmer SC, Craig JC, Navaneethan SD, Tonelli M, Pellegrini F, Strippoli GF. Benefits and harms of statin therapy for persons with chronic kidney disease: a systematic review and meta-analysis. *Ann Intern Med* 2012;157:263–75.

**CASE 2**

**GEORGE HAS PAINFUL TOES**

George, a retired mechanic aged 78 years, has come to see you about his painful toes. His past history includes hypertension for over 30 years, dyslipidaemia and coronary artery disease. He is an ex-smoker with a smoking history of 60 packs per year. He quit smoking at the age of 52 years when he was referred for coronary artery bypass grafts.

He was in hospital recently for management of heart failure and tells you he was discharged last week on 'fluid tablets' and that his kidneys are 'bad'.

You diagnose an acute attack of gout involving George's first metatarsal-phalangeal joints on both feet and his left ankle. His recent hospital discharge summary is shown in *Table 1*.

Parameter	George	Reference
Sodium	132 mmol/L	135–145 mmol/L
Potassium	4.4 mmol/L	3.5–5.0 mmol/L
Chloride	100 mmol/L	95–110 mmol/L
Bicarbonate	28 mmol/L	22–32 mmol/L
Urea	12.0 mmol/L	3–8 mmol/L
Creatinine	239 µmol/L	60–120 µmol/L
Urate	0.58 mmol/L	0.2–0.45 mmol/L
eGFR	26 mL/min/1.73 m <sup>2</sup>	>90 mL/min/1.73 m <sup>2</sup>

While in hospital, George was told that he cannot have analgesia because of his 'bad' kidneys. He says his pain is debilitating.

**QUESTION 1** 

What can be done for George's pain?

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**FURTHER INFORMATION**

George returns to see you 2 years later to discuss his insomnia. On further questioning, he tells you he is unable to lie still at night and spends most nights walking around his bedroom. He describes discomfort in his legs and an urge to move his legs, which is relieved by getting up and walking around; however, he finds it difficult to describe the exact nature of the discomfort.

**QUESTION 2** 

How should this problem be managed?

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**FURTHER INFORMATION**

Several months later, George presents for a routine check-up. He is now 81 years old. His cardiologist told him that his heart failure is worse and that he has concurrent chronic obstructive pulmonary disease from his previous cigarette exposure. He has been in hospital four times in the last 6 months, twice for heart failure, once for exacerbation of airways disease and once for a fall. His daily activities are severely limited by shortness of breath and he uses home oxygen at night. He is unable to care for himself and has moved in with his daughter and her family.

On his recent visit to his nephrologist, he was told that he has kidney failure but that he is not a suitable candidate for dialysis. He agreed that he does not want to be kept alive 'hooked up to a machine' but his family members tell him that he is 'giving up'.

**QUESTION 3**  

What would you say to George?

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**Table 2. Analgesic ladder and their use in CKD<sup>4,5</sup>**

WHO analgesic ladder	Drug/drug class	Suitable for patients with CKD	Comments
Step 1	Paracetamol	Yes	Analgesia of choice in CKD Metabolised in the liver; safe in CKD No need for dose adjustment
	NSAIDs	No	Reduction in GFR (may be irreversible) Sodium retention aggravates hypertension
Step 2	Codeine	No	Metabolised in the liver but active metabolites are renally excreted
	Oxycodone	Reduce dose	Metabolised in the liver but active metabolites are renally excreted
	Tramadol	Reduce dose	90% of drug and metabolites are renally excreted If GFR <30 ml/min or on dialysis, max dose <200 mg/day (eg 50 mg QID) If GFR <15ml/min, max dose <100mg/day (eg 50 mg BD)
Step 3	Morphine	No	Active metabolites accumulate in renal failure. Associated with significant toxicity in CKD
	Hydromorphone	Reduce dose	Metabolised in the liver and renally excreted Safe in CKD at renally adjusted doses Start with 0.5–1 mg q 6 h
	Methadone	Yes	Safe in renal failure Best prescribed by experienced prescriber
	Fentanyl	Yes	Metabolised in the liver; not renally excreted Safe in renal failure
	Buprenorphine	Yes	Metabolised in the liver; metabolites renally excreted Commence at a lower dose

dose can start from 100 mg nocte.<sup>5</sup> Symptoms and response to treatment should be reassessed within 1–2 weeks. Ropinirole can be commenced at 0.5 mg nocte and increased to 1 mg nocte.<sup>9</sup> Pramipexole is another dopamine agonist that can be used.<sup>10</sup>

### ANSWER 3

Patients and their carers should be making informed choices regarding ESKD management and this is best done in a multidisciplinary setting. The options of renal replacement therapy and conservative management should be discussed. Evidence from observational and retrospective studies suggest that dialysis extends life in elderly patients, but this survival advantage is negated by comorbidities, such as ischaemic heart disease, and the fact that most patients tend to lose their independence. Similarly, those with poorer functional status do not have any survival advantage with dialysis. Outcomes are poor in nursing home patients initiated on dialysis and one US study showed a mortality rate of 58% in the first year. In this study only 13% of patients maintained their pre-dialysis functional status.<sup>11</sup>

According to the Renal Physicians Association Clinical Practice Guidelines,<sup>12</sup> it is reasonable to consider forgoing dialysis for patients with a very poor prognosis or for whom dialysis cannot be provided safely. Such patients include:

- those with a medical condition where dialysis cannot be tolerated, such as those with dementia (unable to cooperate), or profound hypotension
- those with a terminal illness from a non-renal cause (such as metastatic cancer with poor prognosis and short life expectancy)
- those aged >75 years with stage 5 CKD who meet two or more of the following statistically significant very poor prognosis criteria:
  - clinicians' response of 'no, I would not be surprised' to the surprise question (ie 'would I be surprised if this patient died in the next year?')
  - high comorbidity score
  - significantly impaired functional status
  - severe malnutrition.

Good communication is essential and it is important to encourage the family and the patient to meet with the nephrologist, so that all concerns can be canvassed within a shared decision-making framework.

**ANSWER 4**

Several symptoms are prevalent in patients with ESKD. Some of these symptoms and their recommended treatments are discussed below:<sup>13–16</sup>

- Fatigue: the causes of fatigue in ESKD are multifactorial. The principal reversible issue is anaemia secondary to reduced erythropoietin (EPO) synthesised by the kidney. This can be treated with synthetic EPO-analogues.
- Pruritus: uraemic pruritus is common in ESKD, with or without dialysis. After excluding other causes of itch (such as allergies or skin infections), pharmacotherapy may include:
  - topical moisturisers: treat dry skin
  - gabapentin: needs to be used at a reduced dose in renal impairment. In patients with eGFR <15mL/min/1.73 m<sup>2</sup>, starting doses of 100 mg on alternate days (or after dialysis for patients on dialysis) would be appropriate. Titrate the dose up as needed
  - evening primrose oil: start at doses of 1 tablet twice daily and consider titrating up to 2 tablets twice daily, depending symptoms.

There is no evidence for the efficacy of antihistamines in uraemic pruritus.
- Nausea is common in ESKD and reduces quality of life and nutritional intake. Constipation should be excluded, especially if opioids are used. Pharmacotherapy includes:
  - metoclopramide: may be used in ESKD and has prokinetic effects (especially preferred in patients with early satiety and/or constipation)
  - haloperidol: may be used in ESKD, starting at 50% of normal dose
  - levomepromazine: may be used if symptoms persist despite above.

Note, metoclopramide and haloperidol have extrapyramidal side-effects.

Cyclizine may induce hypotension and tachyarrhythmias and is usually reserved for refractory cases, after discussion with a specialist palliative care team.

- Dyspnoea is often multifactorial in nature and be due to a combination of anaemia, pulmonary oedema and co-existing cardiac and pulmonary disease. General measures to alleviate symptoms include:
  - provision of education and support for the patient and family on how to cope and respond to breathlessness.
  - sitting upright and use of oxygen therapy if the patient is hypoxic. A fan could be used for cool air.
  - gentle physiotherapy, which may improve function.
  - as disease progresses and non-pharmacological measures

fail, low-dose opioids can be used to relieve breathlessness (see above, principles of use of opioids), combined with low-dose benzodiazepines (such as lorazepam 0.5–1 mg orally or sublingually), especially if there are co-existing symptoms of anxiety.

**ANSWER 5**

End-of-life care aims to provide the best possible quality of life for the patient and the family. The core elements for George and other patients dying from ESKD include:

- anticipated symptoms: these include lethargy, daytime drowsiness and shortness of breath. It is important to have meticulous communication with the family regarding these issues and to address them pre-emptively
- location of care: this will depend on individual circumstances and may be done in the home, a hospice or, in George's case, a high-level residential care facility, where he is expected to remain until death
- meticulous communication and support, including bereavement support, should be provided to the family
- management of terminal symptoms:<sup>14,15</sup>
  - pain: maintain adequate analgesia if the patient is in pain (refer to section on pain management above)
  - terminal agitation: midazolam, used subcutaneously, is recommended to relieve agitation in the dying phase. In CKD, dose reduction and increased dosing interval are recommended.
  - terminal secretions: hyoscine is not recommended as it crosses the blood-brain-barrier more avidly in the uraemic state and may cause heightened agitation. Agents such as glycopyrolate or atropine or will be more appropriate
  - fluid input control and judicious use of frusemide.

**REFERENCES**

1. Rossi S, editor. Gout. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
2. Rossi S, editor. NSAIDs. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
3. Rossi S, editor. Allopurinol. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
4. Davison SN, Ferro CJ. Management of pain in chronic kidney disease. *Prog Palliative Care* 2009;17:186–95.
5. Davison SN, Kincicki H, Brennan F. Pain in chronic kidney disease: a scoping review. *Semin Dial* 2014;27:188–204.
6. Trenkwalder C, Hening WA, Montagna P, et al. Treatment of restless legs syndrome: an evidence-based review and implications for clinical practice. *Mov Disord* 2008;23:2267–2302.
7. Murtagh FEM, Weisbord S. Symptoms in renal disease; their epidemiology, assessment, and management. In: Chambers EJ, Brown EA, Germain M eds. *Supportive care for the renal patient*. Second Edition. Oxford: Oxford University Press. 2010;pp 103–32.

8. Rossi S, editor. Gabapentin. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
9. Rossi S, editor. Ropinirole. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
10. Rossi S, editor. Pramipexole. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
11. Kurella Tamura M, Covinsky KE, Chertow GM, Yaffe K, Landefeld CS, McCulloch CE. Functional status of elderly adults before and after initiation of dialysis. *N Eng J Med* 2009;361:1539–47.
12. Renal Physicians Association of the USA. Clinical Practice Guideline on Shared Decision-Making in the Appropriate Initiation of and Withdrawal from Dialysis. Executive Summary of Guidelines. Rockville: RPA; 2010.
13. Murtagh FEM, Weisbord S. Symptoms in renal disease; their epidemiology, assessment, and management. In: Chambers EJ, Brown EA, Germain M (eds) Supportive care for the Renal patient. Second Edition. Oxford: Oxford University Press. 2010; pp 103–32.
14. Douglas C, Murtagh FEM, Chambers EJ, et al. Symptom management for the adult patient dying with advanced chronic kidney disease: A review of the literature and development of evidence-based guidelines by a United Kingdom Expert Consensus Group. *Palliative Med* 2009;23:103–10.
15. Palliative Care Expert Group. Renal failure in patients receiving palliative care. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 15 April 2014].
16. Brennan F, Siva B, Crail S. Appropriate assessment of symptom burden and provision of patient information. *Nephrology* 2013;13:418–21.

**CASE 3**

**GRAHAM HAS SWOLLEN ANKLES**

Graham, aged 54 years, is a financial advisor who is concerned by swelling of his ankles over the past 2 months. During this time, he has gained 5 kg and has been recently aware of having frothy urine on voiding. Three years ago, he was diagnosed with hypertension and his blood pressure has been well controlled on amlodipine 10 mg daily. Graham quit smoking 3 years ago and drinks a glass of red wine with dinner each night. On examination, Graham has bilateral pitting ankle oedema to the level of the knees. His clinic blood pressure is 155/95 mmHg, his weight is 95 kg (BMI 31.0 kg/m<sup>2</sup>) and his waist circumference is 102 cm. His physical examination is otherwise normal.

**QUESTION 1** 

What are the possible causes of Graham's ankle swelling?

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**QUESTION 2** 

How should Graham be screened for the presence of kidney disease?

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**QUESTION 3** 

What is the recommended test for initial evaluation of albuminuria/proteinuria?

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**FURTHER INFORMATION**

Graham's fasting serum biochemical testing reveals the following:

- urea 9.1 mmol/L (normal 3–8 mmol/L\*)
- creatinine 145 mmol/L (normal 60–120 mmol/L\*)
- eGFR 47 mL/min/1.73 m<sup>2</sup>, glucose 5.1 mmol/L (normal 3–5.4 mmol/L\*)
- albumin 28 g/L (normal 32–45 g/L; varies with age\*)
- total cholesterol 7.8 mmol/L (normal <5.5 mmol/L\*)
- UACR 247 mg/mmol.

(\*normal ranges for Queensland Health Pathology Services; ranges from other laboratories may vary slightly)

**QUESTION 4** 

What is your working diagnosis?

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**QUESTION 5** 

What complications might Graham experience from his kidney disease?

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**QUESTION 6** 

What additional investigations would you perform?

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**QUESTION 7** 

How would you treat Graham initially?

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**QUESTION 8** 

Should Graham be referred to a nephrologist?

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**CASE 3 ANSWERS**

**ANSWER 1**

Bilateral ankle swelling can be caused by a number of conditions, including congestive cardiac failure (CCF), chronic kidney disease (CKD), chronic liver disease, medications (particularly dihydropyridine calcium channel blockers such as amlodipine) and bilateral lower limb venous obstruction (eg deep venous thrombosis). Graham's leg swelling may be related to his amlodipine therapy,<sup>1</sup> although the presence of frothy urine raises the possibility of proteinuria (protein reduces the surface tension of urine). Graham has hypertension and obesity, which are two of the seven recognised risk factors for CKD (the other risk factors are diabetes mellitus, smoking, family history of CKD, established cardiovascular disease (CVD) and Aboriginal or Torres Strait Islander origin).<sup>2-4</sup> The presence of any CKD risk factor warrants screening for the presence of CKD.<sup>2,3</sup>

**ANSWER 2**

According to the Kidney Health Australia-Caring for Australasians with Renal Insufficiency (KHA-CARI) guidelines<sup>4</sup>, Graham should be screened for CKD by testing for albuminuria/proteinuria and requesting an estimated glomerular filtration rate (eGFR), which is automatically reported by all Australian pathology laboratories with every serum creatinine request in adults over the age of 18 years. Optimal detection and subsequent risk stratification of CKD requires simultaneous consideration of both eGFR and albuminuria/proteinuria.<sup>6</sup>

**ANSWER 3**

According to the Chronic Kidney Disease and Measurement of Albuminuria or Proteinuria Position Statement,<sup>6</sup> 'the preferred method for assessment of albuminuria in both diabetic and non-diabetic patients is urinary albumin-to-creatinine ratio (UACR) measurement in a first-void spot urine specimen. Where a first-void specimen is not possible or practical, a random spot urine specimen for UACR is acceptable.'

**ANSWER 4**

Graham has an abnormally low eGFR (<60 mL/min/1.73 m<sup>2</sup>) and very heavy albuminuria that is in the nephrotic range (approximately >220 mg/mmol).<sup>7</sup> Strictly speaking, a diagnosis of CKD requires the documented presence of a low eGFR and/or albuminuria (UACR >2.5 mg/mol in men or >3.5 mg/mol in women) on at least two occasions 3 months apart,<sup>2</sup> but Graham's clinical presentation and laboratory findings are likely to represent stage 3a CKD with macroalbuminuria (*Table 1*).

The constellation of ankle oedema, heavy proteinuria, hypoalbuminaemia and hypercholesterolaemia indicate that Graham has nephrotic syndrome. The most common causes of this presentation in the community are diabetes mellitus and chronic glomerulonephritis.<sup>8</sup> The absence of clinical or biochemical features

Kidney function stage	GFR (mL/min/1.73 m <sup>2</sup> )	Albuminuria stage		
		Normal (UACR mg/mmol) Male: <2.5; Female: <3.5	Microalbuminuria (UACR mg/mmol) Male: 2.5–25; Female: 3.5–35	Macroalbuminuria (UACR mg/mmol) Male: >25; Female: >35
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present		
2	60–89			
3a	45–59			
3b	30–44			
4	15–29			
5	<15 or on dialysis			

Green = low; yellow = moderate; orange = high; red = very high

of diabetes mellitus suggest that Graham most probably has a form of chronic glomerulonephritis. The likely diagnoses in descending order of probability would be membranous nephropathy, IgA nephropathy and focal segmental glomerulosclerosis. Occasionally, nephrotic syndrome can be caused by secondary glomerulonephritis rather than primary glomerulonephritis. Possible secondary causes of nephrotic syndrome are listed in *Table 2*.

Other diseases	Diabetes mellitus, systemic lupus erythematosus, amyloidosis
Cancers	Myeloma Lymphoma
Drugs	Non-steroidal anti-inflammatory drugs Captopril Tamoxifen Lithium
Infections	HIV Hepatitis B and C Mycoplasma Syphilis Malaria
Congenital causes	Alports syndrome Denys-Drash syndrome Congenital nephrotic syndrome of the Finnish type

**ANSWER 5**

Graham has a very high likelihood of having progressive kidney failure, given the combination of nephrotic-range proteinuria, low eGFR, male gender, hypertension and obesity. His nephrotic-range proteinuria, low eGFR and hypercholesterolaemia also place him at a significantly increased risk of atherosclerotic cardiovascular disease. Indeed, Graham is more than 20 times more likely to die from cardiovascular disease than survive to the point of needing kidney replacement therapy (dialysis or kidney transplantation).

Other recognised complications of nephrotic syndrome include

deep venous thrombosis, including renal vein thrombosis (due to intravascular volume depletion, compensatory increase in coagulation factor synthesis in response to hypoalbuminaemia and urinary loss of anticoagulants such as antithrombin III, protein C and protein S), infection (due to low serum IgG concentrations, decreased complement activity and depressed T cell function) and volume depletion (particularly in the context of diuretic therapy).<sup>8</sup>

**ANSWER 6**

The KHA-CARI Guidelines recommend that it is important to consider the underlying cause of Graham’s proteinuria and to pursue the diagnosis sufficiently to exclude treatable pathology, such as obstruction, vasculitis and rapidly progressive glomerulonephritis.<sup>3</sup> In the first instance, it is important to repeat the eGFR measurement to ensure that Graham is not experiencing an acute deterioration in kidney function. It would also be useful to confirm the nephrotic-range proteinuria by performing a timed urine collection to quantify 24-hour proteinuria. Other investigations that should be performed include a full blood count, urine microscopy and culture, renal ultrasound scan, urine and serum electrophoresis, serum free light chains, antinuclear antibodies (ANA), anti-double stranded DNA antibody (dsDNA), anti-neutrophil cytoplasmic antibodies (ANCA), extractable nuclear antigens (ENA), complement studies, hepatitis B and C serology, HIV serology and a serum parathyroid hormone level.<sup>3</sup>

**ANSWER 7**

Graham should receive advice about lifestyle modification, including a low salt (<100 mmol salt or 6 g salt or 2.3 g sodium per day), normal protein (0.75–1.0 g/kg/day) diet high in vegetables and fruit, and low in saturated fats.<sup>3</sup> A low-protein diet is not recommended as patients are at risk of malnutrition and muscle wasting. Graham should also aim for a healthier weight and exercise regularly. An angiotensin converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB) should be started, aiming for a blood pressure <130/80 mmHg. Combination therapy with ACEIs and ARBs is not advised due to the risk of hyperkalemia or acute kidney injury.<sup>10</sup> A repeat eGFR should be ordered within 1–4 weeks after commencing an ACEI or ARB to ensure

these agents do not cause acute kidney injury (serum creatinine rise >30% or eGFR fall >25%) or clinically important hyperkalaemia (serum potassium >6 mmol/L) necessitating a change of therapy.<sup>3</sup>

Controlling hypercholesterolemia through dietary modification and use of a statin has been shown to reduce the risk of CKD progression and cardiovascular disease.<sup>3</sup>

Loop diuretics should be used sparingly to control Graham's salt and water retention. In the opinion of the authors, a starting dose of 40–80 mg daily would be appropriate. In refractory cases, adding a thiazide diuretic or potassium-sparing diuretic can work synergistically to inhibit distal sodium absorption and promote further diuresis. This is best undertaken under the guidance of a nephrologist, as Graham should be monitored closely for clinical evidence of intravascular volume depletion (postural light-headedness and hypotension, low jugular venous pressure, deterioration of kidney function). Graham's amlodipine should be ceased in case this agent is aggravating his ankle oedema. Some clinicians recommend commencing aspirin to mitigate the risk of deep venous thrombosis, but this should be postponed if a kidney biopsy is imminent (as would apply in Graham's case).

### ANSWER 8

Graham's heavy proteinuria (UACR 330 mg/mmol) meets the Kidney Check Australia Taskforce's recommended indications for referral to a nephrologist (Table 3).

#### Table 3. Recommendations for referral to a specialist renal service or nephrologist<sup>2</sup>

Referral to a specialist renal service or nephrologist is recommended in the following situations:

- eGFR <30 mL/min/1.73 m<sup>2</sup>
- Persistent significant albuminuria (UACR ≥30 mg/mmol)
- A consistent decline in eGFR from a baseline of <60 mL/min/1.73 m<sup>2</sup> (a decline >5 mL/min/1.73 m<sup>2</sup> over a 6-month period, which is confirmed on at least three separate readings)
- Glomerular haematuria with macroalbuminuria
- CKD and hypertension that is hard to get to target despite at least three anti-hypertensive agents.

### CONCLUSION

A subsequent biopsy confirmed a diagnosis of membranous glomerulonephritis. Graham was commenced on immunosuppressive therapy with cyclosporine.

### REFERENCES

1. Webster J, Robb OJ, Jeffers TA, Scott AK, Petrie JC. Once-daily amlodipine in the treatment of mild to moderate hypertension. *J Cardiovasc Pharmacol* 1988;12(Suppl 7):S72–75.
2. Kidney Health Australia. Chronic Kidney Disease (CKD) Management in General Practice 2nd edition, Melbourne 2012. Available at [www.kidney.org.au/LinkClick.aspx?fileticket=vfDcA4sEUMs%3d&tabid=635&id=1584](http://www.kidney.org.au/LinkClick.aspx?fileticket=vfDcA4sEUMs%3d&tabid=635&id=1584) [Accessed 28 April 2014].
3. Johnson DW, Atai E, Chan M, et al. KHA-CARI guideline: early chronic kidney disease: detection, prevention and management. *Nephrology* 2013;18: 340–50.

4. Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice 8th edition. Melbourne: RACGP; 2012. Available at [www.racgp.org.au/download/Documents/Guidelines/Redbook8/redbook8.pdf](http://www.racgp.org.au/download/Documents/Guidelines/Redbook8/redbook8.pdf) [Accessed 28 April 2014].
5. Johnson DW, Jones GR, Mathew TH, et al. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: new developments and revised recommendations. *Med J Aust* 2012;197:222–23.
6. Johnson DW, Jones GR, Mathew TH, et al. Chronic kidney disease and measurement of albuminuria or proteinuria: a position statement. *Med J Aust* 2012;197:224–225.
7. Montero N, Soler MJ, Pascual MJ, et al. Correlation between the protein/creatinine ratio in spot urine and 24-hour urine protein. *Nefrologia: Publicacion Oficial de la Sociedad Espanola Nefrologia* 2012;32:494–501.
8. Hull R, Goldsmith D. Nephrotic syndrome in adults. *BMJ* 2008;336:1185–89.
9. Keith D, Nichols G, Gullion C, Brown J, Smith D. Longitudinal follow-up and outcomes among a population with chronic kidney disease in large managed care organization. *Arch Int Med* 2004;164:659–63.
10. Ontarget Investigators. Telmisartan, ramipril, or both in patients at high risk for vascular events. *N Engl J Med* 2008;358:1547–59.

**CASE 4**

**RUBY REFUSES DIALYSIS**

Ruby, aged 82 years, is a resident at a nursing home. She attends outpatient dialysis at the local hospital 3 times a week. She was admitted to her nursing home 6 months ago because of increasing frailty and mobility problems.

Her past history includes ischaemic heart disease and mild congestive cardiac failure (CCF) following an acute myocardial infarct (AMI) 3 years ago. She has chronic lower back pain with right-sided sciatica and mild gastroesophageal reflux disease (GORD).

She commenced renal dialysis 2 years ago for chronic renal failure, presumably related to atherosclerosis, and has required renal dialysis since that time.

Ruby's general health has declined since her admission to the nursing home. She is now virtually bed- and chair-bound and has developed a chronic ulcer over her sacrum. She experiences considerable pain with movement and requires ambulance transportation to attend dialysis.

This morning she claims that she has had enough and refuses to go to dialysis. Ruby is usually lucid and coherent despite her physical frailty; however, over the last few weeks she has become slightly confused and irrational. She is uncharacteristically resistant to nursing attention and assistance with activities of daily living (ADLs). She asks to speak to you as her regular GP.

**QUESTION 1** 

How would you respond to Ruby's request to withdraw from dialysis?

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**FURTHER INFORMATION**

When Ruby first entered the nursing home, she and her family were encouraged to consider treatment limitations. She has a written advance care directive (ACD) and nominated her older daughter, who is a registered nurse, as her enduring medical guardian.

Ruby has become slightly vague in recent weeks but is determined not to get into the ambulance today. Her ACD states that if her quality of life deteriorated such that she was either bed-bound, confused or both, she would wish to cease dialysis. Ruby's ACD states that in the event of a cardiorespiratory arrest, she is not to have cardiopulmonary resuscitation (CPR). Ruby had previously discussed this matter with her three children, who have also met with her renal physician and the social worker at the dialysis unit over the past few months.

The family feels comfortable supporting Ruby's decision to withdraw from dialysis. They have requested that Ruby remain at her nursing home rather than be transferred to a hospital or hospice for palliative care.

**QUESTION 2** 

What is Ruby's life expectancy without dialysis?

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**QUESTION 3**   

Is withdrawing from dialysis euthanasia?

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**QUESTION 4** 

What are the general considerations regarding palliative care?

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**FURTHER INFORMATION**

Ruby's current medications include:

- paracetamol 1 g QID
- fentanyl 12 µg/hour patch every 3 days
- calcium 600 mg BD
- gabapentin 100 mg nocte
- oxycodone 2.5 mg every 4 hours PRN for breakthrough pain
- pantoprazole 40 mg daily.

**QUESTION 5** 

Which of Ruby's medications can and/or should be continued?

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**QUESTION 6** 

Will pain be a problem for Ruby? How can it be managed in the nursing home?

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**QUESTION 7** 

What other symptoms associated with renal failure might require management?

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**CASE 4 ANSWERS**

**ANSWER 1**

Withdrawal from dialysis is a significant decision in Ruby's life and has very definite and serious implications for her life expectancy. It is vital that an urgent meeting is held with Ruby and her family to discuss the implications of dialysis withdrawal for Ruby. For additional information about communicating prognosis and end-of-life issues see *Resources for doctors* section.

**ANSWER 2**

A small study reviewing the charts of 35 patients who withdrew from dialysis reported a mean survival time of 10 days (range 1–48 days).<sup>1</sup> Other studies suggest that 75% of patients will die within 10 days or fewer following withdrawal from dialysis.<sup>2,3</sup> Ruby's prognosis without dialysis may also be affected by other intercurrent medical problems, her level of hydration and the presence or absence of hyperkalaemia.<sup>4</sup>

Withdrawal from dialysis is a common cause of death and in a 2008 Australian report<sup>5</sup> was reported as the cause of 35% of dialysis-related deaths.

**ANSWER 3**

Withdrawing from dialysis is not an act of euthanasia. Euthanasia is defined as a deliberate act with the intention of ending a person's life in the context of serious illness. Withdrawing from treatment when the burden to the patient is outweighed by any benefit is considered ethically, legally and medically valid.<sup>6</sup> Current guidelines specifically state that 'Withholding or withdrawing dialysis is not euthanasia. Equally it does not constitute physician-assisted suicide'.<sup>6</sup>

**ANSWER 4**

Palliative care focuses on attaining the best quality of life for the person with advanced disease, where further active interventions are considered futile as the risks outweigh the benefits.

Palliative care involves a careful and thorough clinical assessment, wise choice of symptom-directed care interventions and excellent nursing care, as well as spiritual and psychological support for the patient and close family members.

For Ruby, remaining in her nursing home, an environment that is familiar to her and being known to her carers, is likely to be of importance for her dignity, confidence, comfort and quality of life.

If Ruby's decision is to remain in her nursing home, community palliative care resources can be utilised to assist the general practitioner (GP) and nursing home staff to provide the best possible care. Given the goals of care in a palliative care setting, any fluid restrictions can be relaxed a little.

In palliative care settings, reduced renal function complicates the choice of medications and doses suitable for symptom control, as many drugs require the kidneys for effective elimination of their metabolites.

These considerations should be taken into account when prescribing medications for patients. Ruby will probably progress quickly to a stage when she will not be able to take her usual medications or even oral symptom-management drugs, which may be prescribed for her. Hence, parenteral medication options should be considered early.

### ANSWER 5

Paracetamol can be continued,<sup>7</sup> although Ruby may need to take it as an elixir if she has any difficulty swallowing. No dose adjustment is required in renal failure. Although not a potent analgesic, paracetamol is considered a much safer option than non-steroidal anti-inflammatory drugs (NSAIDs).<sup>8</sup>

Fentanyl is part of Ruby's chronic pain management and should be continued. Palliative care guidelines recommend use of fentanyl or methadone where opioid-based pain management is required in renal failure. Fentanyl and methadone are considered the safest opioids because they do not have active renally excreted metabolites.<sup>7,8</sup> Other opioids (eg morphine, hydromorphone, oxycodone and their metabolites) are cleared by dialysis and rapidly accumulate after stopping dialysis.<sup>4</sup>

A low dose of gabapentin can be continued with care for neuropathic pain as long as Ruby can swallow. Care must be taken with higher doses in renal failure,<sup>8</sup> as it accumulates and has significant adverse effects.<sup>4</sup> The contents of the capsule can be administered in yoghurt or something similar if Ruby's swallowing is impaired; however, there is no parenteral option.

Continued use of pantoprazole and calcium tablets is often limited by practical issues with swallowing and alertness so are often readily ceased.

### ANSWER 6

Many doctors anecdotally regard death from renal failure as painless; however, it is now becoming clear that pain is often a significant problem for patients with advanced renal disease.<sup>9</sup> Patients may experience pain from the discomfort associated with poor mobility, pressure areas or from other pre-existing medical conditions. With adequate palliative care and appropriate analgesia, death from ESRF/cessation of dialysis can still be 'good'.<sup>10</sup>

Ruby has chronic sciatica and there is the possibility of general discomfort arising from her increasing immobility and so management of pain will be important. Patients will also have difficulty taking oral analgesics as uraemia increases and alertness decreases.

As discussed previously, fentanyl and methadone are opioids without actively excreted renal metabolites and are, theoretically, the preferred options in renal failure.<sup>4,8,11</sup> However, fentanyl patches are very slow to titrate<sup>11</sup> and may be quite potent in opioid-naive patients. The parenteral formulation of fentanyl is also expensive and often difficult to access in the community. Methadone is very difficult to titrate,<sup>11</sup> so hydromorphone is usually the opioid of choice for parenteral use, with allowances made for reduced dose or dosing frequency.

For Ruby, it would be appropriate to continue her usual fentanyl patch and to use hydromorphone as a breakthrough analgesic at a

starting dose of 0.5 mg subcutaneously every 4 hours PRN. If multiple injections are required, a subcutaneously inserted butterfly needle can allow multiple doses of medication to be administered with minimal distress to the patient.

In an opioid-naive patient, hydromorphone 0.25–0.50 mg subcutaneously every 4 hours PRN would be an appropriate starting dose, remembering that in renal failure the duration of action may be significantly longer than the expected 4 hours. From a practical point of view hydromorphone is considered to be 5–7 times stronger than morphine.<sup>11</sup>

If a patient not usually on opioids were to need frequent PRN doses of analgesia, consideration should be given to commencing a regular dose via a syringe driver (*Figure 1*). This would also allow concurrent infusion of an antiemetic and/or sedative if these medications were required. A syringe driver is a battery-driven portable device that allows a 24-hour infusion of compatible medications to be infused evenly via a subcutaneous butterfly needle. Some nursing homes are proficient in the use of syringe drivers and have their own machines, but most community palliative care teams can lend a device and provide education to nursing home staff on its use.



Figure 1. Syringe driver

### ANSWER 7

Guidelines highlight that symptoms in patients with end-stage kidney disease are consistently under-assessed and not adequately managed.<sup>7</sup> Symptoms that might need to be managed in renal failure are discussed below.

#### Nausea and vomiting

Haloperidol is recommended for uraemia-related nausea and vomiting. The starting dose is 0.5 mg twice daily subcutaneously PRN, up to a total daily dose of 7.5 mg.<sup>12</sup> If used regularly, haloperidol could be added to other medications, such as hydromorphone, in a syringe driver.

Metoclopramide should be used with caution in renal failure because of a higher risk of extrapyramidal side effects, although it may still help if gastroparesis was a problem. Note, the maximum dose is 30 mg in 24 hours. Metoclopramide can be used in syringe driver combinations.<sup>13</sup>

#### Dyspnoea

Low-dose opioids, as per pain recommendations, are appropriate medications for management of dyspnoea.<sup>10,14</sup>

For anxiety and panic associated with dyspnoea, sublingual lorazepam at a dose of 0.5–1.0 mg every 4 hours PRN provides quick relief and is shorter acting than clonazepam.<sup>15</sup>

For increasingly more significant distress, clonazepam drops 0.25–0.5 mg every 6 hours sublingually can be used. Alternatively, midazolam 2.5 mg every 2 hours subcutaneously PRN, although more sedating and possibly amnesic for a short while, can be very helpful. Midazolam can also be added to a syringe driver for terminal agitation and its dose titrated to effect.

### Uraemic pruritus (itch)

This can be difficult to resolve and in the terminal phase medications that may be used usually provide sedation. Ensure that the patient does not have another cause for itch such as allergies or scabies.<sup>7</sup> Liberal use of bland skin moisturisers is advised. Gabapentin in low dose may be of some benefit early.<sup>7</sup>

### Restless legs syndrome

Antiparkinsonian drugs, such as levodopa, or gabapentin at low doses may be helpful; however, the patient may not be able to take these orally for very long.<sup>16</sup> Occasionally, metoclopramide can cause restlessness, so if it is being used, it might need to be stopped.

In a rapidly deteriorating situation, management of restless legs with parenteral benzodiazepines may be the most appropriate option as in palliative sedation for myoclonus or terminal agitation. Clonazepam can be used orally or sublingually (oral liquid) once or twice daily in 0.5–1 mg doses, or subcutaneously (injection formulation) up to 4 mg a day in divided doses.

### Terminal agitation

Haloperidol and clonazepam can be titrated for comfort with increased and PRN doses, but in Ruby's case, the midazolam dose could be titrated up as required, incorporating it into the regular syringe driver regime.

At some point, the increasing uraemia will itself produce significant sedation.

Hyoscine and/or glycopyrrolate for 'death rattles', if they occur, are compatible with hydromorphone, midazolam and haloperidol in a syringe driver.

It is important to note that although there is a parenteral formulation of clonazepam, it is usually given intermittently as a subcutaneous injection. This is because 50% of the clonazepam is absorbed into the infusion tubing if it made of polyvinyl chloride, and as the drug has a long half-life, intermittent doses are an effective option.<sup>17</sup>

## REFERENCES

1. Chater S, Davison SN, Germain MJ, Cohen LM. Withdrawal from dialysis: a palliative care perspective. *Clin Nephrol* 2006;66:364–72.
2. Cohen LM, Germain M, Poppel DM, Woods A, Kjellstrand CM. Dialysis discontinuation and palliative care. *Am J Kidney Dis* 2000;36:140–44.
3. Cohen LM, Germain M, Poppel DM, Woods A, Pekow P, Kjellstrand CM. Dying well after discontinuing the life-support treatment of dialysis. *Arch Intern Med* 2000;160:2513–18.

4. Palliative Care Expert Group. Renal failure in patients receiving palliative care. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 9 April 2014].
5. McDonald S, Excell L, Livingstone B, editors. The thirty-first report. Adelaide: Australian and New Zealand Dialysis and Transplant Registry; 2008. Available at [www.anzdata.org.au/anzdata/AnzdataReport/31stReport/FullReport.pdf](http://www.anzdata.org.au/anzdata/AnzdataReport/31stReport/FullReport.pdf) [Accessed 29 April 2014].
6. Cameron S, Brennan F. Legal issues concerning withholding and withdrawal of dialysis. *Nephrology* 2013;13:444–49.
7. Brennan F, Siva B, Crail S. Appropriate assessment of symptom burden and provision of patient information. *Nephrology* 2013;13:418–21.
8. Cervelli MJ, editor. The Renal Drug Reference Guide 1st Edition. Adelaide: South Australia Kidney Health; 2007. Available at [www.renaldrugreference.com.au](http://www.renaldrugreference.com.au) [Accessed 29 April 2014].
9. Murtagh F, Weisbord SD. Symptoms in renal disease: their epidemiology, assessment and management. In: Chambers EJ, Brown E, Germain M, editors. Supportive care for the renal patient. 2nd ed. Oxford: Oxford University Press; 2011.
10. Fassett RG, Robertson IK, Mace R, Youl L, Challenor S, Bull R. Palliative care in end-stage kidney disease. *Nephrology* 2011;16:4–12.
11. Palliative Care Expert Group. Pharmacological treatment of pain in patients receiving palliative care: use of individual opioids. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 9 April 2014].
12. Palliative Care Expert Group. Nausea and vomiting in patients receiving palliative care. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 9 April 2014].
13. A Katalin Urban. Models of care – end of life pathways. *Nephrology* 2013;13:425–30.
14. Palliative Care Expert Group. Pharmacological care emergencies: acute airways obstruction. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 9 April 2014].
15. McLeod R, Vella-Brincat J, Macleod S. The Palliative Care Handbook, 7th edition. Sydney: Hammond Press; 2013.
16. Neurology Expert Group. Periodic limb movements of sleep/wakefulness and restless legs syndrome. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 14 April 2014].
17. Twycross R, Wilcock A. Palliative care formulary. 4th ed. Nottingham: Palliativedrugs.com; 2014

## RESOURCES FOR PATIENTS

- The Renal Resource Centre. An introduction to conservative care of advanced kidney disease. Renal Resource Centre. [www.renalresource.com/pdf/IntroCCACKD.pdf](http://www.renalresource.com/pdf/IntroCCACKD.pdf)
- The Renal Resource Centre. Withdrawing from Dialysis Treatment. [www.renalresource.com/pdf/Withdrawing%20from%20Dialysis%20Treatment%20RENAL%20RESOURCE%20CENTRE.pdf](http://www.renalresource.com/pdf/Withdrawing%20from%20Dialysis%20Treatment%20RENAL%20RESOURCE%20CENTRE.pdf)
- [www.racgp.org.au/guidelines/advancecareplans](http://www.racgp.org.au/guidelines/advancecareplans)
- <http://advancecareplanning.org.au>
- [www.health.nsw.gov.au/patients/acp/pages/default.aspx](http://www.health.nsw.gov.au/patients/acp/pages/default.aspx)
- [www.renalresource.com](http://www.renalresource.com)

## RESOURCES FOR DOCTORS

- Clayton JM, Hancock KM, Butow PN, Tattersall MHN, Currow DC. Clinical practice guidelines for communicating prognosis and end-of-life issues with adults in advanced stages of a life-limiting illness, and their caregivers. [www.mja.com.au/journal/2007/186/12/clinical-practice-guidelines-communicating-prognosis-and-end-of-life-issues-adults?0=ip\\_login\\_no\\_cache%3Ddd452decf475a7b0eeef24b835d121e](http://www.mja.com.au/journal/2007/186/12/clinical-practice-guidelines-communicating-prognosis-and-end-of-life-issues-adults?0=ip_login_no_cache%3Ddd452decf475a7b0eeef24b835d121e)

**CASE 5**

**IRENE'S BLOOD PRESSURE IS DIFFICULT TO CONTROL**

Irene, 59 years of age, is new to your practice and has presented for renewal of her prescriptions. She reports a history of hypertension and has osteoarthritis in both knees. Her current medications are amlodipine 10 mg once daily, metoprolol 50 mg twice daily and hydrochlorothiazide 12.5 mg daily. On examination, her seated blood pressure, using an appropriately sized cuff, is 164/88 mmHg in the right arm and 140/86 mmHg in the left arm with no postural drop.

**QUESTION 1** 

Does Irene have resistant hypertension?

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**QUESTION 2** 

What other enquiries should you make in your history and look for in your examination as you assess Irene's hypertension?

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**QUESTION 3** 

Which arm blood pressure recording should be used to guide Irene's medical management?

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**FURTHER INFORMATION**

Irene's hypertension was first diagnosed 5 years ago during a 'wellness check' at work and was subsequently confirmed by three blood pressure measurements by her previous general practitioner (GP), after which she was commenced on amlodipine 5 mg daily. Her clinic and home blood pressure readings were 'all right' until last year when her blood pressure control worsened, necessitating an increase in her amlodipine dose (12 months ago) and subsequent commencement of metoprolol (6 months ago) and a thiazide diuretic (2 months ago). She reports good compliance with her medication. She is an ex-smoker (30 pack years), drinks 1–2 glasses of red wine with her evening meal, does not exercise regularly because of her knees and eats a lot of processed foods. Her father was diagnosed with type 2 diabetes at 48 years of age and had a myocardial infarction at 52 years of age. The only significant findings on physical examination were silver wiring of her retinal vessels, heart rate of 54 beats per minute, body mass index (BMI) of 31 kg/m<sup>2</sup> and waist circumference of 95 cm. Irene states that she has had some blood tests performed previously, but does not recall the results.

**QUESTION 4** 

How would you further investigate Irene?

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**FURTHER INFORMATION**

Irene returns 1 week later for a follow-up. A repeat blood pressure measurement in her right arm is 166/95 mmHg. Her 24-hour ambulatory blood pressure monitoring shows the following:

- daytime average 162/90 mmHg
- night-time average 156/85 mmHg
- 24-hour average 159/88 mmHg.

Irene's blood tests reveal:

- sodium 136 mmol/L (reference range 135–150)
- potassium 3.8 mmol/L (3.5–5.0)
- urea 12.6 mmol/L
- creatinine 146 µmol/L (70–110)
- eGFR 38 mL/min/1.73 m<sup>2</sup>
- serum albumin concentration 38 g/L (32–40)
- total cholesterol 5.8 mmol/L
- HDL cholesterol 1.8 mmol/L
- LDL cholesterol 3.6 mmol/L
- triglycerides 1.6 mmol/L.

Her full blood count, the remainder of her biochemistry and her plasma aldosterone-to-renin ratio are normal. A urinalysis reveals 10 x 10<sup>6</sup>/L red and white cells and a UACR of 22 mg/mmol (<5.0 mg/mmol). Her ECG shows evidence of left ventricular hypertrophy. Records obtained from Irene's previous GP showed that 2 years ago her serum creatinine was 125 mmol/L and eGFR was 40 mL/min/1.73 m<sup>2</sup>.

**QUESTION 5** 

What is your diagnosis?

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**QUESTION 6** 

What is Irene's absolute cardiovascular risk (ie her risk of cardiovascular disease in the next 5 years)?

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**QUESTION 7** 

What is Irene's target blood pressure?

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**QUESTION 8** 

How should Irene's BP be managed?

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**QUESTION 9** 

What additional investigations should be performed?

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**QUESTION 10** 

Should Irene be referred to a nephrologist?

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**QUESTION 11** 

Should Irene be referred for renal denervation?

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**CASE 5 ANSWERS**

**ANSWER 1**

Irene may have resistant hypertension, which is defined as blood pressure that remains above 140/90 mmHg in spite of concurrent use of three antihypertensive drugs from different classes (including a diuretic) for at least 1 month.<sup>1</sup> Although Irene meets these criteria, it is important to first exclude pseudo-resistant hypertension, which is due to poor blood pressure measuring technique, white coat effect or poor adherence to medication.<sup>1</sup> Careful measurement of Irene's blood pressure with an appropriately sized cuff and concordance of her home and clinic blood pressure measurements suggest it is not due to poor technique and white coat effect, respectively. Assessment of Irene's medication adherence would be very important before concluding that she has resistant hypertension.<sup>1</sup>

**ANSWER 2**

The aims of history taking and physical examination should be to establish:<sup>1</sup>

- the course and severity of hypertension
- the presence of end-organ damage (eg retinopathy, cardiac failure, proteinuria, reduced eGFR, CKD)
- cardiovascular risk
- the presence of clinical features suggestive of secondary hypertension, particularly chronic kidney disease (CKD), obstructive sleep apnoea and rare causes such as phaeochromocytoma, Cushing's syndrome, hypothyroidism and aortic coarctation.

With regards to history taking, it is important to know how Irene's hypertension was diagnosed, how well her blood pressure has been controlled, which antihypertensive agents have been trialled previously and, if relevant, the reasons for their discontinuation, how adherent she has been to her treatment regimen and whether she takes any medications known to increase blood pressure (*Table 1*).<sup>1</sup> It is also important to determine if Irene has a personal or family history of premature hypertension, kidney disease, diabetes, dyslipidaemia and/or cardiovascular disease.<sup>1</sup>

Lifestyle factors, such as sodium intake (processed food, added salt), physical activity, smoking, alcohol consumption and obesity, should be considered.<sup>1</sup>

**Table 1. Examples of medications/agents that interfere with blood pressure control<sup>2</sup>**

- NSAIDs: conventional and COX-2 (most common)
- Sympathomimetics (eg nasal, oral decongestants, amphetamines)
- Oral contraceptives
- Bupropion
- Corticosteroids
- Hepatic enzyme inducers (eg rifampicin, antidepressants, monoamine oxidase inhibitors)
- Calcineurin inhibitors (eg tacrolimus, cyclosporine)
- Cholestyramine
- Erythropoiesis stimulating agents (eg erythropoietin, darbepoetin)
- Licorice
- Caffeine
- Alcohol
- Ginseng preparations
- Ephedra (ma huang)
- Melatonin

**ANSWER 3**

The National Heart Foundation guidelines<sup>1</sup> recommend measuring blood pressure in both arms at the first assessment. The discrepancy in measurements between Irene’s left and right arms probably reflects an atherosclerotic lesion in the arm with the lower reading (ie her left arm). Diagnosis and management should always be guided by the readings from the arm with the higher measurement (in this case, Irene’s right arm).<sup>1</sup>

**ANSWER 4**

Irene has hypertension and obesity, which are two of the seven recognised risk factors for CKD. The other risk factors are diabetes

mellitus, smoking, family history of CKD, established cardiovascular disease and Aboriginal or Torres Strait Islander origin.<sup>3,4</sup> The presence of any CKD risk factor warrants screening for the presence of CKD by requesting a urinary albumin-to-creatinine ratio (UACR) in a spot urine sample (preferably a first morning void) and an estimated glomerular filtration rate (eGFR), which is automatically reported by all Australian pathology laboratories with every serum creatinine request in an adult over the age of 18 years.<sup>4,5</sup>

Irene should also have blood tests for a full blood count, general biochemistry, fasting lipids, urine microscopy/culture and plasma aldosterone-to-renin ratio.<sup>6</sup> An aldosterone-to-renin ratio is best performed when patients are not taking any interfering drugs, including diuretics, beta-blockers, angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs). Verapamil, hydralazine and prazosin do not interfere with the test. However, the investigation still has a good negative predictive value (for ruling out primary hyperaldosteronism) even when patients are on interfering medication.<sup>6</sup> Irene should also have an ECG (or possibly an echocardiograph) to investigate for cardiac structure-function abnormalities (particularly left ventricular hypertrophy).

Ambulatory blood pressure monitoring is often useful in situations where there is unusual variation between blood pressure readings in the clinic, suspected white coat hypertension (eg clinic hypertension in a person without evidence of target-organ damage), suspected hypotensive episodes (eg in those who are elderly or have diabetes) or, as in Irene’s case, resistant hypertension.<sup>7</sup> Home blood pressure measurements are acceptable if ambulatory monitoring is not available. It is also important to try to obtain records of Irene’s previous blood tests.

Irene, therefore, has confirmed resistant hypertension with target-organ damage (CKD and left ventricular hypertrophy).

**Table 2. Risk of progressive CKD<sup>3</sup>**

Kidney function stage	GFR (mL/min/1.73 m <sup>2</sup> )	Albuminuria stage		
		Normal (UACR mg/mmol) Male: <2.5; Female: <3.5	Microalbuminuria (UACR mg/mmol) Male: 2.5–25; Female: 3.5–35	Macroalbuminuria (UACR mg/mmol) Male: >25; Female: >35
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present		
2	60–89			
3a	45–59			
3b	30–44			
4	15–29			
5	<15 or on dialysis			

Green = low; yellow = moderate; orange = high; red = very high

**ANSWER 5**

On the basis of eGFR <60 mL/min/1.73 m<sup>2</sup> for at least 3 months and albuminuria in the microalbuminuric range, Irene has stage 3b CKD with microalbuminuria (*Table 2*).

The underlying cause of her kidney disease is uncertain at this stage. The most common causes of CKD in the community are diabetic kidney disease (DKD), chronic glomerulonephritis, hypertensive nephrosclerosis (including renovascular disease) and polycystic kidney disease.<sup>8</sup> Irene's ambulatory blood pressure readings are considerably elevated above the upper reference limits (24-hour average 135/85 mmHg, daytime average 140/90 mmHg, night-time average 125/75 mmHg).

**ANSWER 6**

Irene's eGFR is <45 mL/min/1.73 m<sup>2</sup>, indicating that she is automatically in the highest absolute cardiovascular risk category (ie >15% of cardiovascular disease within the next 5 years).<sup>9</sup> Using the Australian Absolute Cardiovascular Risk Calculator ([www.cvdcheck.org.au/](http://www.cvdcheck.org.au/)) to assess Irene's risk level would lead to an erroneously low risk assessment (10% risk). *Table 3* indicates the conditions that denote very high cardiovascular risk and for which use of the Australian Absolute Cardiovascular Risk Calculator is inappropriate.

**Table 3. People at very high cardiovascular risk (>15% in 5 years) for whom use of the Australian Absolute Cardiovascular Risk Calculator is inappropriate<sup>9</sup>**

- Established cardiovascular disease
- Diabetes and age >60 years
- Familial hypercholesterolaemia
- Serum total cholesterol above 7.5 mmol/L
- Severe hypertension (systolic ≥180 mmHg, diastolic ≥110 mmHg)
- Diabetes and microalbuminuria
- <45 mL/min/1.73m<sup>2</sup> or persistent proteinuria

**ANSWER 7**

According to the Kidney Health Australia-Caring for Australasians with Renal Insufficiency (KHA-CARI) and Kidney Check Australia Taskforce (KCAT) guidelines,<sup>3,4</sup> Irene's blood pressure target is <130/80 mmHg (*Table 4*) as she has CKD with microalbuminuria.

**Table 4. Recommended BP targets<sup>9,10</sup>**

People with...	Blood pressure target (mmHg)
Albuminuria (micro- or macro-)	<130/80
Diabetes	<130/80
CKD	<140/90
All others	<140/90

**ANSWER 8**

Lifestyle modification is important in the management of Irene's blood pressure. She should be encouraged to follow a low salt (<100 mmol salt or 6 g salt or 2.3 g sodium per day), normal protein (0.75–1.0 g/

kg/day) diet, high in vegetables and fruit and low in saturated fats.<sup>4</sup> The KHA-CARI guidelines recommend that people with progressive CKD, particularly obese patients, should have an individualised diet intervention involving an accredited practicing dietician.<sup>4</sup> Irene should also exercise regularly and moderate her alcohol intake. Such lifestyle interventions can achieve blood pressure reductions of 5–20 mmHg.<sup>2</sup>

Irene will also require additional antihypertensive therapy. Given that she has proteinuric CKD, it would be appropriate to commence either an ACEI or ARB.<sup>9</sup> This could be prescribed as a combination medication with either the calcium channel blocker or the thiazide diuretic to minimise pill burden and optimise medication adherence. Initiation of an ACEI or ARB should always be followed by a repeat measurement of serum potassium and creatinine levels 5–7 days after commencement.<sup>11</sup> If hyperkalaemia occurs, it should be managed by dietary potassium restriction. Serum potassium levels <6.0 mmol/L are acceptable.<sup>11</sup>

An increase in serum creatinine following the commencement of an ARB or an ACEI is not unusual and is acceptable as long as the increase is ≤25% of the baseline value and stabilises within the first month.<sup>11</sup> If creatinine increases by >25% of baseline within the first month, the ACEI or ARB should be ceased, as it suggests that the patient has functionally significant bilateral renal artery stenosis.<sup>11</sup> If Irene's blood pressure remains poorly controlled, consideration should be given to adding other classes of antihypertensive agents, including alpha<sub>1</sub>-adrenoceptor antagonists, centrally acting alpha<sub>2</sub> adrenoceptor agonists and vasodilators. Consideration could also be given to changing her thiazide diuretic to a loop diuretic if her eGFR falls below 30 mL/min/1.73 m<sup>2</sup>.<sup>12</sup> The guiding principles for medical management of resistant hypertension are shown in *Table 5*.

**Table 5. Principles of management of resistant hypertension<sup>2</sup>**

- Check adherence – use combinations, minimise number of pills
- Add antihypertensive medications rather than substitute
- Maximise individual doses before adding another agent
- Choice of antihypertensive should primarily be determined by tolerability and comorbid conditions, as systematic reviews show no major differences between classes of medication with respect to reducing cardiovascular risk
- Night-time administration of once daily medications may be more effective
- ACEIs and ARBs should not be combined
- Patients with underlying CKD often require at least 3–4 antihypertensive agents to achieve control (particularly at lower eGFRs)
- Persist, encourage and be prepared to vary regimens
- Do not compromise on the target!
- Refer for nephrologist opinion if not at target and on ≥3 drugs

**ANSWER 9**

As Irene has confirmed CKD, the KHA-CARI guidelines recommend that it is important to consider the underlying cause and to pursue the diagnosis sufficiently to exclude treatable pathology, such as

obstruction, vasculitis and rapidly progressive glomerulonephritis.<sup>4</sup> Irene should have a renal tract ultrasound scan to assess the anatomy of her renal tract, specifically to assess kidney size and discrepancy and to exclude outflow obstruction. Her renal function should be monitored regularly. Although Irene's likely diagnosis is hypertensive nephrosclerosis, it is reasonable to request urine and serum electrophoresis and serum free light chains.

Investigating for the possibility of underlying renal artery stenosis (eg with a renal artery duplex or spiral computed tomography) is not recommended in Irene's case.<sup>13,14</sup> Two large randomised controlled trials have shown that endovascular intervention for either uni- or bilateral atherosclerotic renal artery stenoses did not improve blood pressure control, antihypertensive agent requirements or renal function deterioration.<sup>13,14</sup> Thus, investigation for renal artery stenosis should not be first line and should be reserved for rare cases associated with flash pulmonary oedema or rapidly deteriorating renal function.

### ANSWER 10

In line with the recommendations of the KCAT and the KHA-CARI Guidelines,<sup>3,4</sup> Irene should be referred to a nephrologist as she has CKD and hypertension that is difficult to get to target despite at least three antihypertensive agents (*Table 6*).

#### Table 6. Recommendations for referral to a specialist renal service or nephrologist<sup>3</sup>

Referral to a specialist renal service or nephrologist is recommended in the following situations:

- eGFR <30mL/min/1.73 m<sup>2</sup>
- Persistent significant albuminuria (UACR ≥30mg/mmol)
- A consistent decline in eGFR from a baseline of <60mL/min/1.73 m<sup>2</sup> (a decline >5mL/min/1.73 m<sup>2</sup> over a 6-month period, confirmed on at least three separate readings)
- Glomerular haematuria with macroalbuminuria
- CKD and hypertension that is difficult to get to target despite at least three anti-hypertensive agents.

### ANSWER 11

Renal denervation is aimed at reducing renally stimulated sympathetic activity; however, a recent randomised patient-blinded controlled trial of 535 participants failed to show a significant reduction in hypertension in patients treated with renal denervation.<sup>15</sup> Therefore, this therapy cannot be recommended.

### CONCLUSION

Irene was referred to a nephrologist. Her blood pressure was ultimately controlled following lifestyle modification and a combination of amlodipine, metoprolol, perindopril, frusemide and methyldopa.

### REFERENCES

1. National Heart Foundation. Guide to management of hypertension 2008 (updated December 2010). Available at [www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf) [Accessed 15 April 2014].

2. Johnson DW. Resistant Hypertension. *Australian Doctor* 2007;27:34 Available at [www.australiandoctor.com.au/cmspages/getfile.aspx?guid=cf6401c9-dc29-494f-85e7-5b0c099d96fa](http://www.australiandoctor.com.au/cmspages/getfile.aspx?guid=cf6401c9-dc29-494f-85e7-5b0c099d96fa) [Accessed 29 April 2014].
3. Johnson DW, Mathew TH, Ludlow M J, et al. (2012). Chronic Kidney Disease (CKD) Management in General Practice. *Kidney Health Australia*. Available at [www.kidney.org.au/LinkClick.aspx?fileticket=vfDcA4sEUMs=](http://www.kidney.org.au/LinkClick.aspx?fileticket=vfDcA4sEUMs=) [Accessed 29 April 2014].
4. Johnson DW, Atai E, Chan M, et al. KHA-CARI guideline: Early chronic kidney disease: detection, prevention and management. *Nephrology* 2013;18:340–50.
5. Johnson DW, Jones G R, Mathew TH, et al. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: new developments and revised recommendations. *Med J Aust* 2012;197:224–25.
6. Funder JW, Carey RM, Fardella C, et al. Case detection, diagnosis, and treatment of patients with primary aldosteronism: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab* 2008;93:3266–81.
7. National Heart foundation and High Blood Pressure Research Council of Australia Ambulatory Blood Pressure Monitoring Consensus Committee. Ambulatory blood pressure monitoring. *Aust Fam Physician* 2011;11:877–80.
8. Australia and New Zealand Dialysis and Transplant Registry. Summary of Australia and New Zealand Dialysis and Transplantation, 2012. Adelaide: ANZDATA; 2013. Available at [www.anzdata.org.au/anzdata/AnzdataReport/36thReport/2012\\_Summary\\_v1.pdf](http://www.anzdata.org.au/anzdata/AnzdataReport/36thReport/2012_Summary_v1.pdf) [Accessed 2 June 2014].
9. National Vascular Disease Prevention Alliance. Guidelines for the management of absolute cardiovascular disease risk. Canberra: National Health and Medical Research Council of Australia; 2012. Available at [http://strokefoundation.com.au/site/media/AbsoluteCVD\\_GL\\_webready.pdf](http://strokefoundation.com.au/site/media/AbsoluteCVD_GL_webready.pdf) [Accessed 16 April 2014].
10. James PA, Oparil S, Carter BL, et al. Evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA* 2014;311:507–20.
11. Johnson DW, Phoon R. Medical therapies to reduce chronic kidney disease progression and cardiovascular risk: anti-hypertensive agents. CARI guidelines. *Kidney Health Australia*. Available at [www.cari.org.au/CKD/CKD%20early/Medical\\_Th\\_Anti-platelet.pdf](http://www.cari.org.au/CKD/CKD%20early/Medical_Th_Anti-platelet.pdf) [Accessed 2 June 2014].
12. Kidney Disease Outcomes Quality Initiative (K/DOQI). K/DOQI clinical practice guidelines on hypertension and antihypertensive agents in chronic kidney disease. *Am J Kidney Dis* 2004;43(Suppl 1):S1–290. Available at [www.kidney.org/professionals/kdoqi/guidelines\\_bp/guide\\_12.htm](http://www.kidney.org/professionals/kdoqi/guidelines_bp/guide_12.htm) [Accessed 2 June 2014].
13. Cooper C J, Murphy TP, Cutlip DE, et al. Stenting and medical therapy for atherosclerotic renal-artery stenosis. *N Engl J Med* 2014;370:13–22.
14. Wheatley AK, Ives N, Gray R, et al. Revascularization versus medical therapy for renal-artery stenosis. *N Engl J Med* 2009;361:1953–62.
15. Bhatt DL, Kandzari DE, O'Neill WW, et al. A controlled trial of renal denervation for resistant hypertension. *N Engl J Med* 2014;370:1393–1401.

### RESOURCES FOR DOCTORS

- Johnson DW. Resistant Hypertension. *Australian Doctor* 2007;27:34 [www.australiandoctor.com.au/cmspages/getfile.aspx?guid=cf6401c9-dc29-494f-85e7-5b0c099d96fa](http://www.australiandoctor.com.au/cmspages/getfile.aspx?guid=cf6401c9-dc29-494f-85e7-5b0c099d96fa)
- [www.kidney.org.au/LinkClick.aspx?fileticket=vfDcA4sEUMs%3d&tabid=635&mid=1584](http://www.kidney.org.au/LinkClick.aspx?fileticket=vfDcA4sEUMs%3d&tabid=635&mid=1584).

**CASE 6**

**ANNA HAS NAUSEA AND INCREASED URINARY FREQUENCY**

Anna, aged 29 years, has been attending your practice since she and her husband moved to your large rural town 5 years ago. Anna is currently 8 weeks pregnant and today you are reviewing the results of her routine antenatal tests, which were done last week. Her blood results are normal. The results of her midstream urine (MSU) are shown in *Table 1*.

Anna is feeling well apart from mild nausea in the mornings. She has had an increase in urinary frequency since being pregnant, but no dysuria or suprapubic pain. Anna is normally very healthy and has no significant past medical history.

**Table 1. Anna's MSU results**

Chemistry	Microscopy	Culture
pH: 5.5	Leucocytes: 8 x 10 <sup>6</sup> /L (<10)	Organism 1: <i>Escherichia coli</i> >10 <sup>8</sup> organisms/L
Protein: nil	Erythrocytes: 5 x 10 <sup>6</sup> /L (<10)	
Glucose: nil	Epithelial cells: ++	
Blood: nil		
Antimicrobial sensitivity		
Ampicillin/amoxicillin	R (resistant)	
Amoxicillin/clavulanic acid	S (sensitive)	
Cephalexin	S	
Trimethoprim	S	
Nitrofurantoin	S	
Norfloxacin	S	

**QUESTION 1** 

How would you interpret Anna's MSU results?

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**FURTHER INFORMATION**

Anna's repeat MSU results and antimicrobial sensitivity are shown in *Table 2*.

**Table 2. Results of repeat MSU**

Chemistry	Microscopy	Culture
pH: 5.4	Leucocytes: 2 x 10 <sup>6</sup> /L (<10)	Organism 1: <i>Escherichia coli</i> >10 <sup>8</sup> organisms/L
Protein: nil	Erythrocytes: 3 x 10 <sup>6</sup> /L (<10)	
Glucose: nil	Epithelial cells: nil	
Blood: nil		
Antimicrobial sensitivity		
Ampicillin/amoxicillin	R (resistant)	
Amoxicillin/clavulanic acid	S (sensitive)	
Cephalexin	S	
Trimethoprim	S	
Nitrofurantoin	S	
Norfloxacin	S	

**QUESTION 2** 

How would you manage Anna, given the above results?

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**FURTHER INFORMATION**

Anna completes the recommended treatment and her follow-up MSU result is normal. Her pregnancy progresses normally until 26 weeks, when she presents feeling 'terrible'. She says she has felt feverish for 24 hours, is nauseated and has pain on her left side. The baby seems to be moving normally. On examination she has a temperature of 38.8°C, is mildly dehydrated and has left loin tenderness. Fundal height is appropriate for dates and the fetal heart rate is approximately 140 beats per minute.

**QUESTION 3** 

What is your diagnosis? How will you manage Anna?

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**FURTHER INFORMATION**

Anna is discharged 4 days after her admission. She has been prescribed nitrofurantoin 100 mg 12-hourly.

**QUESTION 4** 

What follow up, if any, does she need during this pregnancy, with respect to her urine infection?

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**FURTHER INFORMATION**

Anna has a normal delivery, giving birth to a healthy baby boy, Thomas, at 39 weeks gestation. She comes to see you for a postnatal checkup 6 weeks later. She is feeling well and has no urinary symptoms.

**QUESTION 5** 

Does Anna require any further investigation for the UTIs she had during pregnancy?

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**CASE 6 ANSWERS**

**ANSWER 1**

The presence of significant numbers of epithelial cells indicates contamination of the urine sample with cells from the distal urethra and/or perineum.<sup>1</sup> Contaminated samples are frequently associated with false-positive urine culture results, as epithelial cells can carry large numbers of bacteria. Guidelines suggest that a positive culture in the presence of raised epithelial cells should be interpreted with caution.<sup>1</sup> The urine test should be repeated with a carefully collected MSU sample.<sup>1,2</sup>

**ANSWER 2**

Anna has asymptomatic bacteriuria, which is defined as bacteriuria (>100,000/mL) in the absence of specific symptoms of a urinary tract infection (UTI).<sup>2</sup> Women should be offered routine screening for asymptomatic bacteriuria by MSU culture early in pregnancy.<sup>3,4</sup> Dipstick testing (for leucocytes and nitrites) and urine microscopy alone (for the presence of bacteria and leucocytes) are screening methods shown to have high false-negative rates<sup>5</sup> and should not be substituted for microscopy and culture. Screening and treatment of bacteriuria has been shown to be cost-effective.<sup>6</sup>

According to UK studies, the incidence of asymptomatic bacteriuria in pregnant women is 2–5%.<sup>7</sup> The risk is increased in those with a history of diabetes, previous UTIs or structural abnormalities of the urinary tract, and in mothers with lower socioeconomic status.<sup>2</sup> If untreated, up to 30% of mothers may develop acute cystitis and up to 50% may develop acute pyelonephritis.<sup>8</sup>

*Escherichia coli* is the most common pathogen isolated (approximately 80% of cases) in urine samples. Other pathogens include *Staphylococcus saprophyticus*, which is the second most common pathogen, as well as other gram-negative bacteria.<sup>2</sup> Group B *Streptococcus*, although isolated less often, can cause asymptomatic bacteriuria in pregnancy, and women with this finding require treatment with penicillin during labour to help prevent neonatal sepsis.<sup>2,8</sup>

Asymptomatic bacteriuria is associated with low birth weight and preterm delivery. A 2007 Cochrane review of 14 studies showed that treatment of asymptomatic bacteriuria with antibiotics reduces the risk of pyelonephritis and low birth weight but did not show a significant difference in the incidence of premature delivery.<sup>9</sup>

Anna should be treated with an antibiotic according to her sensitivity testing results and taking into account any allergies. Appropriate choices are:<sup>10</sup>

- cephalixin, 500 mg 12-hourly for 5 days (TGA pregnancy category A)

**OR**

- nitrofurantoin, 100 mg 12-hourly for 5 days (TGA pregnancy category A)

**OR**

- amoxicillin 500 mg + clavulanic acid 125 mg, 12-hourly for 5 days (TGA pregnancy category B1)

Although urinary alkalinisers are considered safe in pregnancy, they should not be used in combination with nitrofurantoin as this can result in a loss of treatment efficacy.<sup>2,11</sup>

A repeat urine culture should be performed at least 48 hours after treatment is completed to confirm clearance of the infection.<sup>10</sup>

### ANSWER 3

Anna has developed pyelonephritis, which is defined as significant bacteriuria in the presence of systemic illness and symptoms such as flank or renal angle pain, pyrexia, rigors, nausea and vomiting.<sup>2</sup> Pregnant women with fever and pyelonephritis require hospital admission as there is a risk of preterm labour and maternal renal complications.<sup>2,12</sup> Investigations usually include blood cultures; full blood evaluation; urea; electrolytes and creatinine (UEC); vaginal swab; MSU and urinalysis for proteinuria.<sup>2</sup> Fetal wellbeing should be assessed with cardiotocography (CTG) monitoring. A specialist obstetrician should be involved if possible because of the risk of preterm labour. Consideration may need to be given to the use of antenatal steroids and/or tocolysis.<sup>8</sup>

Anna should be commenced on empirical intravenous antibiotics until sensitivity testing is available.<sup>13</sup> Intravenous gentamycin 4–6mg/kg for one dose then subsequent doses, determined by renal function, plus intravenous amoxicillin/ampicillin 2 g 6-hourly is recommended. In cases of penicillin allergy, gentamycin alone is usually sufficient. If gentamycin is contraindicated, intravenous ceftriaxone or cefotaxime are suitable alternatives.<sup>13</sup>

Parenteral treatment should continue until the patient is afebrile for at least 24 hours, after which oral therapy can be commenced, guided by susceptibility testing results.<sup>13</sup>

### ANSWER 4

It is recommended that oral antibiotics are continued for 10–14 days, but this may need to be extended up to 21 days if improvement is slow.<sup>13</sup> A repeat MSU should be done 48 hours after ceasing treatment to check that the infection has been cleared,<sup>13</sup> and then at every subsequent pregnancy visit.<sup>2</sup>

There is no consensus regarding the use of prophylactic antibiotics for recurrent UTIs in pregnancy. Some guidelines recommend antibiotic prophylaxis after two or more documented separate episodes of cystitis or pyelonephritis.<sup>2</sup> Suitable antibiotic options are:<sup>2</sup>

- nitrofurantoin 50 mg oral at night

Note, caution should be exercised when administering nitrofurantoin at term or in cases of possible preterm birth. In these situations there is the possibility of producing haemolytic anaemia in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency and due to immature enzyme systems in the early neonatal period.

OR

- cephalexin 250 mg oral at night

OR

- trimethoprim 150 mg oral at night.

Avoid use in the first trimester and in pregnant women with established folate deficiency, low dietary folate intake, or in women taking folate antagonists.

A Cochrane review of 24 studies investigating the effectiveness of cranberry products for the prevention of recurrent UTIs has recently been published. The meta-analysis showed that cranberry products did not significantly reduce the occurrence of symptomatic UTI overall or for the subgroup of pregnant women with recurrent UTIs.<sup>14</sup>

### ANSWER 5

The incidence of asymptomatic bacteriuria in healthy, non-pregnant premenopausal women is 1–5%.<sup>16</sup> Treatment of asymptomatic bacteriuria does not decrease the frequency of symptomatic UTI or prevent further episodes of bacteriuria.<sup>15</sup> Additionally, asymptomatic bacteriuria has not been shown to be associated with detrimental long-term outcomes (eg hypertension, renal failure, genitourinary cancer or decreased survival).<sup>16</sup> For these reasons, screening for or treatment of asymptomatic bacteriuria in premenopausal non-pregnant women is not recommended.<sup>4,16</sup> Following guideline recommendations and not treating asymptomatic bacteriuria helps reduce antimicrobial resistance.<sup>17</sup>

Anna is asymptomatic after the birth of her baby and does not require a screening MSU at this visit. She should be screened as early as possible in any subsequent pregnancies, as 6–8% of women experience recurrent pyelonephritis during a subsequent pregnancy.<sup>18–20</sup>

People with structural abnormalities of the urinary tract are more likely to develop an upper urinary tract infection. Referral for investigation of an underlying abnormality of the renal tract is advisable for:<sup>12</sup>

- men, following their first episode of acute pyelonephritis
- women, following two or more episodes of acute pyelonephritis
- all people with a urinary tract infection caused by a *Proteus* species.

There are no commonly accepted guidelines regarding referral for investigation of an underlying abnormality of the renal tract for women develop pyelonephritis during pregnancy. In pregnancy, significant physiological changes occur in the urogenital tract (eg bladder volume increases and detrusor tone decreases), which increase the potential for pathogenic colonisation. Most pregnant women develop ureteric dilatation due to a combination of progestogenic relaxation of ureteric smooth muscle and pressure from the expanding uterus. The overall effect is increased urinary stasis, compromised ureteric valves and vesicoureteric reflux, facilitating bacterial colonisation and ascending infection.<sup>8</sup> In the absence of evidence-based guidelines, the general practitioner will need to use clinical judgement when considering investigation for underlying renal tract abnormality. Urinary tract ultrasonography could be considered, whereas computed tomography (CT) urogram has a higher cost and involves significant radiation exposure and probably cannot be justified.

## REFERENCES

1. Nottingham University Hospitals Trust. Guide to Interpretation of Microbiological Tests. Available at [www.nuh.nhs.uk/healthcare-professionals/microbiology/guide-to-interpretation/](http://www.nuh.nhs.uk/healthcare-professionals/microbiology/guide-to-interpretation/) [Accessed 24 March 2014].
2. South Australian Perinatal Practice Guidelines. Urinary tract infections in pregnancy. Adelaide: Department of Health, Government of South Australia. Available at [www.sahealth.sa.gov.au/wps/wcm/connect/Public+Content/SA+Health+Internet/Clinical+resources/Clinical+topics/Perinatal+practice+guidelines/](http://www.sahealth.sa.gov.au/wps/wcm/connect/Public+Content/SA+Health+Internet/Clinical+resources/Clinical+topics/Perinatal+practice+guidelines/) [Accessed 29 April 2014].
3. National Institute for Health and Clinical Excellence. Antenatal Care June 2010/ NICE Clinical Guideline 62. Available at [www.nice.org.uk/nicemedia/live/11947/40115/40115.pdf](http://www.nice.org.uk/nicemedia/live/11947/40115/40115.pdf) [Accessed 23/03/2014].
4. Antibiotic Expert Group. Urinary tract infections: Asymptomatic bacteriuria In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd: 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 8 April 2014].
5. McNair RD, MacDonald SR, Dooley SL, Peterson LR. Evaluation of the centrifuged and Gram-stained smear, urinalysis, and reagent strip testing to detect asymptomatic bacteriuria in obstetric patients. *Am J Obstet Gynecol* 2000;182:1076–79.
6. Wadland W, Plante D. Screening for asymptomatic bacteriuria in pregnancy. *J Fam Pract* 1989;29:372–76.
7. Foley ME, Farquharson R, Stronge JM. Is screening for bacteriuria in pregnancy worthwhile? *Br Med J* 1987;295:270.
8. McCormick T, Ashe RG, Kearney PM. Urinary tract infection in pregnancy. *Obstet Gynaecol* 2008;10:156–62.
9. Smail FM, Vazquez JC. Antibiotics for asymptomatic bacteriuria in pregnancy. Cochrane Pregnancy and Childbirth Group. Available at <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD000490.pub2/full> [Accessed on 24/03/14].
10. Antibiotic Expert Group. Urinary tract infections: Acute cystitis in adults. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd: 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 17 March 2014].
11. Scottish Intercollegiate Guidelines Network (SIGN). Sign 88: Management of suspected bacterial urinary tract infection in adults. A national clinical guideline. Edinburgh: SIGN; 2012. Available at [www.sign.ac.uk/guidelines/fulltext/88/recommendations.html](http://www.sign.ac.uk/guidelines/fulltext/88/recommendations.html) [Accessed 29 April 2014].
12. National Institute for Health and Clinical Excellence. Clinical Knowledge Summary. Pyelonephritis – acute. [Last revised June 2013]. Available at <http://cks.nice.org.uk/pyelonephritis-acute/#scenariorecommendation> [Accessed on 24 March 2014].
13. Antibiotic Expert Group. Urinary tract infections: Acute pyelonephritis in adults. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd: 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 17 March 2014].
14. Jepson RG, Williams G, Craig JC. Cranberries for preventing urinary infections. Cochrane Renal Group. Available at <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD001321.pub5/full> [Accessed 23/03/2014].
15. Bengtsson C, Bengtsson U, Björkelund C, et al. Bacteriuria in a population sample of women: 24-year follow-up study. Results from the prospective population-based study of women in Gothenburg, Sweden. *Scand J Urol Nephrol* 1998;32:284–89.
16. Cogan R, Nicolle LE, McGlone A, Hooton TM. Asymptomatic bacteriuria in adults. *Am Fam Physician* 2006;74:985–90.
17. Lin E, Bhusal Y, Horwitz D, et al. Overtreatment of enterococcal bacteriuria. *Arch Intern Med* 2012;172:33–38.
18. Wing DA, Hendershott CM, Debuque L, Millar LK. A randomized trial of three antibiotic regimens for the treatment of pyelonephritis in pregnancy. *Obstet Gynecol* 1998;92:249–53.
19. Lenke RR, VanDorsten JP, Schiffrin BS. Pyelonephritis in pregnancy: a prospective randomized trial to prevent recurrent disease evaluating suppressive therapy with nitrofurantoin and close surveillance. *Am J Obstet Gynecol* 1983;146:953–57.
20. Harris RE, Gilstrap LC 3rd. Prevention of recurrent pyelonephritis during pregnancy. *Obstet Gynecol* 1974;44:637–41.

## Renal Problems

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 <http://gplearning.racgp.org.au>
- log into the *gplearning* website at <http://gplearning.racgp.org.au> and answer the following 10 multiple choice questions (MCQs) online
- 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 <http://gplearning.racgp.org.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

Jane, aged 25 years, is 7 weeks pregnant. Her recent MSU result following a routine antenatal visit showed the following:

- microscopy: leucocytes  $9 \times 10^6/L$  (normal  $<10$ )
- erythrocytes  $2 \times 10^6/L$  ( $<10$ )
- epithelial cells 0
- culture: Org 1: *E. coli*  $>10^8$  organisms/L
- antimicrobial sensitivity: ampicillin/amoxicillin R (resistant); amoxicillin/clavulanic acid S (sensitive); cephalexin S; trimethoprim S; nitrofurantoin S; norfloxacin S. She has no urinary symptoms.

Which of the following options would be appropriate management for Jane?

- No further action is required with respect to this MSU result.
- Repeat the MSU test.
- Prescribe trimethoprim 300 mg daily for 5 days.
- Prescribe cephalexin 500 mg twice daily for 5 days.
- Prescribe cephalexin 1000 mg twice daily for 10 days.

### QUESTION 2

People with resistant hypertension require multiple antihypertensive agents to bring their blood pressure within target or close to target.

Which of the following statements regarding hypertension is CORRECT?

- Resistant hypertension is defined as blood pressure that remains above 130/80 mmHg in spite of the concurrent use of three antihypertensive drugs.
- Melatonin and ginseng preparations do not affect blood pressure.
- Sympathomimetics (eg nasal and oral decongestants, amphetamines) and oral contraceptives do not interfere with blood pressure control.
- Diagnosis and management of hypertension should always be guided by the readings from the arm with the higher measurement.
- Alcohol use does not affect blood pressure control.

### QUESTION 3

People with diabetes are at increased risk of developing diabetic kidney disease (DKD). Which of the following statements regarding DKD is the most CORRECT?

- The Nephron study reported that every second patient with diabetes will most probably have DKD.
- Risk factors for DKD, other than diabetes, include having a strong family history of DKD (especially involvement of a first-degree relative), being overweight or obese and being over 60 years of age.
- Levels of DKD have remained stable in the last 2 decades in Australia.
- Statements A, B and C are correct.
- Statements A and B are correct.

### QUESTION 4

Melanie, 84 years of age, is a nursing home resident who wants to stop attending outpatient dialysis sessions. Melanie was an only child and never married. She has no known family/relatives. Past medical history includes polio at age 6 years, acute myocardial infarct (10 years ago), heart failure diagnosed 2 years ago and several transient ischaemic attacks/strokes in the last 3 months, which have reduced her mobility and rendered her bed- and chair-bound. She experiences major pain with any movement. Which of the following statements is correct?

- Allowing Melanie to withdraw from dialysis is tantamount to euthanasia.
- Without dialysis Melanie would be expected to have a mean survival time of 10 days.
- Withdrawal from dialysis is considered a rare cause of death in Australia.
- Withholding or withdrawing dialysis is 'physician-assisted suicide'.
- Melanie's prognosis without dialysis will not be influenced by any other factors (eg illness).

**QUESTION 5**

Pain is a common symptom in patients with chronic kidney disease (CKD). Which of the following statements regarding pain management in people with chronic kidney disease is the most CORRECT?

- A. The principles of pain management are to treat the cause of the pain using non-opioid analgesia initially.
- B. Paracetamol should not be used in people with CKD.
- C. NSAIDs are suitable for people with chronic kidney disease when used at reduced doses.
- D. Morphine can be safely used for people with CKD.
- E. Fentanyl, which is renally excreted, should not be used in CKD.

**QUESTION 6**

Jennifer, aged 72 years, presents with swelling of both ankles. The problem arose after her routine flu immunisation 6 weeks ago. Examination reveals pitted oedema half-way up her knees. She was diagnosed with diabetes mellitus 12 years ago for which she receives insulin. Hypertension was diagnosed 15 years ago for which she received a low dose diuretic and an ACEI inhibitor until recently when she was also prescribed amlodipine. Recent reports indicate an eGFR of 41 mL/min/1.73 m<sup>2</sup> and a urine albumin:creatinine ratio of 31 mg/mmol. Which of the following statements is the most CORRECT?

- A. Jennifer's medication(s) could be contributing to her swollen ankles.
- B. Jennifer's swollen ankles could be symptomatic of a new and as yet undiagnosed illness.
- C. On the basis of the presenting information, Jennifer has CKD.
- D. All of the above.
- E. None of the above.

**QUESTION 7**

Which of the following statements CORRECTLY describes the management of symptoms associated with CKD and/or end-stage kidney disease (ESKD)?

- A. Fatigue is common, with the main reversible cause being anaemia secondary to reduced erythropoietin (EPO) synthesis; it may be treated with synthetic EPO-analogues.
- B. Nausea is common in ESKD; the preferred first-line treatment is levomepromazine.
- C. Pruritus should be treated with gabapentin or antihistamines.
- D. Dyspnoea should be managed using non-pharmacological options.
- E. Restless legs syndrome is treated with gabapentin at normal doses.

**QUESTION 8**

Which one of the following statements about nephrotic syndrome is CORRECT?

- A. Nephrotic syndrome is only caused by primary glomerulonephritis.
- B. Oedema, heavy proteinuria, hypoalbuminaemia and hypercholesterolaemia are features suggestive of the nephrotic syndrome.
- C. Diabetes mellitus is not implicated in nephrotic syndrome.
- D. Hepatitis is not implicated in nephrotic syndrome.
- E. Cancer is not a cause of nephrotic syndrome.

**QUESTION 9**

Anne had a normal first pregnancy. She presented with asymptomatic bacteriuria during her second pregnancy and later developed pyelonephritis, which required hospitalisation. She is currently contemplating a third pregnancy and is anxious that she will have kidney problems again. Which of the following statements is CORRECT?

- A. Anne would not require any special management were she to become pregnant again.
- B. *Escherichia coli* is found in very low levels in pregnant woman with asymptomatic bacteriuria.
- C. Asymptomatic bacteriuria is reported mainly in mothers from a low socioeconomic status.
- D. Women should be offered routine screening for asymptomatic bacteriuria early in pregnancy.
- E. All pregnant women have the same risk for asymptomatic bacteriuria.

**QUESTION 10**

Which of one of the following general statements about CKD is the most CORRECT?

- A. The target blood pressure for people with CKD and microalbuminuria is  $\leq 125/75$  mmHg.
- B. Renal denervation should be considered for those with resistant hypertension and CKD.
- C. People at very high risk of cardiovascular disease do not require calculation of their absolute cardiovascular risk.
- D. All of the above.
- E. None of the above.

**REFERENCES**

1. Antibiotic Expert Group. Urinary tract infections: Acute cystitis in adults. [In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd: 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 9 April 2014].
2. Smaill FM, Vazquez JC. Antibiotics for asymptomatic bacteriuria in pregnancy. Cochrane Pregnancy and Childbirth Group. Published Online: 18 Apr 2007. Available at <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD000490.pub2/full> [Accessed on 9 April 2014].

3. National Heart Foundation (2008 (2010 Update)). Guide to management of hypertension 2008. Available at [www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf) [Accessed 15 April 2014].
4. Thomas MC, Weekes AJ, Broadley O J, Cooper ME, Mathew TH. The burden of chronic kidney disease in Australian patients with type 2 diabetes (the NEFRON study). *Med J Aust* 2006;185:140–44.
5. Grace B, Hurst K, McDonald S. Australia and New Zealand Dialysis and Transplant Registry. New Patients. Adelaide: South Australia; 2012.
6. Chater S, Davison SN, Germain MJ, Cohen LM. Withdrawal from dialysis: A palliative care perspective. *Clin Nephrol* 2006;66:364–72.
7. Cohen LM, Germain M, Poppel DM, Woods A, Kjellstrand CM. Dialysis discontinuation and palliative care. *Am J Kidney Dis* 2000;36:140–44.
8. Cohen LM, Germain M, Poppel DM, Woods A, Pekow P, Kjellstrand CM. Dying well after discontinuing the life-support treatment of dialysis. *Arch Intern Med*. 2000;160:2513–18.
9. Cameron S and Brennan F. Legal issues concerning withholding and withdrawal of dialysis. *Nephrology* 2013;13:444–49.
10. McDonald S, Excell L, Livingstone B, editors. The thirty-first report. Adelaide: Australian and New Zealand Dialysis and Transplant Registry; 2008. Available at [www.anzdata.org.au/anzdata/AnzdataReport/31stReport/FullReport.pdf](http://www.anzdata.org.au/anzdata/AnzdataReport/31stReport/FullReport.pdf) [Accessed 29 April 2014].
11. Palliative Care Expert Group. Renal failure in patients receiving palliative care. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd: 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 9 April 2014].
12. Rossi S, editor. Gout. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
13. Rossi S, editor. NSAIDs. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Medicines Handbook Pty Ltd; 2014.
14. Hull R, Goldsmith D. Nephrotic syndrome in adults. *BMJ* 2008;336:1185–89.
15. National Institute for Health and Clinical Excellence. Antenatal Care. June 2010/ NICE Clinical Guideline 62. Available at [www.nice.org.uk/nicemedia/live/11947/40115/40115.pdf](http://www.nice.org.uk/nicemedia/live/11947/40115/40115.pdf) [Accessed 23 March 2014].
16. Antibiotic Expert Group. Urinary tract infections: Asymptomatic bacteriuria. In: eTG [Internet]. Melbourne: Therapeutic Guidelines Ltd: 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 8 April 2014].
17. Wing DA, Hendershott CM, Debuque L, Millar LK. A randomized trial of three antibiotic regimens for the treatment of pyelonephritis in pregnancy. *Obstet Gynecol* 1998;92:249–53.
18. Lenke RR, VanDorsten JP, Schifrin BS. Pyelonephritis in pregnancy: a prospective randomized trial to prevent recurrent disease evaluating suppressive therapy with nitrofurantoin and close surveillance. *Am J Obstet Gynecol* 1983;146:953–57.
19. Harris RE, Gilstrap LC 3rd. Prevention of recurrent pyelonephritis during pregnancy. *Obstet Gynecol* 1974;44:637–41.
20. South Australian Perinatal Practice Guidelines. Urinary tract infections in pregnancy. Adelaide: Department of Health, Government of South Australia. Available at [www.sahealth.sa.gov.au/wps/wcm/connect/Public+Content/SA+Health+Internet/Clinical+resources/Clinical+topics/Perinatal+practice+guidelines/](http://www.sahealth.sa.gov.au/wps/wcm/connect/Public+Content/SA+Health+Internet/Clinical+resources/Clinical+topics/Perinatal+practice+guidelines/) [Accessed 29 April 2014].
21. National Vascular Disease Prevention Alliance. Guidelines for the management of absolute cardiovascular disease risk. Canberra: National Health and Medical Research Council of Australia; 2012. Available at [http://strokefoundation.com.au/site/media/AbsoluteCVD\\_GL\\_webready.pdf](http://strokefoundation.com.au/site/media/AbsoluteCVD_GL_webready.pdf). [Accessed 16 April 2014].
22. Johnson DW, Atai E, Chan M, et al. KHA-CARI guideline: Early chronic kidney disease: detection, prevention and management. *Nephrology* 2013;18:340–50.
23. Bhatt DL, Kandzari DE, O'Neill WW, et al. A controlled trial of renal denervation for resistant hypertension. *N Engl J Med* 2014;370:1393–1401.

# check

Independent learning program for GPs

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Independent learning program for GPs



Unit 508 August 2014

## Head pain

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## Head pain

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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

## ABOUT THIS ACTIVITY

Headache and head pain are common medical conditions and about 15% of Australians use pain-relieving medication for a headache at any given time.<sup>1</sup> Headaches are classified as being primary or secondary in nature. Primary headaches include migraine and cluster and tension headaches, and are of unknown aetiology, whereas secondary headaches are attributed to underlying problems, such as infection, injury or a tumour. Secondary headaches are very important clinically as some of these, for example thunderclap headache due to a carotid dissection, require urgent medical care.<sup>2-4</sup> In many cases, headaches are benign, but they may decrease overall quality of life.

This edition of *check* considers scenarios of headache and head pain of relevance to general practice.

## LEARNING OUTCOMES

At the end of this activity, participants will be able to:

- describe red flags that are relevant in the management of head pain and headaches
- outline current treatment and management options for cluster headache
- explain the risks of combined oral contraceptives (COCs) use for women with migraine and COC options for those who wish to use them
- summarise the defining traits of thunderclap headaches and their management
- specify secondary prevention strategies for transient ischaemic attacks/stroke.

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## REFERENCES

1. Better Health Channel Fact Sheet: Headache. Available at [www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Headache](http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Headache) [Accessed 26 June 2014].
2. Ducros A, Bousser MG. Thunderclap Headache. *BMJ* 2013;345:1–7.
3. Hainer BL, Matheson EM. Approach to acute headache in adults. *Am Fam Physician* 2013;87:682–87.
4. Schwedt TJ. Thunderclap headaches: A focus on etiology and diagnostic evaluation. *Headache* 2013;53:563–69.

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

ASA	atrial septal aneurysm	MRA	magnetic resonance angiogram	RCVS	reversible cerebral vasoconstriction syndrome
BMI	body mass index	MRI	magnetic resonance imaging	SAH	subarachnoid haemorrhage
COC	combined oral contraceptives	NICE	National Institute for Health and Care Excellence	SUNCT	short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing
CrCl	creatinine clearance	NOAC	non-vitamin K oral anticoagulant	TAC	trigeminal autonomic cephalalgia
CSF	cerebrospinal fluid	NSAIDs	nonsteroidal anti-inflammatory drugs	TIA	transient ischaemic attack
CT	computed tomography	PBS	Pharmaceutical Benefits Scheme	WHO	World Health Organization
ICHD-3	International Classification of Headache Disorders 3rd edition	PFO	patent foramen ovale		
IIH	idiopathic intracranial hypertension				

CASE 1

DEBBIE HAS HAD HEADACHES MOST OF HER LIFE

Debbie is 43 years of age and has four children. During her appointment, she comments that she is sick of having frequent headaches. She can recall having headaches for most of her life but the frequency of her headaches has increased over the past 5 years.

QUESTION 1

How would you differentiate the type of headache Debbie has from other types of headaches?

Blank lines for writing the answer to Question 1.

FURTHER INFORMATION

While taking a more detailed history, Debbie describes throbbing headaches on either side of the head lasting for hours to days. Debbie's headaches are associated with photophobia, phonophobia and nausea on at least 4 days a week and last for up to 6 hours per day. She has never had any visual or other disturbance prior to the headaches. The headaches prevent her from fully participating in various activities and, as she finds that they are triggered by heat and exertion, she avoids exercise. The headaches have not changed in character recently but have increased in frequency over the past 5 years.

QUESTION 2

Does it matter which type of headache Debbie has? What is the classification of Debbie's headaches?

Blank lines for writing the answer to Question 2.

QUESTION 3

What questions would you want to ask about Debbie's lifestyle with regard to her increased headache frequency?

Blank lines for writing the answer to Question 3.

FURTHER INFORMATION

Debbie has become overweight and has avoided exercise for years because of her frequent migraines. Since having her four children (the youngest child is now seven), she has had markedly disrupted sleep and always feels tired, so she drinks two cans of caffeinated soft drink and several cups of tea daily. Her worst migraines are often at the time of her menstrual periods and these don't respond to her usual nonsteroidal anti-inflammatory drugs (NSAIDs). Although she avoids many activities due to frequent disruption by her headaches, she denies any symptoms of depression or anxiety.

QUESTION 4

What management strategies could be used to target these comorbidities?

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QUESTION 5

What pharmacological management would be most appropriate for this patient?

Blank lines for writing the answer to Question 5.

**CONCLUSION**

Debbie agrees to start topiramate as migraine prophylaxis, as her blood pressure was too low to tolerate propranolol previously, and she refuses pizotifen because of the unacceptable risk of further weight gain. Over the next 3 months she tolerates topiramate without significant side effects. She gradually has a marked improvement in her migraines, with predominantly menstrually related migraines remaining, which can be effectively managed with her triptan. She has been able to return to regular exercise and has a part-time job in the school canteen.

**CASE 1 ANSWERS**

**ANSWER 1**

Clinical features define primary headache disorders so a careful history is useful in differentiating between headache types. Salient questions for Debbie would include asking about the characteristics and duration of her headaches, preference for rest or ability to perform activities, and associated features such as sensitivity to light or noise, and nausea or vomiting. *Table 1* summarises information

**Table 1. Diagnosis of tension-type headache, migraine and cluster headache<sup>1</sup>**

Headache feature	Tension-type headache		Migraine (with or without aura)		Cluster headache	
Pain location*	Bilateral		Unilateral or bilateral		Unilateral (around the eye, above the eye and along the side of the head/face)	
Pain quality	Pressing/tightening (non-pulsating)		Pulsating (throbbing or banging in young people aged 12–17 years)		Variable (can be sharp, boring, burning, throbbing or tightening)	
Pain intensity	Mild or moderate		Moderate or severe		Severe or very severe	
Effect on activities	Not aggravated by routine activities of daily living		Aggravated by, or causes avoidance of, routine activities of daily living		Restlessness or agitation	
Other symptoms	None		Unusual sensitivity to light and/or sound or nausea and/or vomiting Aura <sup>†</sup> Symptoms can occur with or without headache and: <ul style="list-style-type: none"> <li>• are fully reversible</li> <li>• develop over at least 5 minutes</li> <li>• last 5–60 minutes.</li> </ul> Typical aura symptoms include visual symptoms such as flickering lights, spots or lines and/or partial loss of vision; sensory symptoms such as numbness and/or pins and needles; and/or speech disturbance		On the same side as the headache: <ul style="list-style-type: none"> <li>• red and/or watery eye</li> <li>• nasal congestion and/or runny nose</li> <li>• swollen eyelid</li> <li>• forehead and facial sweating</li> <li>• constricted pupil and/or drooping eyelid</li> </ul>	
Duration of headache	30 minutes–continuous		4–72 hours in adults 1–72 hours in young people aged 12–17 years		15–180 minutes	
Frequency of headache	<15 days per month	≥15 days per month for more than 3 months	<15 days per month	≥15 days per month for more than 3 months	1 every other day to 8 per day <sup>‡</sup> , with remission <sup>§</sup> >1 month	1 every other day to 8 per day <sup>‡</sup> , with a continuous remission <sup>§</sup> <1 month in a 12-month period
<b>Diagnosis</b>	<b>Episodic tension-type headache</b>	<b>Chronic tension-type headache<sup>¶</sup></b>	<b>Episodic migraine (with or without aura)</b>	<b>Chronic migraine<sup>#</sup> (with or without aura)</b>	<b>Episodic cluster headache</b>	<b>Chronic cluster headache</b>

\*Headache pain can be felt in the head, face or neck.  
<sup>†</sup>See recommendations 1.2.2, 1.2.3 and 1.2.4 in the NICE guidelines<sup>1</sup> for further information on diagnosis of migraine with aura.  
<sup>‡</sup>The frequency of recurrent headaches during a cluster headache bout.  
<sup>§</sup>The pain-free period between cluster headache bouts.  
<sup>¶</sup>Chronic migraine and chronic tension-type headache commonly overlap. If there are any features of migraine, diagnose chronic migraine.  
<sup>#</sup>NICE has developed technology appraisal guidance on botulinum toxin type A for the prevention of headaches in adults with chronic migraine (headaches on at least 15 days per month of which at least 8 days are with migraine).  
 Reproduced with permission from the National Institute for Health and Clinical Excellence (2012) CG150 Headaches: diagnosis and management of headaches in young people and adults.<sup>1</sup>

from the National Institute for Health and Care Excellence (NICE) 2012 guidelines,<sup>1</sup> which can be used to differentiate between migraine, tension type headaches and cluster headaches. These guidelines caution against routinely referring people with migraine for neuroimaging in the absence of red flags solely for the purpose of providing reassurance.<sup>1</sup> The Australian Therapeutic Guidelines red flags for prompting further investigation are listed in *Table 2*.<sup>2</sup>

**Table 2. Warning signs in the diagnosis of headache<sup>2</sup>**

Type of headache	Possible organic causes
Sudden onset, particularly with confusion, drowsiness or vomiting, or with mild stroke-like symptoms or signs (eg mild hemiparesis, ataxia, Horner syndrome, diplopia including sixth nerve palsy)	Subarachnoid or intracranial haemorrhage, carotid or vertebral artery dissection, cerebral venous thrombosis, thunderclap headache, reversible cerebral vasoconstriction syndrome
Recent onset with confusion, drowsiness or fever	Meningitis, encephalitis, intracranial abscess, severe hypertension (hypertensive encephalopathy)
Recent onset in a young obese patient	Idiopathic (benign) intracranial hypertension (look for papilloedema)
Recent onset in a patient over 50 years of age	Brain tumour, giant cell arteritis (temporal arteritis), cervicogenic, medication overuse, subdural collection, herpes zoster, sinusitis
Recent onset with cough, exertion or sexual activity	Subarachnoid haemorrhage, brain tumour
After head injury, particularly with loss of consciousness, or if severe or prolonged	Intracranial haemorrhage

### ANSWER 2

According to the International Classification of Headache Disorders, 3rd edition (ICHD-3), beta version classification, Debbie has chronic migraine.<sup>3</sup>

Chronic migraine occurs in approximately 2% of the adult population in Western countries.<sup>4</sup> It is defined as headache that occurs on 15 or more days per month for more than 3 months and has the features of migraine headache on at least 8 days per month.<sup>3</sup> Chronic migraine is considered a complication of episodic migraine; 2.5% of people progress yearly from episodic to chronic migraine.<sup>5</sup> It is the most disabling of the different types of chronic daily headaches, yet is often underdiagnosed and undertreated.<sup>4</sup> In an Australian cohort, only 20% of patients with more than three headaches per month were receiving prophylaxis, while the remaining 80% had either never received such treatment (66%) or had tried but stopped migraine prophylaxis (33%).<sup>6</sup> Correct diagnosis is important as it determines prognosis and the best ongoing management options.

Debbie has never had aura. Migraine without aura is defined as headaches lasting 4–72 hours (when untreated or unsuccessfully treated) with at least two of the following four characteristics: unilateral location, pulsating quality, moderate or severe pain intensity, and aggravation by or causing avoidance of routine physical activity

(eg walking or climbing stairs), and at least one of the following: nausea and/or vomiting, and photophobia and phonophobia.<sup>1,3</sup>

### ANSWER 3

In patients with episodic migraine, modifiable risk factors for development of chronic migraine include increasing attack frequency, obesity (body mass index >30 kg/m<sup>2</sup>), life stressors, snoring/sleep apnoea/sleep disturbance and caffeine consumption. Age, gender, low socioeconomic status, head injury and allodynia are also associated with chronic migraine but are less modifiable risk factors.<sup>7–9</sup> Anxiety and depression are common comorbidities with chronic migraine and may affect central antinociceptive networks.<sup>7</sup>

### ANSWER 4

Educating the patient about reducing triggers by improving sleep hygiene, having regular meals and caffeine cessation may be useful.<sup>10,11</sup> Relaxation training, biofeedback, stress management and cognitive behavioural therapy are recommended.<sup>2,12</sup> Limited studies suggest a role for weight loss and exercise in the prevention of migraine.<sup>13,14</sup> In this case, by recognising the more difficult-to-treat menstrually related migraines, the patient could be taught to target those specific migraines earlier and more effectively by taking a triptan, without overusing triptans at other times of the month. The lowest dose of a triptan is recommended, followed by the higher dose if the low dose is ineffective.<sup>2</sup>

### ANSWER 5

An effective acute headache management plan is limited in chronic migraine because of the high frequency of headaches. There is a risk of medication overuse headaches if analgesics are taken on more than 15 days per month (only 9 days per month are required if strong analgesics such as codeine or triptans are used frequently).<sup>15</sup> Migraine prophylaxis is therefore important. The goals of migraine prophylaxis are to reduce attack severity, frequency and duration, to improve responsiveness to acute treatments and to reduce disability.<sup>12</sup> The Australian Therapeutic Guidelines recommend choosing the prophylactic medication on the basis of potential adverse side effects. According to these guidelines, amitriptyline, pizotifen and propranolol are recommended as first-line prophylaxis, while sodium valproate, topiramate and verapamil are second-line options.<sup>2</sup> The American Academy of Neurology and the European Federation of Neurological Societies have published evidence-based recommendations regarding migraine prophylaxis, based on the quality of evidence.<sup>12,16</sup> Many of the migraine prophylactic medications, such as candesartan and gabapentin are not indicated for migraine on the Pharmaceutical Benefits Scheme (PBS).<sup>17</sup> Other agents, such as vitamin B2, feverfew and magnesium, could also be offered.<sup>18</sup>

When starting migraine prophylaxis, the patient should be given realistic goals over several months (such as a 50% reduction in headache days), with education about correct dosage and possible side effects. The patient should be reviewed after 2 months to adequately assess effectiveness.<sup>12,19</sup> An objective headache diary is

useful for the doctor and patient's conviction in identifying a gradual response to management over the subsequent months. If headaches are resistant to treatment, referral to a specialist in headache for further migraine prophylaxis options, including onabotulinum toxin A may be appropriate in selected cases.<sup>20</sup> Onabotulinum toxin A (or botulinum toxin) is marketed in Australia for migraine prevention in headache occurring on at least 15 days/month.<sup>21</sup> The overall aim should be to reduce the migraine days from chronic migraine to episodic migraine by a combination of lifestyle and prophylactic management.

## REFERENCES

- NICE clinical guideline. Diagnosis and management of headaches in young people and adults. NICE clinical guideline CG150, 2012. Available at [www.nice.org.uk/guidance/cg150](http://www.nice.org.uk/guidance/cg150) [Accessed 11 July 2014].
- Neurology Expert Group. Headache: Migraine: [Revised June 2011]. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd: 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
- Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edn (beta version). Cephalalgia 2013; 33(9):629–808. Available at [www.ihs-classification.org/\\_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf](http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf) [Accessed 26 June 2014].
- Lipton RB. Chronic migraine, classification, differential diagnosis, and epidemiology. Headache 2011;51:S2:77–83.
- Evans RW. Expert opinion: A rational approach to the management of chronic migraine. Headache 2013;53:168–76.
- Stark RJ, Valenti L, Miller GC. Management of migraine in Australian general practice. MJA 2007;187:142–46.
- Halker RB, Hastriter EV, Dodick DW. Chronic daily headache: An evidence-based and systematic approach to a challenging problem. Neurology Clin Pract 2011;76 (suppl 2):S37–S43.
- Bigal ME, Lipton RB. Concepts and mechanisms of migraine chronification. Headache 2008;48:7–15.
- Manack A, Buse DC, Serrano D, Turkel CC, Lipton RB. Rates, predictors and consequences of remission from chronic migraine to episodic migraine. Neurology 2011;76:711–18.
- Dodick DW. Clinical practice: chronic daily headache. N Engl J Med 2006;354:158–65.
- Buse DC, Rupnow MFT, Lipton RB. Assessing and managing all aspects of migraine: migraine attacks, migraine-related functional impairment, common comorbidities, and quality of life. Mayo Clin Proc 2009;84:422–35.
- Silberstein SD for the US Headache Consortium. Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2000;55:754–63.
- Verrotti A, Agostinelli S, D'Egidio C, et al. Impact of a weight-loss program on migraine in obese adolescents. Eur J Neurol 2013;20:394–97.
- Varkey E, Cider A, Carlsson J, Linde M. A study to evaluate the feasibility of an aerobic exercise program in patients with migraine. Headache 2009;49:563–70.
- Bigal ME, Lipton RB. Excessive acute migraine medication use and migraine progression. Neurology 2008;71:1821–28.
- Evers S, Afra J, Frese A, et al. European Federation of Neurological Societies guideline on the drug treatment of migraine – revised report of an EFNS task force. Eur J Neurol 2009;16:968–81.
- Stark RJ, Stark CD. Migraine prophylaxis. MJA 2008;189:283–88.
- Holland S, Silberstein SD, Freitag F, Dodick DW, Argoff C, Ashman E. Evidence-based guideline update: NSAIDs and other complementary treatments for episodic migraine prevention in adults: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Headache Society. Neurology 2012;78:1346–53.
- Shapiro RE. Preventive treatment of migraine. Headache 2012;52:65–69.
- Lipton RB, Varon SF, Grosberg B, et al. Onabotulinumtoxin A improves quality of life and reduces impact of chronic migraine. Neurology 2011;77:1465–72.
- Rossi S, Editor. Migraine. Australian Medicines Handbook. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd. 2014.

## RESOURCES FOR PATIENTS

- Information and a register for migraine patients is available at <http://headacheaustralia.org.au/migraine>
- An easy-to-use and informative headache diary for patients is available at [www.aspenpharma.com.au/patRes/2014\\_Headache\\_Diary.pdf](http://www.aspenpharma.com.au/patRes/2014_Headache_Diary.pdf)

## RESOURCES FOR DOCTORS

- American Headache Consortium Guidelines, <http://guidelinecentral.com/i/76857/5>
- The International Classification of Headache Disorders, 3rd edn (beta version), [www.ihs-classification.org/\\_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf](http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf)

**CASE 2**

**LISA HAS THROBBING HEADACHES**

Lisa, 21 years of age, smokes and binge drinks on weekends. She has a 2-month history of twice weekly episodes of throbbing headaches lasting less than 12 hours and requiring rest in a dark room. Over-the-counter pain relievers, such as non-steroidal anti-inflammatory drugs (NSAIDs) and paracetamol, have been only marginally effective. You take a history and enquire about headache red flags, and perform a general physical and neurological examination. Vital signs and fundoscopy are normal. You diagnose migraines and provide Lisa with some sample packs of an oral triptan medication. A follow-up appointment in a fortnight is scheduled.

**QUESTION 1** 

What type of historical findings in a headache history would be considered red flags for serious problems?

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**FURTHER INFORMATION**

Lisa arrives for her follow-up appointment accompanied by her mother. Lisa cannot cope with her university studies. Over the past fortnight she used the triptan samples and had to resort to codeine-containing analgesics for six episodes of migraines. Lisa had two episodes of transient visual symptoms in her right eye, manifesting with blurred and shimmering central vision, which lasted for 15 minutes; peripheral vision was unaffected and there was no headache in the aftermath.

After collecting additional history with her mother's help, you establish that Lisa has had infrequent migraines since the age of 14 years, but had never experienced visual disturbances or neurological symptoms. There is a family history of migraines and her brother had a structural heart defect. You learn that 3 months ago Lisa started using a combined oral contraceptive (COC) pill. You instruct Lisa to stop taking the COC pill and start low-dose aspirin. Recently you learned that GPs can request a rebatable magnetic resonance imaging (MRI) scan of the

brain for unexplained headaches with suspected intracranial pathology and you arrange a semi-urgent MRI brain scan to be carried out within 1 week.

**QUESTION 2** 

What are common triggers that can elicit a migraine attack or increase the frequency and severity of migraine attacks in a person who has a history of migraines?

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**QUESTION 3** 

What is your diagnosis for Lisa's headaches? What is your diagnosis for Lisa's new onset visual symptoms? How are you going to evaluate Lisa's new visual symptoms?

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**QUESTION 4** 

What structural cardiac condition is common in patients with migraine that may confer an increased risk of ischaemic stroke?

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**CONCLUSION**

A week later you receive an urgent call from a radiologist. Lisa's MRI revealed evidence of acute embolic stroke mainly in the right anterior cerebral artery territory, as well as evidence of a small ischaemic infarct in the right middle cerebral artery

territory. After discussing the results with Lisa and her mother, you establish that 3 days before the MRI scan, during a family reunion, Lisa had a witnessed episode of sudden-onset left leg weakness and speech arrest, which resolved within 10–15 minutes. She had some mild headaches in the aftermath, but no residual neurological deficit. Lisa is admitted to a tertiary care hospital where she is found to have PFO with ASA. An interventional cardiologist carried out percutaneous closure of PFO using a small umbrella-like device.

**CASE 2 ANSWERS**

**ANSWER 1**

As most patients with headaches have normal general physical and neurological examinations, a thorough history is crucial to screen for headache red flags, which suggest underlying systemic or intracranial pathology. Table 1 lists red flags that might be uncovered during history taking for headache presentations and possible differential diagnoses for red flag findings.<sup>1</sup>

**Table 1. Headache red flags and possible differential diagnoses<sup>1</sup>**

Red flags	Differential diagnoses
<ul style="list-style-type: none"> <li>Sudden onset of headaches</li> <li>Thunderclap headaches</li> <li>Precipitation of headache with Valsalva manoeuvres</li> <li>Persistent strictly unilateral headaches</li> </ul>	Subarachnoid haemorrhage Pituitary apoplexy Haemorrhage into a mass lesion or vascular malformation Mass lesion (especially posterior fossa mass) Upper cervical spine pathology
<ul style="list-style-type: none"> <li>Headache beginning after 50 years of age</li> </ul>	Temporal arteritis Mass lesion
<ul style="list-style-type: none"> <li>A change in frequency and severity of headaches and emergence of new neurological symptoms</li> </ul>	Vascular pathology Mass lesion Subdural haematoma Medication overuse
<ul style="list-style-type: none"> <li>Headache in young overweight people</li> </ul>	Idiopathic intracranial hypertension Cerebral sinus thrombosis
<ul style="list-style-type: none"> <li>Headaches subsequent to trauma</li> </ul>	Intracranial haemorrhage Subdural haematoma Epidural haematoma Post-traumatic headache
New onset headache in: <ul style="list-style-type: none"> <li>a patient with systemic illness, fever, cancer or human immunodeficiency virus</li> <li>pregnancy or the postpartum period</li> <li>patients on anticoagulants and immunosuppressant medications</li> </ul>	Meningitis Encephalitis Metastasis Vasculitis Any of the differential diagnoses in the above categories

**ANSWER 2**

Common triggers that may elicit a migraine attack or increase the frequency and severity of migraine attacks include:<sup>2</sup>

- stress or relaxation after periods of stress (stress can include bright lights, loud noise, long-distance travel and extremes of weather)
- anxiety and depression
- dehydration
- sleep deprivation or excessive sleep
- missed meals
- trauma to the head or neck
- oral contraceptives and vasodilators such as glyceryl trinitrate.

Dietary factors include cheese, chocolate, alcohol and citrus fruits (these are only occasionally important in management and too much effort in identifying them may be counterproductive).

**ANSWER 3**

According to the International Classification of Headache Disorders, 3rd edition (ICHD-3), beta version diagnostic criteria, Lisa's headaches are consistent with the diagnosis of migraine without aura.<sup>3</sup> According to these criteria, her visual symptoms are consistent with the diagnosis of migraine manifesting with typical aura without headache.<sup>3</sup> An older term that describes these headaches is 'acephalgic migraine'.

If a patient has a long history of stereotypical migraine auras with and without headaches, but has no deficits found on the physical or neurological examination, a complete work-up with laboratory and imaging tests is probably not mandatory. However, a complete evaluation should be undertaken if a patient presents with new-onset migraine aura without headaches.<sup>4</sup> An MRI scan of the brain should be carried out within 10–14 days of having such an episode of migraine to rule out an ischaemic stroke. A cerebral MR angiography and carotid Doppler are used to assess for intra- and extracranial stenosis, due either to arteriosclerosis or a vasculitis. Other ancillary investigations may include serological testing for thrombophilia and systemic vasculitis. Practitioners are reminded that Medicare rebates apply for thrombophilia screening or testing for specific factor deficiencies only if the patient has a personal proven history of venous thromboembolism or has a first-degree relative with a proven factor defect.<sup>4</sup> Epidemiological studies have suggested that the risk of ischaemic stroke is slightly increased in women with migraines, with the risk reported to be substantially higher in certain sub groups of women.<sup>5,6</sup>

**ANSWER 4**

Over the last decade, patent foramen ovale (PFO) associated with atrial septal aneurysm (ASA) has been identified as an independent risk factor for ischaemic stroke, particularly in young adults with cryptogenic stroke.<sup>7</sup> Recently, migraine was found to be significantly associated with PFO when compared with controls. At least 50% of patients with migraine with aura were found to have a PFO, and the size of the intracardiac shunts was larger in patients with migraines

than in controls.<sup>8</sup> In susceptible women, the association between migraines with aura and PFO leads to an increased risk of ischaemic stroke.<sup>7</sup> In terms of pathophysiology, it has been proposed that in this population of women, COCs may trigger venous embolism with subsequent paradoxical embolism through PFO or formation of a mural thrombus locally in the atrial septum within the conduit of PFO.<sup>9</sup>

Importantly, an isolated finding of PFO without any other high-risk features in an asymptomatic individual is of equivocal clinical significance. Routine screening for PFO is not recommended.<sup>7–9</sup>

## REFERENCES

1. Newman LC, Lipton RB. Emergency department evaluation of headache. *Neurol Clin* 1998;16:285–303.
2. Wober C, Wober-Bingol C. Triggers of migraine and tension-type headache. *Handb Clin Neurol* 2010;97:161–72.
3. The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia* 2013;33:629–808. Available at [www.ihs-classification.org/\\_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf](http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf) [Accessed 26 June 2014].
4. Kunkel RS. Migraine aura without headache: benign, but a diagnosis of exclusion. *Cleve Clin J Med* 2005;72:529–34.
5. Schürks M, Rist PM, Bigal ME, Buring JE, Lipton RB, Kurth T. Migraine and cardiovascular disease: systematic review and meta-analysis. *BMJ* 2009;339:b3914.
6. Schürks M, Buring JE, Kurth T. Migraine, migraine features, and cardiovascular disease. *Headache* 2010;50:1031–40.
7. Overell JR, Bone I, Lees KR. Interatrial septal abnormalities and stroke: a meta-analysis of case-control studies. *Neurology* 2000 55;1172–79.
8. Anzola GP, Meneghetti G, Zanferrari C et al; SAM Study Group. Is migraine associated with right-to-left shunt a separate disease? Results of the SAM study. *Cephalalgia* 2008;28:360–66.
9. Amarenco P. Patent foramen ovale and the risk of stroke: smoking gun guilty by association? *Heart* 2005;91:441–43.

**CASE 3**

**STEVEN HAS AN ONGOING HEADACHE**

Steven, 57 years of age, comes to see you today with his wife to discuss a 3-week history of a constant unilateral retro-orbital headache. His wife states that he has looked more tired than usual since the onset of the headache.

**QUESTION 1** 

What are the red flags in this history? What would point to a secondary cause of headache during history taking?

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**FURTHER INFORMATION**

Steven has been healthy without any significant past medical history and he does not take any regular medication. He denies significant headaches in the past. One night 3 weeks ago, Steven was woken up by a sudden-onset, severe headache. He took paracetamol and the headache improved slightly. The pain was over his left eye and it was constant. He denies having had any head or neck trauma before the symptoms started. The pain over the left eye continued for several weeks and has gradually improved.

**QUESTION 2** 

What are your differential diagnoses for thunderclap headache?

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**FURTHER INFORMATION**

On examination, Steven has left partial ptosis and miosis (*Figure 1*). The difference in pupil size is enhanced in the dark and pupillary responses are normal. The remainder of the visual and neurological examinations are normal. There is no lack of sweating on the left side versus the right. Left carotid artery is palpable without any bruits.



Figure 1. Eye examination

**QUESTION 3** 

What is his diagnosis? How would you investigate further?

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**FURTHER INFORMATION**

You order a CT scan of Steven's orbit, cavernous sinus and pituitary fossa as part of the initial workup investigation and it is unremarkable. A magnetic resonance imaging scan (MRI) with a magnetic resonance angiogram (MRA) of the aortic arch to the Circle of Willis, which is now covered by Medicare when ordered by a GP, is performed and reveals a left internal carotid artery dissection (*Figure 2*).

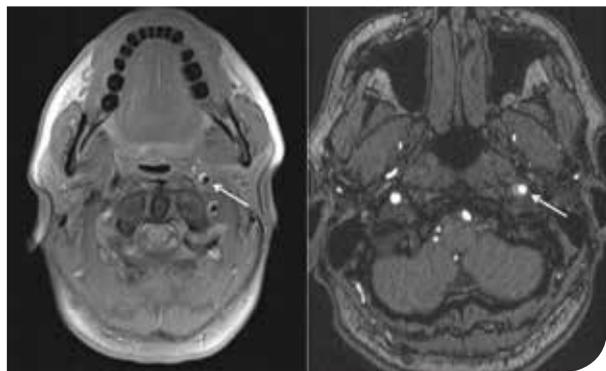


Figure 2. Axial T1 MRI and time-of-flight MRA showing a crescent sign in the left internal carotid artery

**QUESTION 4** 

How would you manage Steven?

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**FURTHER INFORMATION**

Steven was started on aspirin and comes back for follow up 3 months later. He asks 'will it happen again?'.

**QUESTION 5**  

How would you respond to Steven's question?

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**CASE 3 ANSWERS**

**ANSWER 1**

Given the very short history in this presentation, it is difficult to determine the underlying cause of the headache and whether it is likely to have a serious pathophysiology. Primary headache presentations (ie migraine, tension, cluster headache) most often have a history of similar attacks in the past.<sup>1</sup> Steven's age (57 years) is another factor that may indicate that this headache may not be benign in aetiology.<sup>2</sup> Patients over the age of 50 years who present with a new headache should always be investigated and their concerns taken seriously.<sup>3</sup> A good rule of thumb is that the 'first or worst' headache should prompt investigation. However, severity is not always a reliable guide as primary headaches are some of the most severe on the pain spectrum.<sup>1</sup>

Other important points on history that may indicate a secondary cause include information regarding headache onset (ie gradual versus thunderclap presentations; *Table 1*).<sup>4-6</sup> The latter tend to indicate a vascular presentation.<sup>7</sup>

**Table 1. Warning signs for secondary headache<sup>4-6</sup>**

<b>Thunderclap presentation</b>
<ul style="list-style-type: none"> <li>Usually indicative of vascular presentations</li> </ul>
<b>Age &gt;50 years</b>
<ul style="list-style-type: none"> <li>Unusual for primary headaches to present for the first time in this age group</li> </ul>
<b>New headache</b>
<ul style="list-style-type: none"> <li>Sinister aetiologies usually become apparent within 6 months</li> </ul>
<b>Systemic symptoms</b>
<ul style="list-style-type: none"> <li>Infectious or inflammatory disorders may have fever or other organ involvement</li> </ul>
<b>Focal signs</b>
<ul style="list-style-type: none"> <li>Persistent signs such as cranial nerve dysfunction, diplopia and papilloedema should always suggest secondary cause and warrants careful investigation</li> </ul>
<b>Confusion/agitation</b>
<ul style="list-style-type: none"> <li>Can be the end result of several secondary causes</li> </ul>
<b>Cough-only headache</b>
<ul style="list-style-type: none"> <li>Indicative of foramen magnum lesions</li> </ul>
<b>Postural headache</b>
<ul style="list-style-type: none"> <li>May suggest low or high pressure headache</li> </ul>
<b>Medical comorbidities</b>
<ul style="list-style-type: none"> <li>Cancer history</li> <li>Immunosuppression/HIV</li> <li>Pregnancy</li> <li>Anticoagulants</li> </ul>

Focal neurological signs are also paramount in a headache presentation.<sup>8–10</sup> Patients with cranial nerve symptoms or signs require a thorough investigation of the base of the skull, cavernous sinus or subarachnoid space. Aphasia, hemiparesis, hemisensory loss or visual loss are also important to note as they may indicate a space-occupying lesion.<sup>11</sup>

Systemic symptoms such as fever, myalgia, anorexia and weight loss should be sought and may suggest underlying infectious, inflammatory or malignant processes.<sup>3,12</sup> Confusion or agitation may occur with a number of different processes and is a potentially serious finding.<sup>13</sup>

Several medical conditions should lower the threshold for further investigation. A prior significant cancer history should prompt concern for intracerebral metastasis. Immunosuppression should raise concern about a meningitic process or opportunistic infection.<sup>3</sup> Anticoagulant use and a history of mild trauma should prompt investigation for a subdural haemorrhage.<sup>14</sup> Pregnancy and the postpartum period is a risk factor for cerebral venous sinus thrombosis.<sup>15</sup> Previous neurosurgery or ventriculoperitoneal shunting is a risk factor for intracranial infection or cerebrospinal fluid (CSF) pressure-related headache.<sup>16</sup> Obesity or use of retinoids or tetracycline should raise concern about idiopathic intracranial hypertension (IIH).<sup>17–19</sup>

Other characteristics of Steven's headache may provide further clues. A cough-only headache can suggest a foramen magnum lesion, particularly a Chiari malformation.<sup>20</sup> A headache that changes significantly with posture (ie lying versus standing) tends to suggest a low or high CSF pressure headache.

A few other rules of thumb apply with headache presentations:

- Headache severity is not always a reliable sign as migraine presentations can be associated with severe pain and frank neurological signs.<sup>21</sup>
- Space-occupying lesions and brain tumours rarely present with headache in isolation and almost always have focal signs or symptoms.<sup>22</sup>
- Fundoscopy should be performed in all patients as IIH is easily missed if papilloedema is not looked for specifically.

**ANSWER 2**

Thunderclap headache is used to describe a headache that reaches its peak intensity within 60 seconds.<sup>4,6</sup> The most feared condition with this presentation is subarachnoid haemorrhage.<sup>6</sup> Urgent assessment is usually indicated with an emergency department admission and computed tomography (CT) imaging.<sup>6</sup> Most of the other important differential diagnoses are vascular in origin and are listed in *Table 2*.<sup>4,6</sup> If there has been a delay of several weeks with the presentation and the patient is otherwise well, a less urgent approach can be undertaken. This is because the danger period is fairly early with most of the differential diagnoses.<sup>23,24</sup> Primary thunderclap headache is a diagnosis of exclusion,<sup>6</sup> and vascular causes require exclusion first.

**Table 2. Differential diagnoses for thunderclap headache presentations<sup>4,6</sup>**

- Subarachnoid haemorrhage
- Acute subdural haemorrhage
- Dissection of cervical arteries
- Reversible cerebral vasoconstriction syndrome
- Cerebral venous thrombosis
- Pituitary apoplexy
- Intracranial hypotension
- Hypertensive crisis/phaeochromocytoma
- Primary thunderclap headache/migraine

**ANSWER 3**

Steven has Horner's syndrome, affecting the left eye. Horner's syndrome is classified into first-, second- and third-order neuron lesions (*Table 3*). The different degrees of neuron lesions may be associated with other clinical signs.<sup>25</sup> The sympathetic nerve supply to the eye takes a long course from the hypothalamus, down the brainstem and cervical cord, and exits at the lower cervical level near the brachial plexus. It then ascends in the neck and lies in close proximity to the internal carotid artery all the way into the orbit. Given the long course of this nerve supply, various differential diagnoses are possible, but can be narrowed by looking for associated features. *Table 3* summarises the location, aetiology and suggested investigations for Horner's syndrome.<sup>25</sup> More sophisticated pharmacological tests can also be used to determine the site of Horner's syndrome.<sup>25</sup> This is usually only performed in difficult cases and by those with experience. If the diagnosis and underlying aetiology are unclear, referral to a neurologist may be indicated.

**Table 3. Horner's syndrome<sup>25</sup>**

Neurone	Location	Aetiology	Investigations
First-order	Brainstem C-spine C8–T2 spine	Brainstem stroke Arnold-Chiari Trauma, syringomyelia	Brain MRI and cervicomedullary junction
Second-order	C8–T2 nerve root Lung apex Sympathetic chain	Cervical rib Lower brachial plexus injury Tumours Subclavian artery aneurysm	MRI of the cervical spine, chest CT +/- MRI brachial plexus
Third-order	Carotid artery Cavernous sinus orbit	Carotid dissection, aneurysm, arteritis Base of skull tumours or mass <i>Herpes zoster</i>	Vascular imaging of the carotid artery via CTA/MRA MRI of base of skull

The sudden onset of symptoms in Steven's case suggest a vascular cause is highly likely and further vascular imaging of the carotid arteries should be obtained.

**ANSWER 4**

Involvement of a stroke neurologist/physician may be indicated. The priority in carotid artery dissection is stroke prevention. The true incidence of stroke following dissection is unknown.<sup>26</sup> It is likely that many patients have minimal symptoms and do not present at all. Carotid artery dissection is a common cause of stroke in the younger population (<45 years of age) and may be responsible for up to 20% of strokes in this subgroup.<sup>27</sup>

Antithrombotic therapy has a theoretical advantage in that it may prevent thrombus formation at the site of occlusion and also distal embolisation.<sup>26</sup> There are no randomised controlled trials that compare the effectiveness of antiplatelet therapy or anticoagulation with placebo or with each other.<sup>28</sup> A 2010 Cochrane review found no evidence to support either strategy.<sup>28</sup> Several observational studies have been conducted and a meta-analysis published in 2012 showed that there is no difference in outcomes with either treatment in terms of mortality or risk of ischaemic stroke.<sup>29</sup>

Notwithstanding the above, a minimum of 3–6 months of antithrombotic treatment is usually necessary as this is the time frame in which recanalisation and vessel healing occur.<sup>30</sup> Most experts tend to use anticoagulants for dissections associated with ischaemic symptoms or stroke, in line with 2011 American Heart Association/American Stroke Association (AHA/ASA) guidelines, which recommend use of antithrombotic therapy for 3–6 months for patients with ischaemic stroke or transient ischaemic attack, and arterial dissection, even in light of the uncertainties about the relative efficacy of antiplatelet agents versus anticoagulation therapy.<sup>31</sup> Those without ischaemic symptoms are managed with antiplatelet therapy.<sup>26</sup>

**ANSWER 5**

The risk of recurrent dissection is 2% in the first 3 months and the risk of recurrent stroke or ischaemia is 2%.<sup>32,33</sup> The risk of stroke is highest within the first few weeks following a dissection.<sup>33–35</sup> Steven can be reassured, therefore, that his risk of another episode is very low.

**REFERENCES**

- Lipton RB, Bigal ME, Steiner TJ, Silbertson SD & Olesen J. Classification of primary headaches. *Neurol* 2004;63:427–35.
- Edlow JA, Panagos PD, Godwin SA, Thomas TL & Decker WW. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute headache. *Annals of Emerg Med* 2008;52:407–36.
- Rothman RE, Keyl PM, McArthur JC et al. A decision guideline for emergency department utilization of noncontrast head computed tomography in HIV-infected patients. *Acad Emerg Medic* 1999;6:1010–19.
- Ducros A. and Bousser MG. Thunderclap headache. *BMJ* 2013;345:1–7.
- Hainer BL, Matheson EM. Approach to acute headache in adults. *Am Fam Physician* 2013;87:682–87.
- Schwedt TJ. Thunderclap headaches: a focus on etiology and diagnostic evaluation. *Headache* 2013;53:563–69.
- Pascual J, Gonzalez-Mandly A, Martin A, Otero A. Headaches precipitated by cough, prolonged exercise or sexual activity: a prospective etiological and clinical study. *J Headache Pain* 2008;9:259–66.
- ACEP Clinical Policies Subcommittees on Acute Headache. Clinical policy: critical issues in the evaluation and management of patients presenting to the emergency department with acute headache. *Ann Emerg Med* 2002;39:108–22.
- Detsky ME, McDonald DR, Baerlocher MO, Tomlinson GA, McCrory DC, Booth CM. Does this patient with headache have a migraine or need neuroimaging? *JAMA* 2006;296:1274–83.
- Locker TE, Thompson C, Rylance JM, Mason SM. The utility of clinical features in patients presenting with nontraumatic headache: an investigation of adult patients attending an emergency department. *Headache*, 2006;46:954–61.
- Kocaeli H, Hakyemez B, Bekar A, et al. Unusual complications and presentation of intracranial abscess: experience of a single institution. *Surg Neurol* 2008;69:383–91.
- Clinch CR. Evaluation of acute headaches in adults. *Am Fam Physician* 2001;63:685–92.
- Chugh C, Wadhvani N, Biller J. Confusion and slurred speech in a 34-year-old woman from India. *Front Neurol* 2011;2:34.
- Levine MN, Raskob G, Beyth RJ, Kearon C, Schulman S. Hemorrhagic complications of anticoagulant treatment: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004;126:287S–310S.
- Cantu C, Barinagarrementeria F. Cerebral venous thrombosis associated with pregnancy and puerperium: Review of 67 cases. *Stroke* 1993;24:1880–84.
- Reddy GK, Bollam P, Caldito G, et al. Ventriculoperitoneal shunt complications in hydrocephalus patients with intracranial tumors: an analysis of relevant risk factors. *J Neurooncol* 2011;103:333–42.
- Wall M, George D. Idiopathic intracranial hypertension: a prospective study of 50 patients. *Brain* 1991;114:155–80.
- Kesler A, Goldhammer Y, Hadayer A, Pianka, P. The outcome of pseudotumor cerebri induced by tetracycline therapy. *Acta Neurol Scand* 2004;110:408–11.
- Tabssi A, Salmasi AH, Jalali M. Serum and CSF vitamin A concentrations in idiopathic intracranial hypertension. *Neurology* 2005;64:1893–96.
- Pascual J, Inglesias F, Oterino A, Vazquez-Barquero A, Berciano J. Cough, exertional, and sexual headaches: an analysis of 72 benign and symptomatic cases. *Neurology* 1996;46:1520–24.
- Goadsby PJ, Lipton RB, Ferrari MD. Migraine – Current understanding and treatment. *New Eng J Med* 2002;346:257–70.
- Bradley D, Rees J. Brain tumour mimics and chameleons. *Pract Neurology* 2013;13:359–71.
- Hop JW, Rinkel GJE, Algra A, van Gijn J. Case-fatality rates and functional outcome after subarachnoid hemorrhage: a systematic review. *Stroke* 1997;28:660–64.
- Todd J, Schwedt. Thunderclap headaches: a Focus on etiology and diagnostic evaluation. *Headache* 2013;53:563–69.
- Davagnanam I, Fraser CL, Miszkiele K, Daniel CS, Plant GT. Adult Horner's syndrome: a combined clinical, pharmacological, and imaging algorithm. *Eye (Basingstoke)* 2013;27:291–98.
- Baumgartner RW. Management of spontaneous dissection of the cervical carotid artery. *Acta Neurochir Suppl* 2010;107:57–61.
- Redekop GJ. Extracranial carotid and vertebral artery dissection: a review. *Can J Neurol Sci* 2008;35:146–52.
- Lyrer P, Engelter S. Antithrombotic drugs for carotid artery dissection. *Cochrane Database Syst Rev* 2010;10:CD000255. doi: 10.1002/14651858.CD000255.pub2.
- Kennedy F, Lanfranco S, Hicks C, et al. Antiplatelets vs anticoagulation for dissection: CADISS nonrandomized arm and meta-analysis. *Neurology* 2012;79:686–89.
- Nedeltchev K, Bickel S, Arnold M, et al. R2-recanalization of spontaneous carotid artery dissection. *Stroke* 2009; 40:499–504.

31. Furie KL, Kasner SE, Adams RJ, et al. Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the American heart association/American stroke association. *Stroke* 2011;42:227–76.
32. Debette S, Grond-Ginsbach C, Bodenat M, et al. Differential features of carotid and vertebral artery dissections The CADISP study. *Neurol* 2011;77:1174–81.
33. Touze E, Gauvrit JY, Moulin T, Meder JF, Bracard S, Mas JL. Risk of stroke and recurrent dissection after a cervical artery dissection: A multicenter study. *Neurology* 2003;61:1347–51.
34. Kremer C, Mosso M, Georgiadis D, et al. Carotid dissection with permanent and transient occlusion or severe stenosis: Long-term outcome. *Neurology* 2003;60:271–75.
35. Leys D, Moulin T, Stojkovic T, et al. Follow-up of patients with history of cervical artery dissection. *Cerebrovasc Dis* 1995;5:43–49.

**CASE 4**

**EMMA HAS HEADACHES WITH ARM TINGLING AND NUMBNESS**

Emma, aged 20 years, is a university student who presents with a history of headaches for the past 3 years. The first episode occurred during Year 12, shortly after exams. At the time she experienced tingling in her left hand that gradually progressed up to the shoulder and then spread to her lips and tongue. Symptoms lasted for about 20 minutes and were followed by numbness in the affected area for an additional 10–15 minutes before normal sensation returned. However, 30 minutes later, a moderately severe headache started in the left retro-orbital and temporal regions. It progressed to a severe level over 30 minutes, at which point she also experienced nausea and vomiting and was compelled to lie down in a dark, quiet room. The headache waxed and waned over 2 days, after which she felt tired and ‘washed out’. Over the past 3 years, similar episodes have occurred sporadically, every 1–3 months and usually in the setting of stress. She finds that paracetamol and ibuprofen sometimes decrease the pain but generally she needs to ‘wait it out’. Recently, she was unable to do an exam due to a headache, which prompted a visit to another GP at your practice.

**QUESTION 1** 

What is Emma’s headache diagnosis?

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**QUESTION 2** 

What are the stages of migraine?

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**QUESTION 3** 

What are the typical features of migraine aura?

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**QUESTION 4** 

Are any investigations required?

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**QUESTION 5** 

How should Emma treat her migraine attacks?

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**QUESTION 6** 

Is preventive treatment indicated? What considerations should be made when choosing a treatment?

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**QUESTION 7** 

What lifestyle modifications would you recommend?

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**FURTHER INFORMATION**

Six months later Emma returns for a follow-up consultation. She has had three attacks of migraine with aura, which responded within 30 minutes to a combination of sumatriptan nasal spray and naproxen. She is here today to speak to you about starting the birth control pill.

**QUESTION 8** 

What do you advise her about oral contraceptives?

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**CASE 4 ANSWERS**

**ANSWER 1**

Emma’s headaches are most consistent with migraine with aura.

Migraine is a recurrent headache disorder characterised by attacks lasting 4–72 hours. Typical features of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity, and association with nausea and/or photophobia and phonophobia.<sup>1</sup> Paraesthesia and numbness are characteristic of migraine aura and are discussed in detail in the following questions and answers.

The 3-item ID Migraine screener<sup>2</sup> is a validated and reliable screening instrument for migraine headaches and can improve migraine recognition in primary care settings. It consists of three questions:

- Are you nauseated or sick to your stomach when you have a headache?
- Does light bother you when you have a headache?
- Has a headache limited your activities for a day or more in the last 3 months?

A positive answer to two or more questions translates into a 93% positive predictive value for migraine.

The differential diagnoses for Emma’s presentation include:

- Hemiplegic migraine, which is a form of migraine with aura in which the aura includes motor weakness.<sup>1</sup>
- Chronic migraine, defined as a headache occurring on 15 or more days per month for more than 3 months, which has the features of migraine headache on at least 8 days per month.<sup>1</sup>
- Tension-type headache, which is usually bilateral and mild or moderate in severity and lasts from minutes to days. It is not associated with nausea but photophobia or phonophobia may be present.<sup>1</sup>

- Cluster headaches, which are characterised by attacks of severe, unilateral pain in the orbital, supraorbital and/or temporal region, and has ipsilateral autonomic features (ie conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, facial sweating, miosis, ptosis and/or eyelid oedema). Attacks last for 15–180 minutes and occur from once every other day to eight times per day during ‘cluster periods’, which may last for weeks or months.<sup>1</sup>

## ANSWER 2

Migraine comprises several stages:

- prodrome
- aura
- headache
- postdrome.

Patients may experience some or all of the stages. Most migraineurs have a prodrome phase but it is often unrecognised, as symptoms may occur hours to days before the headache onset. Prodromal or premonitory symptoms are often vague or non-specific but tend to be consistent for the individual. Typical prodromal symptoms include euphoria, depression, difficulty concentrating, hyperactivity, repetitive yawning, excessive thirst, anorexia or cravings for sweet or salty foods.<sup>3</sup>

Postdromal symptoms are more readily recognisable and may persist for 24 hours after the headaches. Typical postdromal symptoms include irritability, fatigue, euphoria, muscle weakness or myalgias, anorexia or food cravings.<sup>3</sup>

## ANSWER 3

Typical aura may be visual, sensory and/or dysphasic in nature. Visual aura is the most common type of aura and can present as positive features (flickering lights, spots or lines) and/or negative features (loss of vision). It often presents as a ‘fortification spectrum’, a zigzag figure that may gradually spread right or left, developing into a convex shape with a scintillating edge and leaving absolute or variable degrees of scotoma after it fades.<sup>1</sup> Sensory auras are less common but often follow a typical pattern of positive features (‘pins and needles’) in the point or origin with gradual involvement of all or part of one side of the body, face and/or tongue. Numbness may occur after paraesthesia fades, but numbness may be the only symptom. Dysphasic speech disturbances are least common. Aura symptoms may occur in succession. Although the sequence usually progresses from visual to sensory to aphasic symptoms, other sequences may occur.<sup>1</sup>

Symptoms of migraine aura are characterised by several features:<sup>1</sup>

- complete reversibility
- gradual development, either alone or in succession, over at least 5 minutes
- duration of each symptom is 5–60 minutes
- at least one aura symptoms is unilateral
- the aura is accompanied, or followed within 60 minutes, by headache.

Although weakness occurs during the aura in patients with familial or sporadic hemiplegic migraine, motor weakness is not a typical aura symptom and warrants further evaluation. Of note, patients with aura characterised by numbness may describe ‘heaviness’ in the affected limb, which can be mistaken for weakness.

## ANSWER 4

Emma’s aura symptoms are stereotypical of migraine with aura and therefore imaging and investigations are not required. Patients should not be referred for neuroimaging solely for the purposes of providing reassurance.<sup>4</sup> Investigations are indicated for patients who present with or without migraine headache and any of the following atypical aura symptoms:<sup>4</sup>

- motor weakness
- double vision
- visual symptoms affecting only one eye
- poor balance
- decreased level of consciousness.

Other headache red flags that warrant further evaluation include:<sup>5</sup>

- first or worst severe headache
- change in the pattern of previous migraine
- abnormal neurologic examination
- recent head trauma
- headache triggered by cough or Valsalva
- onset of migraine after age 50 years
- new onset of headache in an immunocompromised patient
- headache with fever.

## ANSWER 5

In patients with mild or moderate migraine pain, a combination of antiemetics, simple analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) can be used as an initial step. Triptans should be used if pain progresses to a moderate-to-severe intensity.<sup>6</sup>

Moderate-to-severe migraines should be treated early in the attack with a triptan. The evidence for combining triptans with aspirin or other NSAIDs and metoclopramide is limited but these combinations could be considered if a triptan alone is not beneficial.<sup>6</sup> Triptans are efficacious in about two-thirds of people and some benefit is usually experienced within 30–60 minutes of oral ingestion.<sup>6</sup> Antiemetics (metoclopramide or prochlorperazine) may be added as well.<sup>6</sup> Given the occurrence of nausea and vomiting early in Emma’s migraines, non-oral formulations (eg sumatriptan nasal spray, NSAID suppositories, rizatriptan wafers) are likely to be most effective. If the migraine improves initially but subsequently recurs, the triptan dose may be repeated in 2–4 hours. If, however, there is no initial response, a second dose is unlikely to be effective. If the triptan is consistently ineffective in treating migraines other triptans should be trialled.<sup>6</sup>

Opioids, including codeine preparations with paracetamol or aspirin, should be avoided in acute migraine management<sup>7</sup> as opioids have

little evidence for efficacy and they can predispose patients to the development medication-overuse headache.<sup>6</sup> As codeine-containing medications are easily accessible in Australia, patients should be asked directly about their intake of these medications and should be educated early about the risks of transformation of their problem to a chronic daily headache pattern.

### ANSWER 6

In general, preventive treatment should be initiated in patients with migraines with 2–3 or more attacks per month.<sup>6</sup> However, preventive medications may be considered for migraines occurring at any frequency that interferes with the patient's quality of life. In Emma's case, as the migraines occur every 1–3 months, the optimisation of acute treatment and implementation of lifestyle modifications are appropriate first steps in management.

The goals of migraine prevention are to decrease the frequency and severity of the attacks, reduce headache-associated disability and improve the response to acute treatment when breakthrough migraines occur.<sup>6</sup> An adequate trial of a preventive medication is usually at least 2 months (as it may take 1–3 months for the full effect to be experienced), during which time the medication is slowly titrated to an effective or maximally tolerated dose.<sup>6</sup> During this time headache frequency should be monitored with a calendar or diary.<sup>4,6</sup>

When choosing preventive treatments, the patient's comorbidities and the risk of adverse effects should be taken into account. First-line preventive agents for migraines include tricyclic antidepressants (amitriptyline, nortriptyline), beta-blockers (propranolol, metoprolol, atenolol) and pizotifen.<sup>6,8</sup> Of the beta-blockers, propranolol has the most evidence for efficacy and is approved for use in migraine, as is metoprolol, whereas atenolol is not. Although data are lacking, other tricyclic antidepressants such as nortriptyline and dothiepin may be effective if side effects limit the use of amitriptyline.<sup>8</sup> Pizotifen may be effective but its use is often limited by side effects including drowsiness and weight gain.<sup>8</sup>

If the first-line agents are ineffective or associated with unacceptable side effects, second-line options include sodium valproate, topiramate or verapamil sustained release.<sup>6,8</sup> Sodium valproate should be avoided in women of childbearing potential.<sup>6</sup> According to the Australian Medicines Handbook 2014, topiramate is as effective as valproate and probably as effective as propranolol. Topiramate is associated with a risk of fetal malformations and can impair the effectiveness of hormonal contraceptives.<sup>9</sup> Sodium valproate and verapamil sustained release are not marketed for migraine. Other drugs, such as gabapentin, are also used but often have limited evidence or efficacy.

If headaches are well controlled for 3–6 months, slow tapering of the preventive drug may be feasible, particularly in patients with less frequent migraine attacks, fewer years of migraine and fewer comorbid conditions such as depression, anxiety and fibromyalgia.<sup>10</sup>

### ANSWER 7

Lifestyle modifications can be very effective in reducing migraines and should be incorporated into Emma's daily routine. These include:

- hydration – at least 8 glasses of non-caffeinated drinks daily. Although caffeine is often beneficial in the acute treatment of migraines, daily consumption of more than 1 or 2 caffeinated beverages can result in the development of chronic daily headache
- sleep hygiene – recommending 8 hours of sleep at night. Bedtime and awakening should be at consistent times each evening and morning throughout the week, as lack of sleep and too much sleep can both trigger headaches
- exercise – incorporating 30–60 minutes of aerobic exercise at least 3 times per week
- meals – making sure Emma consumes three daily meals, including mid-morning and mid-afternoon snacks if hunger triggers headaches. Meals should be high in protein, vegetables and fibre, and low in fat and sugar. Highly processed foods or those with additives and preservatives are best avoided.

### ANSWER 8

Women with migraine with aura should be advised to avoid combined oral contraceptive (COC) pills.<sup>11,12</sup> A 2009 meta-analysis found that the risk of stroke was doubled in persons with migraine with aura, and a 3-fold increase was observed in the women with migraine with aura. The risk was increased further in those aged >45 years, smokers and women who use COCs.<sup>13</sup>

COCs may be used in women with migraine without aura who need or want contraception.<sup>11</sup> Although the use of COCs in migraine prevention is not clearly supported by the medical literature, the use of a monophasic low-dose (35 µg ethinyl oestradiol or less) COC may be beneficial in reducing menstrually related migraines by keeping oestradiol levels steady. For those women who continue to have menstrually related migraines, a continuous-dose regimen (skipping placebo pills) may be considered.<sup>14</sup>

Women with migraine without aura who are prescribed COCs should be counselled to report new-onset aura symptoms, and cardiovascular risk factors should be monitored.<sup>14</sup> If migraines worsen after starting the COC or if the patient develops an aura, the COC should be discontinued.<sup>14</sup> Note, guidelines generally do not recommend the use of COCs for women >35 years of age who have migraine without aura.<sup>15</sup>

Progestin-only contraceptives (oral or depot forms) have not been associated with an increased risk of stroke. The etonogestrel implant, the levonorgestrel-releasing intrauterine device and copper-containing intrauterine devices may be safer contraceptive options than the COC in women with migraine.<sup>16</sup>

The World Health Organization (WHO) has also set out medical criteria for the continuation of COCs when a woman develops migraine during their use. If migraine without aura is present and the woman is >35 years old, she should preferably stop using COCs (WHO Category 3). If she develops migraine without aura at an older age, or migraines with aura at any age, she must stop them (WHO Category 4).<sup>11</sup> If migraines with aura start during the use of progestin-only contraceptives, they should generally be suspended unless other more appropriate methods of contraception are not available (WHO Category 3).<sup>11,17</sup>

## REFERENCES

1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edn (beta version). *Cephalalgia* 2013;33:629.
2. Lipton RB, Dodick D, Sadovsky R, et al. A self-administered screener for migraine in primary care: the ID Migraine™ validation study. *Neurology* 2003;61:375–82.
3. Silberstein SD, Saper JF, Freitag FG. Migraine: diagnosis and treatment. In: Silberstein SD, Lipton RB, Dalessio DJ, editors. *Wolff's headache and other head pain*. 7th edn. New York: Oxford University Press, 2001. pp121–237.
4. National Institute for Clinical Excellence (NICE) guidelines. Headaches: Diagnosis and management of headaches in young people and adults. NICE clinical guideline 150 [Issued September 2012] Available at [www.nice.org.uk/nicemedia/live/13901/60853/60853.pdf](http://www.nice.org.uk/nicemedia/live/13901/60853/60853.pdf) [Accessed 5 May 2014].
5. Silberstein SD, Lipton RB, Dalessio DJ. Overview, diagnosis, and classification of headache. In: Silberstein SD, Lipton RB, Dalessio DJ, editors. *Wolff's headache and other head pain*. 7th edn. New York: Oxford University Press; 2001. pp6–26.
6. Rossi S, Editor. Migraine. *Australian Medicines Handbook*. In: *Australian Medicines Handbook 2014*. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
7. Neurology Expert Group. Headache: Migraine: Acute migraine attack. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
8. Neurology Expert Group. Headache: Migraine: prophylaxis of migraine attacks. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
9. Drugs and their categories in pregnancy and breastfeeding: In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 6 May 2014].
10. Evans RW, Loder E, Bondi DM. When can successful migraine prophylaxis be discontinued? *Headache* 2004;44:1040–42.
11. Endocrinology Expert Group. Hormonal contraception: combined hormonal contraception. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
12. World Health Organization. Family and Reproductive Health Programme. Improving access to quality care in family planning: medical eligibility criteria for contraceptive use (3rd edn). Geneva: World Health Organization, 2004.
13. Schürks M, Rist PM, Bigal ME et al. Migraine and cardiovascular disease: systemic review and meta-analysis. *BMJ* 2009;339:b3914.
14. Calhoun AH, Hutchinson S. Hormonal therapies for menstrual migraine. *Curr Pain Headache Rep* 2009;13:381–85.
15. Rossi S, editor. Combined oral contraceptives. *Australian Medicines Handbook*. In: *Australian Medicines Handbook 2014*. Adelaide: Australian Medicines Handbook Pty Ltd 2014.
16. Neurology Expert Group. Headache: Migraine: Migraine in women. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
17. ACOG Committee on Practice Bulletins-Gynecology. ACOG practice bulletin. No73: Use of hormonal contraception in women with coexisting medical conditions. *Obstet Gynecol* 2006;107:1453–72.



## CASE 5 ANSWERS

## ANSWER 1

Headache is one of the most common neurological problems that presents to a GP or a neurologist. It is also one of the most common acute and chronic pain conditions. Tension-type headache and migraine are the second and third most prevalent medical disorders.<sup>1</sup> Migraine accounts for 30% of the global burden and more than 50% of the disability burden attributable to all neurological diseases worldwide. Overall, migraine is the fourth ranking cause among women and the seventh ranking cause of all disease-associated disability worldwide.<sup>1</sup> Thus headache and migraine are major problems worldwide. The *Atlas of Headache Disorders and Resources in the World 2011*<sup>2</sup> states that 'Headache disorders are ubiquitous, prevalent and disabling. Yet they are under-recognized, under-diagnosed and under-treated'.

Taking a detailed history is very important in a presentation such as this. It is important to establish:

- how quickly the headache came on
- how severe it was (was it 10/10?)
- what was Michael doing at the time
- how long did the headache last, including the severe component
- whether there was any residual pain
- whether there were any associated features.

A thunderclap headache is a severe headache, often the worst headache the patient has experienced, that comes on usually within a matter of seconds and which reaches its maximum within seconds to about a minute. A headache that reaches 7/10 or more in terms of pain within less than a minute is a thunderclap headache.<sup>3</sup> It is one of the red flags of headache.<sup>3</sup> The initial severe headache can persist for a variable period of time and there is often a less severe headache lasting for a variable amount of time thereafter. Some thunderclap headaches may be associated with loss of consciousness, nausea and vomiting, neck stiffness and other neurological symptoms. They may occur only once or be recurrent, depending on the cause.<sup>4</sup>

## ANSWER 2

Irrespective of whether there are accompanying symptoms and even if the patient has a past history of headache, the first diagnosis to consider as the cause for a thunderclap headache is a subarachnoid haemorrhage (SAH). Seventy percent of patients with a SAH present with a headache, 50% of which are thunderclap headache.<sup>4</sup> Up to 25% of patients with a thunderclap headache have a subarachnoid haemorrhage, usually due to a ruptured aneurysm, but other serious conditions that should be considered are cervical artery dissection, intracerebral haemorrhage, cerebral vein thrombosis and the reversible cerebral vasoconstriction syndrome (RCVS).<sup>4</sup>

The patient needs urgent referral to an emergency department for an initial non-contrast computed tomography (CT) brain scan and lumbar puncture, if the CT shows no blood. A brain CT without contrast, performed in the first 12–24 hours, has been shown to be highly sensitive and specific for the diagnosis of subarachnoid haemorrhage.<sup>5</sup> The sensitivity of CT decreases in the ensuing days from 86% on day 2, to 58% after 5 days.<sup>6</sup> Other investigations, such as magnetic resonance imaging (MRI) of the brain, may also be needed.<sup>4,5</sup>

## ANSWER 3

Additional questions that Michael could be asked include:

- Did the attacks occur randomly or were they associated with certain activities and what was the temporal pattern, if any?
- Has he been taking any new medications or illicit drugs recently?

Some thunderclap headaches occur repeatedly with coughing, sneezing or other Valsalva manoeuvre, or with sexual activity.<sup>4</sup> These headaches need to be thoroughly investigated for an underlying structural cause such as an Arnold-Chiari malformation, hydrocephalus, a colloid cyst of the third ventricle or intracranial hypotension.<sup>7</sup> In the RCVS, thunderclap headaches occur repeatedly over a short period of time (several weeks to a month) and are self-limiting.<sup>7</sup> They can occur with or without neurological symptoms and severe hypertension may be present.

In addition to the above investigations, imaging of the cerebral vasculature with magnetic resonance angiography (MRA) and CT or digital subtraction angiography of the cerebral arteries is mandatory. The characteristic radiological findings are of alternating segments of cerebral arterial constriction and dilatation (so called beading). This may need to be repeated over time as the changes may be present only intermittently despite recurrent attacks.<sup>7</sup> RCVS is usually benign, but ischaemic infarction and intracerebral or subarachnoid haemorrhage can occur and will be detected with CT and MRI. The RCVS can be triggered by sexual activity, exertion, emotion or by certain drugs such as sympathomimetic or illicit drugs that can cause vasoconstriction.<sup>7</sup> Women in the postpartum period are particularly at risk of the RCVS but can also have thunderclap headaches due to eclampsia or cerebral vein thrombosis.<sup>8</sup>

## ANSWER 4

Primary headaches are those where, at present, there is no known underlying structural pathology.<sup>7</sup> Migraine and tension type headache are the most frequently occurring primary headaches. Primary cough headache, primary headache associated with sexual activity and primary thunderclap headaches can all present with thunderclap headaches. Primary cough headache is more common in older men and responds to indomethacin.<sup>7</sup> Sixty percent of patients with isolated sexual headaches have RCVS.<sup>4</sup> Thus a thunderclap headache due to a primary headache is a diagnosis of last resort and can only be made after extensive investigations of the patient to rule out an underlying cause.<sup>9</sup>

**REFERENCES**

1. World Health Organization. Atlas of Headache Disorders and Resources in the World. Trento: WHO; 2011. p1–160. Available at [www.who.int/mental\\_health/management/who\\_atlas\\_headache\\_disorders.pdf?ua=1](http://www.who.int/mental_health/management/who_atlas_headache_disorders.pdf?ua=1) [Accessed 8 July 2014].
2. Murray CJL, Vos T, Lozano R, et al. Disability adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2197–223.
3. Dodick DW. Diagnosing headache: clinical clues and clinical rules. *Adv Studies Med* 2003;3:S550–55.
4. Ducros A, Bousser MG. Thunderclap headache. *BMJ* 2012;345:1–7.
5. Schwedt TJ, Dodick DW. Thunderclap headache. In: Swanson JW, Dashe KF, editors. *UpToDate*. Wolters Kluwer, 2014. Available at [www.uptodate.com/contents/thunderclap-headache?topicKey=NEURO%2F3346&elapsedTimeMs=13&view=print&displayedView=full](http://www.uptodate.com/contents/thunderclap-headache?topicKey=NEURO%2F3346&elapsedTimeMs=13&view=print&displayedView=full) [Accessed 6 May 2014].
6. van Gijn J, van Dongen KJ. The time course of aneurysmal haemorrhage on computed tomograms. *Neuroradiology* 1982;23:153–56.
7. Headache Classification Committee of the International Headaches Society. The International Classification of Headache, 3rd edition (beta version). *Cephalalgia*. 2013;33:629–808. Available at [www.ihs-headache.org/binary\\_data/1437\\_ichd-iii-beta-cephalgia-issue-9-2013.pdf](http://www.ihs-headache.org/binary_data/1437_ichd-iii-beta-cephalgia-issue-9-2013.pdf) [Accessed 29 May 2014].
8. Treadwell SD, Thanvi B, Robinson TG. Stroke in pregnancy and the puerperium. *Postgrad Med J* 2008;84:238–45.
9. Schwedt TJ. Thunderclap headaches: a focus on etiology and diagnostic evaluation. *Headache* 2013;53:563–69.

**CASE 6**

**DAVID HAS SPEECH DIFFICULTIES**

David is 65 years of age and presents to your clinic at 1 pm with a history of speech difficulties, which he describes as 'talking gibberish', that lasted about 10 minutes when he was out shopping at 11 am. He has a history of hypertension treated with perindopril 2.5 mg daily and smokes 5 cigarettes a day. His body mass index (BMI) is 32 kg/m<sup>2</sup> and his blood pressure today is 145/85 mmHg.

**QUESTION 1** 

What clinical features will help localise the pathology in the nervous system?

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**QUESTION 2** 

What is the differential diagnosis?

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**QUESTION 3** 

What investigations need to be performed and with what urgency?

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**FURTHER INFORMATION**

You now suspect that David had a TIA.

**QUESTION 4** 

What management should be initiated and with what urgency?

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**FURTHER INFORMATION**

You find an irregular pulse on examination and ECG confirms atrial fibrillation.

**QUESTION 5** 

What management should be initiated and with what urgency?

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**FURTHER INFORMATION**

Left internal carotid artery stenosis of 70% and right internal carotid artery stenosis of 60% was found on Doppler ultrasound.

**QUESTION 6** 

How is this best managed?

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**FURTHER INFORMATION**

On more detailed examination you find that David still has difficulty with high-level naming and there is a loss of the right nasolabial fold.

**QUESTION 7** 

Would the diagnosis still be TIA?

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**QUESTION 8** 

What management should occur?

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**CASE 6 ANSWERS**

**ANSWER 1**

Speech disturbance can be due to dysarthria (poor articulation) or dysphasia (language disturbance – comprehension, fluency, naming). In general, patients can distinguish between slurring the correct words (dysarthria) and using the wrong words or ‘talking gibberish’ (dysphasia). Dysphasia localises to the left hemisphere in right-handed patients and most left-handed patients. Dysarthria can involve either hemisphere or brainstem (non-localising). The presence of associated symptoms, including facial droop, sensory loss, ‘heaviness’/weakness, is very helpful to confirm the affected hemisphere. Diplopia or vertigo localise to the posterior circulation (brainstem/cerebellum).

**ANSWER 2**

The following conditions could be considered in the differential diagnoses for this presentation.

**Transient ischemic attack (TIA) or stroke**

TIA occurs when there is a temporary occlusion of a cerebral artery that resolves without causing brain infarction. It is the most important possible diagnosis. The definition of TIA has evolved with the removal of the old ‘resolves within 24-hour’ criterion. This caused confusion when a patient presented with symptoms of a few hours’ duration, leading to a frequent question ‘how do you know it’s not a TIA as it could still resolve by 24 hours?’. The average duration of a TIA is about 10 minutes.<sup>1</sup> If symptoms are still present when the patient is seen then the diagnosis is stroke not TIA and needs to be treated differently. The new definition is based on the ‘absence of infarction on brain imaging’.<sup>1</sup> However, this depends on the type of imaging modality used, either computerised tomography (CT) or the much more sensitive diffusion magnetic resonance imaging (MRI). Up to 40% of fully resolved events that are regarded as TIA on the basis of clinical presentation have small diffusion lesions on MRI (technically a stroke) and have a much higher risk of recurrent stroke.<sup>1</sup>

**Migraine**

Migraine can cause transient dysphasia, dysarthria, sensory symptoms and weakness. The headache can follow or be mild or absent in some cases. It is unusual to have a first episode of migraine at the age of 65 years so the patient’s history is important. A history of symptom migration (eg paraesthesia spreading from face to hand over about 15 minutes) is suggestive of migraine as cortical spreading depression (a wave of electrophysiological hyperactivity followed by a wave of inhibition) propagates across the cortex at about 2–5 mm/min.

**Seizure**

Seizure usually causes positive phenomena (paraesthesia or jerking) rather than loss of function.

**Metabolic disturbance**

A metabolic disturbance, such as hypoglycaemia, often causes generalised symptoms and can lead to a focal neurological deficit in some cases.

**Intercurrent illness**

Intercurrent illness with worsening of an old deficit can occur (eg facial droop re-emerging in the context of a urinary tract infection).

**Functional illness**

Functional illness is surprisingly common in neurological practice. There may be features on examination (eg Hoover's sign: first test hip extension in the 'weak' leg; leave your hand under the ankle while testing the contralateral hip flexion. Normal co-activation of the contralateral 'weak' hip extensor muscles indicates functional weakness) but this can be difficult to identify even for neurologists.

**ANSWER 3**

The following investigations need to be performed, preferably on the same day.<sup>2,3</sup> The ability to arrange these investigations will depend on the location and the resources available. The local hospital may have a rapid access TIA clinic or a rapid assessment pathway through the emergency department.

- CT brain scan provides a relatively low yield but is important to exclude small bleeds, subdural haematoma, tumour, etc. MRI diffusion imaging has greater sensitivity for differentiating stroke (abnormal) from TIA (normal) but may not be readily available.
- Carotid imaging using carotid Doppler ultrasound or CT angiogram can reveal symptomatic carotid artery stenosis. When performed early, carotid endarterectomy has been found to significantly reduce subsequent stroke risk.<sup>2</sup>
- An electrocardiogram should be performed routinely to look primarily for atrial fibrillation (also old Q-waves may indicate an akinetic ventricular segment that occasionally can lead to mural thrombus).<sup>2</sup> Note, atrial fibrillation may be paroxysmal (and therefore not present at the time the patient is seen). Paroxysmal atrial fibrillation has the same stroke risk as permanent atrial fibrillation.<sup>4</sup> Holter monitor data may be useful in detecting some cases of atrial fibrillation but may still miss some paroxysmal atrial fibrillation so it is important to continue to monitor the patient clinically at every opportunity.

**ANSWER 4**

The current approach to stroke secondary prevention is based on the concept of absolute cardiovascular risk. By definition, a patient with a TIA is at high risk and should receive rapid treatment of all vascular risk factors, regardless of the individual risk factor level.<sup>2</sup>

The following should be considered:

- Antiplatelet treatment – aspirin is generally used (eg a loading dose of 300 mg followed by 100 mg daily).<sup>5</sup> A combination of aspirin plus dipyridamole is also available and slightly more effective but is generally commenced once daily in combination with 100 mg aspirin and then increased to twice daily after 1–2 weeks (with cessation of separate aspirin) to reduce the incidence of headache. Clopidogrel, which is slightly more effective than aspirin and equally effective as aspirin plus dipyridamole,<sup>6</sup> is an option for those intolerant of aspirin or who have had their TIA while already taking aspirin. Combined aspirin plus clopidogrel is not recommended long term for stroke prevention but a recent trial

(CHANCE) has suggested some benefit of short-term combination therapy for about 1 month in high-risk patients.<sup>7</sup>

- Statins – on the basis of a randomised trial using 80 mg atorvastatin,<sup>8</sup> statins reduce recurrent stroke regardless of baseline cholesterol.
- Antihypertensive agents – hypertension is a particularly potent risk factor for stroke with no 'threshold' for benefit (ie the lower the blood pressure, the lower the stroke risk).<sup>2</sup> Angiotensin converting enzyme inhibitors, angiotensin 2 receptor antagonists and calcium antagonists are all reasonable first-line choices.<sup>2</sup> Given that David's blood pressure is 145/85 mmHg, increasing his perindopril dose to target his blood pressure to <135/80 mmHg would be a reasonable start, although, as mentioned, the target level is arbitrary since lower is better provided postural hypotension is not problematic.

With regards to urgency, management of risk factors should commence on the same day (after CT scan). A study in the UK (EXPRESS) showed that stroke risk could be reduced by 80% simply by starting these medications immediately rather than delaying by a couple of weeks.<sup>9</sup>

Additional factors that should be considered for risk factor reduction are listed below. Management should be individualised and delivered using behavioural techniques, such as motivational interviewing techniques:<sup>2</sup>

- smoking cessation – offer counselling and/or behavioural therapy and consider use of pharmacological therapies (eg nicotine replacement therapy, bupropion)
- screen for diabetes with fasting glucose (oral glucose tolerance test (OGTT) could also be performed and manage according to national diabetes guidelines
- increasing exercise – for example, recommend a 30 minute brisk walk every day
- diet – provide information/education on following a low-fat diet, high in fruit and vegetables, and advise to avoid fried and processed foods, not to add salt to food (following a Mediterranean diet and eating tree nuts may reduce risk<sup>10</sup>).

**ANSWER 5**

Atrial fibrillation is a major risk factor for stroke and anticoagulation is underused. Unless there is a very strong reason not to use anticoagulants (eg active gastrointestinal bleeding) David should start anticoagulation treatment immediately. He could be started on warfarin, using an appropriate protocol to initiate dosing, to a target INR of 2–3.<sup>4</sup> If the creatinine clearance is >50 ml/min, a non-vitamin K oral anticoagulant (NOAC; eg apixaban 5 mg BD, dabigatran 150 mg BD or rivaroxaban 20 mg daily) could be used.<sup>4,11</sup> If the creatinine clearance (CrCl) is 30–50 ml/min, dose modification is generally required and all patients on NOACs should have CrCl monitored periodically.<sup>4,11,12</sup>

Note, onset of anticoagulation is 2–3 hours for NOACs versus several days for warfarin, and there are no accepted antidotes for the NOACs at this time.<sup>11</sup>

**ANSWER 6**

It is important to localise the hemisphere affected, as an asymptomatic stenosis does not warrant surgical intervention. As David has dysphasia, the left internal carotid artery stenosis is symptomatic.

Enderarterectomy reduces the rate of recurrent stroke for symptomatic stenosis by >70% with more marginal benefit for stenosis 50–70%. If there is uncertainty regarding the severity of stenosis on Doppler ultrasound, a CT angiogram could be performed. Surgery should be performed as soon as possible after a TIA and certainly within 2 weeks, as the highest risk of recurrent stroke is early. The asymptomatic right carotid stenosis will benefit from intensive medical management (statins, antiplatelet and antihypertensive agents) and there is no need for surgical intervention.<sup>2</sup>

**ANSWER 7**

The absence of residual symptoms at the time of examination indicates the diagnosis is probably stroke (in the left middle cerebral artery territory) not TIA.

**ANSWER 8**

You should call an ambulance. The patient will be taken to the nearest stroke-equipped hospital. As the patient is within 4.5 hours from the onset of symptoms, thrombolysis may be an option depending on severity, brain imaging findings and other clinical criteria. Thrombolysis with intravenous administration of tissue plasminogen activator within 4.5 hours of the 'last known well time' has level 1 evidence (meta-analysis of randomised trials) showing significant reductions in disability and a neutral effect on mortality.<sup>13</sup> Regardless of time window, all patients with stroke should be managed in a stroke unit, which reduces the risk of disability and death for all age groups, stroke subtypes and severities.<sup>2</sup>

**KEY POINTS**

- TIA is a medical emergency. It is characterised by a sudden onset of focal neurology with complete resolution and has an average duration of about 10 minutes.
- If symptoms/signs are still present when the patient is seen then the diagnosis is stroke not TIA; call 000 and send the patient to a stroke-equipped emergency department.
- The aim of TIA management is to prevent a subsequent stroke (the highest risk is in the subsequent 48 hours).
- A CT brain scan excludes diagnoses that mimic TIA (tumour, small bleed); carotid imaging (Doppler US or CT angiogram) can be used to investigate for symptomatic carotid stenosis.
- Detecting atrial fibrillation is critical but difficult as it can be paroxysmal so an electrocardiogram should be performed and use of a Holter monitor considered, but the patient should be monitored clinically at every opportunity.
- NOACs (subject to CrCl) or warfarin should be commenced immediately if a TIA occurs as a consequence of atrial fibrillation
- In the absence of atrial fibrillation, most patients should be immediately commenced on antiplatelet treatment (aspirin, aspirin

plus dipyridamole or, if PBS criteria are met, clopidogrel), statin and antihypertensive agents.

- Carotid endarterectomy reduces the risk of recurrent stroke for symptomatic carotid stenosis by >70% (with a smaller benefit if 50–70% stenosis).
- Asymptomatic carotid stenosis should be managed with intensive medical therapy if symptoms/signs are still present (the diagnosis is stroke) the time window for standard thrombolysis is 4.5 hours from the time the patient was last seen well.
- Even if >4.5 hours have elapsed, every stroke patient should be assessed and managed in a stroke unit.

**REFERENCES**

1. Easton JD, Saver JL, Albers GW, et al. Definition and Evaluation of Ttransient Ischemic Attack: A Scientific Statement for Healthcare Professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. *Stroke* 2009;40:2276–93
2. National Stroke Foundation. Clinical Guidelines for Stroke Management 2010. Melbourne: National Stroke Foundation. Available at [http://strokefoundation.com.au/site/media/clinical\\_guidelines\\_stroke\\_managment\\_2010\\_interactive.pdf](http://strokefoundation.com.au/site/media/clinical_guidelines_stroke_managment_2010_interactive.pdf) [Accessed 30 May 2014].
3. Neurology Expert Group. Stroke and transient ischemic attack: Management: Transient ischemic attack. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 30 April 2014].
4. Cardiovascular Expert Group. Atrial tachyarrhythmias: atrial fibrillation. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 30 April 2014].
5. Rossi S, Editor. Aspirin. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
6. Sacco RL, Diener HC, Yusuf S, et al. Aspirin and extended-release dipyridamole versus clopidogrel for recurrent stroke. *N Engl J Med* 2008;359:1238–51.
7. Wang Y, Zhao X, Liu L, et al. Clopidogrel with aspirin in acute minor stroke or transient ischemic attack. *N Engl J Med* 2013;369:11–19.
8. Amarenco P, Bogousslavsky J, Callahan A, 3rd, et al. High-dose atorvastatin after stroke or transient ischemic attack. *N Engl J Med* 2006;355:549–59.
9. Rothwell PM, Giles MF, Chandratheva A, et al. Effect of urgent treatment of transient ischaemic attack and minor stroke on early recurrent stroke (express study): a prospective population-based sequential comparison. *Lancet* 2007;370:1432–42.
10. Estruch R, Ros E, Salas-Salvadó J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med* 2013;368:1279–90.
11. Rossi S, Editor. Oral anticoagulants. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
12. Rossi S, Editor. Factor Xa inhibitors. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
13. Lees KR, Bluhmki E, von Kummer R, et al. Time to treatment with intravenous alteplase and outcome in stroke: an updated pooled analysis of ECASS, ATLANTIS, NINDS and EPITHET trials. *Lancet* 2010;375:1695–1703

**RESOURCES FOR DOCTORS**

- National Stroke Foundation Clinical Guidelines for Stroke Management 2010, [http://strokefoundation.com.au/site/media/clinical\\_guidelines\\_stroke\\_managment\\_2010\\_interactive.pdf](http://strokefoundation.com.au/site/media/clinical_guidelines_stroke_managment_2010_interactive.pdf)

**CASE 7**

**ROB HAS A DISABLING HEADACHE**

Rob is a labourer aged 30 years. He comes to your practice with his wife to discuss a 5-year history of headaches. These headaches are disabling, causing 1–2 months of sick leave each year when they occur. The pain is mainly behind the left eye and temple, and radiates to the vertex. He rarely experiences pain on the right side. The pain comes on rapidly (within minutes) and Rob describes it as ‘20/10’ in intensity. It is so excruciating that it often causes him to double over and writhe around. It occurs, predictably, soon after falling asleep. His wife states that he may have multiple episodes of headache (sometimes up to 4) occurring daily. Each episode seems to last 0.5–2 hours. After 1–2 months of this pattern, the headaches subside and he feels well again ‘for months until another attack’. Magnetic resonance imaging (MRI) of the brain was performed recently and was normal. His neurological examination is normal.

**QUESTION 1** 

What are differential diagnoses for Rob’s headache?

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**QUESTION 2** 

Of the short-lasting head pain syndromes, what headache types are consistent with the attack frequency described by Rob?

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**FURTHER INFORMATION**

Rob experiences an attack in the examination room. During the attack, he has sudden onset of severe pain behind the left eye and grabs his left side with his hands. You notice that he is shielding his left eye and is pacing restlessly around the room. The pain is so severe that he indicates that he cannot converse or answer any further questions. He vomits. When questioned, his wife indicates that this is a typical event for Rob. The attack subsides after 30 minutes. On examination you notice that Rob’s left eye is red and he has had tearing. He also has obvious nasal congestion.

**QUESTION 3**  

What further information do you need to clarify the diagnosis? Specifically, what direct questions do you need to ask?

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**FURTHER INFORMATION**

Rob’s wife states that they have seen five different GPs so far over the years and the most common diagnoses have been sinusitis or migraine headache. He has been previously treated with muscle relaxants, gabapentin, beta-blockers and long- and short-acting nonsteroidal anti-inflammatory drugs (NSAIDs). Recently, a doctor started him on indomethacin, which ‘did absolutely nothing’ to relieve the headaches. He has now become reliant on opioids, often requiring intramuscular morphine for acute attacks.

**QUESTION 4** 

How does the insensitivity to indomethacin help in further defining the diagnosis?

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**QUESTION 5** 

What is your approach to acute treatment now, during the attack?

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**QUESTION 6** 

What preventive treatment options are most likely to be effective in this patient?

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**FURTHER INFORMATION**

The severity of his condition has led to Rob often losing his job as a laborer around the times of the cluster headache attacks every year. He has tried to decrease his opioid use but has had symptoms of palpitations, nausea and sweating when attempting to cut down. He also reports fatigue, frequent spells of crying, night sweats and generally feeling 'helpless'. He tells you that he 'cannot take this anymore' but denies suicidality.

**QUESTION 7** 

What is the most appropriate course of action at this time?

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**CASE 7 ANSWERS**

**ANSWER 1**

Possibilities for unilateral headache include migraine, hemicrania continua, and trigeminal autonomic cephalalgias (TAC). The latter include cluster headache, paroxysmal hemicranias and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT). In addition to TAC, other short-lasting headaches, primary stabbing headache (ie icepick headache) and trigeminal neuralgia, should also be considered (Table 1).<sup>1</sup>

**Table 1. Short-lasting headaches<sup>1</sup>**

Trigeminal autonomic cephalalgias (TAC)	Cluster headache Paroxysmal hemicranias SUNCT
Other short-lasting headaches	Hemicrania continua Primary stabbing headache Trigeminal neuralgia Primary cough headache Primary exertional headache Primary sex headache

Although migraine is unilateral in approximately 60% of patients and can have a rapid/sudden onset, it is normally of a longer duration (by definition 4–72 hours). Also, this patient is not in a typical age group where new-onset migraine would be common.<sup>1,2</sup> Therefore, this is unlikely to be a primary migraine headache.

Hemicrania continua is a constant, continuous, strictly one-sided headache punctuated by severe pain exacerbations. It affects the temple or periorbital region and usually affects the same side of the head. It is routinely misdiagnosed because patients only report the exacerbations that occur, which often have varying frequency several times a week. These exacerbations can last from minutes to several days with an intensity ranging from mild to severe.<sup>3</sup> The underlying pain is constantly present, 24 hours a day, every day of the week.<sup>1,4</sup> However, the pain of hemicrania continua is reported to be not as excruciatingly severe as the pain of cluster headache.<sup>3</sup> Some patients have associated symptoms common to migraine, such as nausea, vomiting, photophobia and phonophobia. They may also have autonomic symptoms and similarities in clinical presentation, treatment and pathophysiology.<sup>4</sup> Thus, hemicranias continua is commonly regarded as the fourth TAC-type headache, but it differs from the TACs in having continuous underlying pain. For this reason they are not included as a TAC in the International Classification for Headache Disorders.<sup>1</sup>

Trigeminal neuralgia consists of severe, sharp, electric or knifelike repetitive attacks lasting seconds, usually in a V2 or V3 distribution, sometimes radiating into the teeth. It rarely occurs in a V1 distribution and thus is not consistent with this Rob's pain distribution.<sup>5</sup>

Primary stabbing headache is predominantly reported over a V1 distribution (orbit, temple, parietal areas) and is stabbing in character as the name suggests, so it should be considered in Rob's case. The

main feature that helps to distinguish primary stabbing headache from the TAC headaches is the presence of any associated autonomic features.<sup>5,6</sup>

### ANSWER 2

Attack duration and frequency are key components in differentiating headache type and thus management (*Table 2*).<sup>7</sup>

Cluster headaches are typically 15–180 minutes in duration (average 45–90 minutes) and are more variable in their frequency (range 3–8 per day).<sup>1</sup> They also have a consistent circadian rhythm, with attacks occurring at a similar time each day and they commonly occur soon after falling asleep. There also tends to be a seasonal periodicity with cluster headaches.<sup>8</sup> Typically, the headache occurs daily for 8–10 weeks per year (*Table 3*).<sup>9</sup> Patients are generally asymptomatic between attacks. Rob's history is most consistent with this type of headache.

Attack duration in paroxysmal hemicranias last for 2–45 minutes and patients report 15 or more attacks per day (usual frequency being 1–40).<sup>1,8</sup> Therefore, the shorter duration and the greater frequency of paroxysmal hemicranias are inconsistent with Rob's headache syndrome.

In SUNCT, pain episodes last for 15–120 seconds and the frequency of attacks is about 3–100 episodes per day,<sup>1,8</sup> which clearly does not fit Rob's case.

Cluster headache can be further differentiated into episodic and chronic. Episodic cluster headache is defined by recurrent episodes of headache daily or every other day and lasting more than 1 week, separated by remissions lasting more than 1 month. This can occur once or twice a year, but can continue in this pattern for several years. Chronic cluster headache occurs in only 20% of those with diagnosed cluster headache and is defined as no remission within 1 year or a remission period that lasts <1 month.<sup>5,10</sup>

### ANSWER 3

It would be important to clarify the presence or absence of associated features. A significant differentiating feature between the TAC group and other short-lasting head pain syndromes is the presence of associated features, particularly autonomic features in the former (*Table 4*).<sup>7</sup> Cranial autonomic symptoms are cardinal features of the TACs, especially cluster headache. This includes lacrimation, nasal congestion, conjunctival injection and rhinorrhoea, miosis, eyelid swelling or ptosis.<sup>8</sup> Because of this, misdiagnosis of sinus headache is common in these patients and in a clinical study, nearly 25% of these patients were being treated by an ear, nose and throat specialist for sinusitis.<sup>11</sup> The underlying pathology is thought to be cranial parasympathetic activation related to pain (rather than local inflammation).<sup>12</sup>

Other associated features can also include migraine-like features, such as nausea or vomiting, photophobia and phonophobia. The latter two symptoms are usually on the same side as the headache in TAC.<sup>5,8</sup> This lateralisation of symptoms and signs is an important feature of TAC attacks, particularly for cluster headache, and is a key differentiating feature of TAC headaches.<sup>8</sup> By contrast, in migraine the features are usually bilateral, less prominent and variable in presentation.

Other important information to ascertain about Rob's headaches is whether there are specific triggers for the headaches. Alcohol is often a strong precipitating factor of the pain in cluster headache,<sup>7</sup> differentiating it from SUNCT (which does not have an alcohol trigger)<sup>7</sup> and paroxysmal headache, where only one-fifth of patients have this trigger.<sup>7</sup> Cutaneous triggers, such as touching the skin, talking and chewing, do not trigger cluster headache, whereas they are significant triggers in SUNCT.<sup>13,14</sup>

The history should include detailed knowledge of the patient's past history, including headaches, and his family history. This information may affect treatment choices and further management.

**Table 2. Differential diagnosis of short lasting headaches<sup>7</sup>**

Feature	Cluster headache	Paroxysmal hemicrania	SUNCT*	Idiopathic stabbing headache	Trigeminal neuralgia	Hypnic headache
Sex (male:female)	5:1	1:2	2:1	F>M	F>M	5:3
Pain						
Type	Boring	Boring	Stabbing	Stabbing	Stabbing	Throbbing
Severity	Very severe	Very severe	Severe	Severe	Very severe	Moderate
Location	Orbital	Orbital	Orbital	Any	V2/V3>V1	Generalised
Duration	15–180 minutes	2–45 minutes	15–120 seconds	<30 seconds	<1 seconds	15–30 minutes
Frequency	1–8/day	1–40/day	1/day–30/hour	Any	Any	1–3/night
Autonomic	+	+	+	–	–†	–
Trigger	Alcohol, nitrates	Mechanical	Cutaneous	None	Cutaneous	Sleep
Indomethacin	?	+	–	+	–	+

\*Short lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing

†Cranial autonomic activation may be seen in first division trigeminal neuralgia

**Table 3. Diagnostic criteria for cluster headache<sup>9</sup>**

3.1 Diagnostic criteria
A. At least five attacks fulfilling B–D
B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15–180 minutes if untreated
C. Headache is accompanied by at least one of the following <ol style="list-style-type: none"> <li>1. Ipsilateral conjunctival injection and/or lacrimation</li> <li>2. Ipsilateral nasal congestion and/or rhinorrhoea</li> <li>3. Forehead and facial sweating</li> <li>4. Ipsilateral eyelid oedema</li> <li>5. Ipsilateral forehead and facial sweating</li> <li>6. Ipsilateral miosis and/or ptosis</li> <li>7. A sense of restlessness or agitation</li> </ol>
D. Attacks have a frequency from one every other day to eight per day
E. Not attributed to another disorder

**3.1.1 Episodic cluster headache**

Description: Occurs in periods lasting seven days to one year separated by pain free periods lasting one month or more  
 Diagnostic criteria:

- a. All fulfilling criteria A–E of 3.1
- b. At least two cluster periods lasting from 7 to 365 days and separated by pain free remissions of one month or more

**3.1.2 Chronic cluster headache**

Description: Attacks occur for more than one year without remission or with remissions lasting less than one month  
 Diagnostic criteria:

- a. All alphabetical headings of 3.1
- b. Attacks recur for more than one year without remission periods or with remission periods lasting less than one month

**Table 4. Primary short-lasting headache<sup>7</sup>**

Prominent autonomic features	Sparse or no autonomic features
Cluster headache	Trigeminal neuralgia
Paroxysmal hemicrania	*Idiopathic stabbing headache
SUNCT syndrome	Cough headache
	Benign exertional headache
	Headache associated with sexual activity
	Hypnic headache

\*Likely to be renamed primary stabbing headache when the International Headache Society Classification<sup>1</sup> is revised.<sup>9</sup>

**ANSWER 4**

Most of the headache types mentioned thus far respond to indomethacin, except for cluster headache. Paroxysmal hemicranias and hemicranias continua, in particular, are responsive to indomethacin. Indeed, a response to indomethacin is required in these cases for diagnostic certainty.<sup>15</sup> Rob's lack of response to indomethacin is helpful in eliminating these forms of headache.<sup>16</sup>

**ANSWER 5**

Cluster headache is often considered one of the most painful of all headache types. During attacks, patients are unable to remain still and they move constantly, as was seen in Rob's case earlier. By contrast, patients with migraine prefer to remain still.

Acute treatment needs to reach the full therapeutic response rapidly and have a rapid onset. Many patients respond well to oxygen inhalation, which should be administered as at 10 L/min (range 7–12 L/min) for 15 minutes.<sup>17,18</sup> This is best given via a mask to enable the high-flow rate.<sup>17–20</sup> According to the *Australian Medicines Handbook*, inhaled oxygen relieves symptoms in 70–80% of people within 15 minutes.<sup>18</sup> Oxygen therapy should be stopped after 15 minutes if there is no improvement in status, as there is a risk of oxygen toxicity with longer treatment.<sup>20</sup> Of note, oxygen therapy does not alleviate the other forms of TAC headache.

Other acute treatment options include subcutaneous sumatriptan at a dose of 6 mg.<sup>18,21</sup> Sumatriptan has a rapid onset of action and is the only drug that has approval from the US Food and Drug Administration (FDA) for treatment for cluster headache. Nasal sprays of sumatriptan (20 mg) or zolmitriptan (5 mg) are also effective in acute cluster headache.<sup>22</sup> Note, zolmitriptan nasal spray is not currently registered in Australia. The *Australian Medicines Handbook* indicates that although there is less evidence for the efficacy of sumatriptan nasal spray, compared with subcutaneous sumatriptan, it may improve or eradicate a cluster headache within 30 minutes of use.<sup>18</sup> The bitter taste of the nasal spray may render this treatment unacceptable for some.<sup>21</sup> Conversely, treatment with oral sumatriptan (eg 100 mg tds) is ineffective and can be associated with medication-overuse headache.<sup>23</sup>

Table 5 outlines current treatment recommendations for cluster headache supported by Australian guidelines. The National Institute for Clinical Excellence 2012 headache guidelines do not recommend use of paracetamol, NSAIDs, opioids, ergotamine or oral triptans for the acute treatment of cluster headache.<sup>17</sup>

**Table 5. Current Australian treatment recommendations for cluster headache**

Type of treatment	Treatment
Acute	Oxygen inhalation for up to 15 minutes <sup>18,20</sup> Sumatriptan, subcutaneous (6 mg) <sup>18,20</sup> Sumatriptan nasal spray (20 mg) – authority streamlined <sup>18,20,24</sup> Lignocaine 4% solution instilled into the nose on the side of the pain <sup>20</sup> A short course of high-dose corticosteroids <sup>18</sup>
Prevention	Initially, verapamil 240 mg daily (usual range 240–960 mg daily in 1–4 doses depending on the formulation) <sup>25</sup> OR Verapamil sustained-release 160 or 180 mg orally, once daily, up to 360 mg daily <sup>20</sup> OR Lithium 250 mg orally, twice daily, titrate according to clinical response and tolerance, guided by serum concentration levels <sup>20</sup>

**ANSWER 6**

Verapamil is considered first-line for preventive treatment<sup>18</sup> and although it is not marketed for this indication in Australia, prophylaxis of cluster headache is an accepted indication.<sup>25</sup> For cluster headache, a patient should be started on 240 mg daily (usual dose range 240–960 mg daily in 1–4 doses depending on the particular formulation used).<sup>25</sup> Alternatively, the Therapeutic Guidelines recommend use of verapamil sustained-release at doses of 160 or 180 mg orally, once daily, with total daily doses up to 360 mg daily.<sup>20</sup> As patients often require high doses, side-effects must be adequately screened and monitored. Common side-effects (frequency >1%) include nausea, vasodilatory effects such as headache and flushing and peripheral oedema.<sup>25</sup> Infrequent side effects (0.1–1% incidence) include gingival hyperplasia and constipation.<sup>25,26</sup> An ECG must be done before initiation of treatment and monitoring for cardiac arrhythmias, particularly heart block, must be undertaken.<sup>20</sup> Effects of slowing conduction on the atrioventricular node can take up to 10 days to appear and therefore 2-weekly intervals are recommended between dose changes. ECGs should be done before each dose escalation and routinely every 6 months after the drug has been initiated.<sup>27</sup>

Other preventive options include lithium (which is comparable in efficacy to verapamil),<sup>26</sup> topiramate and gabapentin. However, there have been no controlled studies for the latter two medications, and recommendations have been based on case reports only.<sup>8</sup> For patients with shorter episodes of cluster headache, limited courses of oral corticosteroids can also be useful.<sup>8,18,23</sup>

Please refer to *Table 5* for a summary of preventive treatment options.

**ANSWER 7**

Although a correct diagnosis of cluster headache has been made and a treatment regimen has been planned, the chronicity of his symptoms has affected his life to the point that medications alone are unlikely to help his current mental state. His opioid use could also be contributing to a medication-induced depression and, ultimately, also contributing to his headache. The most efficacious course of action at this time would be referral for a period of inpatient treatment for help to wean him off the opioids and to allow him to receive ongoing psychological support during this time.

**REFERENCES**

- Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders. 2nd edition. Cephalalgia 2004;24(suppl 1):9–160.
- Charles A. Advances in the basic and clinical science of migraine. Ann Neurol 2009;65:491–98.
- Cittadini E, Goadsby PJ. Hemicrania continua: a clinical study of 39 patients with diagnostic implications. Brain 2010;133:1973–86.
- Peres MF, Silberstein SD, Nahmias S, et al. Hemicrania continua is not that rare. Neurology 2001;57:948–51.
- Goadsby PJ, Cohen AS, Matharu MS. Trigeminal autonomic cephalalgias: diagnosis and treatment. Curr Neurol Neurosci Rep 2007;7:117–25.
- Newman L, Goadsby PJ. Unusual primary headache disorders. In: Silberstein SD, Lipton RB, Dalessio DJ, editors. Wolff's Headache and Other Head Pain 7th edn. New York: Oxford University Press, 2001:310–21.
- Matharu MS, Goadsby PJ. Trigeminal autonomic cephalalgias. J Neurol Neurosurg Psychiatry 2002;72(suppl II):ii19–26.
- Goadsby PJ, Cittadini E, Burns B, Cohen AS. Trigeminal autonomic cephalalgias: diagnostic and therapeutic developments. Curr Opin Neurol 2008;21:323–30.
- Goadsby PJ. Trigeminal autonomic cephalalgias: fancy term or constructive change to the HIS classification? J Neurol Neurosurg Psychiatry 2005;76:301–05.
- Bahra A, May A, Goadsby PJ. Cluster headache: a prospective clinical study with diagnostic implications. Neurology 2002;58:354–61.
- Schreiber CP, Hutchinson S, Webster CJ, Ames M, Richardson MS, Powers C. Prevalence of migraine in patients with a history of self-reported or physician diagnosed 'sinus' headache. Arch Intern Med 2004;164:1769–72.
- Eross E, Dodick D, Eross M. The Sinus Allergy and Migraine Study (SAMS). Headache 2007;47:213–24.
- Cohen AS, Matharu MS, Goadsby PJ. Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) or cranial autonomic features (SUNA) – a prospective clinical study of SUNT and SUNA. Brain 2006;129:2746–60.
- Goadsby PJ, Lipton RB. A review of paroxysmal hemicranias, SUNCT syndrome and other short-lasting headaches with autonomic features, including new cases. Brain 1997;120:193–209.
- Matharu MS, Boes CJ, Goadsby PJ. Management of trigeminal autonomic cephalalgias and hemicranias continua. Drugs 2003;63:1637–77.
- May A, Leone M, Afra J, et al. EFNS guidelines on the treatment of cluster headache and other trigeminal-autonomic cephalalgias. Eur J Neurol 2006;13:1066–77.
- National Institute for Clinical Excellence (NICE) guidelines. Headaches: diagnosis and management of headaches in young people and adults. NICE clinical guideline 150 [Issued September 2012] Available at [www.nice.org.uk/nicemedia/live/13901/60853/60853.pdf](http://www.nice.org.uk/nicemedia/live/13901/60853/60853.pdf) [Accessed 12 May 2014].
- Rossi S, Editor. Migraine (cluster headache). In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
- Cohen AS, Burns B, Goadsby PJ. High-flow oxygen for treatment of cluster headache: a randomized trial. JAMA 2009;302:2451–57.
- Neurology Expert Group. Headache: cluster headache. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 12 May 2014].
- Rossi S, Editor. Sumatriptan. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
- The Sumatriptan Cluster Headache Study Group. Treatment of acute cluster headache with sumatriptan. N Engl J Med 1991;325:322–26.
- Whyte C, Tepper S. Pearls and Oysters: trigeminal autonomic cephalalgias. Neurology 2010;74: e40–42.
- Australian Government Department of Health. Pharmaceutical Benefits Scheme. Available at [www.pbs.gov.au/medicine/item/8341B](http://www.pbs.gov.au/medicine/item/8341B) [Accessed 21 May 2014].
- Rossi S, Editor. Calcium channel blockers: verapamil. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
- Bussone G, Leone M, Peccarisi C, et al. Double-blind comparison of lithium and verapamil in cluster headache prophylaxis. Headache 1990;30:411–17.
- Cohen AS, Matharu MS, Goadsby PJ. Electrocardiographic abnormalities in patients with cluster headache on verapamil therapy. Neurology 2007;69:668–75.

## Head pain

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 <http://gplearning.racgp.org.au>
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The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office.

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**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

Selena is a lawyer aged 26 years and was diagnosed with migraine without aura last year. She recently moved in with a new partner and has presented today requesting the pill. Which of the following statements regarding the use of oral contraceptives by Selena, given her migraine without aura diagnosis, is CORRECT?

- Combined oral contraceptive pills (COCs) are contraindicated.
- COCs are contraindicated in all women younger than 35 years of age.
- If she smoked, she could still use COCs as COCs are not contraindicated in smokers.
- If Selena were diagnosed with migraine with aura, COCs could be prescribed as they are not contraindicated.
- On the basis of the information given, COCs are not contraindicated for Selena.

### QUESTION 2

With regards to secondary prevention of stroke and current guideline recommendations, which of the following options is the most CORRECT?

- Only patients deemed to be at high risk require management of their risk factors.

- All patients who have had a stroke or TIA require management of all of their risk factors, irrespective of their risk factor level.
- Antiplatelet agents, statins and antihypertensive agents use should be considered as well as lifestyle changes such as optimising diet, increasing physical activity and smoking cessation.
- Answers A and C are correct.
- Answers B and C are correct.

### QUESTION 3

With regard to thunderclap headaches, which of the following statements is the most CORRECT?

- An urgent referral to an emergency department is generally required and CT imaging needs to be performed; additional investigations may also be required.
- Suspected recent thunderclap headache is not a medical emergency.
- Subarachnoid haemorrhage, acute subdural haemorrhage and dissection of cervical arteries are not differential diagnoses for thunderclap headache presentations.
- Up to 2.5% of patients with a thunderclap headache have a subarachnoid haemorrhage.
- Thunderclap headaches do not present as primary headaches.

### QUESTION 4

Which of the following statements is CORRECT with regard to the incidence and management of carotid artery dissection?

- Carotid artery dissection is a common cause of stroke in older people.
- The priority in carotid artery dissection is stroke prevention.
- Randomised controlled trials report similar efficacy for antiplatelet and anticoagulation therapy for stroke prevention in carotid artery dissection.
- Randomised controlled trials report superior efficacy for antiplatelet therapy over anticoagulation therapy for stroke prevention in carotid artery dissection.
- Randomised controlled trials report superior efficacy for anticoagulation therapy over antiplatelet therapy for stroke prevention in carotid artery dissection.

### QUESTION 5

Alice, aged 33 years, is a part-time childcare worker with two children of her own. She comes to see you wanting to discuss her frequent headaches. She has had regular headaches since her teenage years but they have increased in frequency in the past few years. After taking a detailed history, you diagnose chronic migraine without aura. Which of the following statements is the most CORRECT?

- NICE guidelines recommend referral of people with migraine for neuroimaging.

- B. Anxiety and depression are rare comorbidities with chronic migraine.
- C. Chronic migraine is defined as headache occurring on 15 or more days per month for at least 3 months, which has the features of migraine headache on at least 8 days per month.
- D. Migraine risk factors are all modifiable.
- E. Onabotulinum toxin A (or botulinum toxin) is not marketed in Australia for migraine prevention.

**QUESTION 6**

Headaches may be due to primary or secondary causes. Which of the following statements is the most CORRECT with regard to causes of headache?

- A. Thunderclap headaches should raise suspicion of underlying vascular cause(s) and patients with these presentations should be evaluated urgently.
- B. It is unusual for primary headaches to present for the first time in people aged over 50 years.
- C. Postural headaches may suggest low intracranial pressure.
- D. Persistent focal signs presenting with headache suggest a secondary cause for the headache.
- E. All of the above are correct.

**QUESTION 7**

With regard to the pharmacological management of migraine headache which of the following statements is the most CORRECT?

- A. Opioids, including codeine preparations with paracetamol or aspirin, are recommended for the acute treatment of migraine.
- B. The Australian Therapeutic Guidelines recommend medications such as amitriptyline, pizotifen, propranolol, sodium valproate, topiramate and verapamil as migraine prophylactic options.
- C. Migraine prophylactic medications recommended by the Australian Therapeutic Guidelines, such as candesartan and gabapentin, are all licensed and listed on the Pharmaceutical Benefits Scheme for the indication of migraine.
- D. Propranolol, sodium valproate and topiramate are all safe for use in women of childbearing potential.
- E. Triptans should be used to treat all types of migraines.

**QUESTION 8**

Amanda, a speech therapist aged 27 years, has been diagnosed with chronic migraine with aura. With regard to management options, which of the following statements is the most CORRECT?

- A. A headache diary is a useful way of documenting Amanda's response to therapy.
- B. Development of medication overuse headaches is a risk if analgesics are taken on more than 15 days per month.
- C. The goals of migraine prophylaxis are to reduce attack severity, frequency and duration, to improve responsiveness to acute treatments and to reduce disability.

- D. When starting migraine prophylaxis, the patient should be given realistic goals, education about correct dosage and information about potential medication side effects.
- E. All of the above are correct.

**QUESTION 9**

Which one of the following statements regarding cluster headaches is CORRECT?

- A. Oxygen inhalation administered as 100% for 30 minutes is recommended for management of an acute attack of cluster headache.
- B. Subcutaneous sumatriptan is recommended for prevention of cluster headaches.
- C. Sumatriptan nasal spray is recommended for prevention of cluster headaches.
- D. Verapamil is considered first-line for preventive treatment of cluster headache and although not marketed for this indication, prophylaxis of cluster headache is an accepted indication.
- E. Lithium is useful for management of an acute attack of cluster headache.

**REFERENCES**

1. World Health Organization. Family and Reproductive Health Programme. Improving access to quality care in family planning: medical eligibility criteria for contraceptive use, 3rd edn. Geneva: WHO, 2004.
2. ACOG Committee on Practice Bulletins-Gynecology. ACOG practice bulletin. No73: Use of hormonal contraception in women with coexisting medical conditions. *Obstet Gynecol* 2006;107:1453–72.
3. Endocrinology Expert Group. Hormonal contraception: combined hormonal contraception. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2012. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 30 April 2014].
4. National Stroke Foundation. Clinical Guidelines for Stroke Management 2010. Melbourne: National Stroke Foundation. Available at [http://strokefoundation.com.au/site/media/clinical\\_guidelines\\_stroke\\_managment\\_2010\\_interactive.pdf](http://strokefoundation.com.au/site/media/clinical_guidelines_stroke_managment_2010_interactive.pdf) [Accessed 30 April 2014].
5. Ducros A, Bousser MG. Thunderclap headache. *BMJ* 2012;345:1–7.
6. Schwedt TJ. Thunderclap headaches: A focus on etiology and diagnostic evaluation. *Headache* 2013;53:563–69.
7. ACEP Clinical Policies Subcommittees on Acute Headache. Clinical policy: critical issues in the evaluation and management of patients presenting to the emergency department with acute headache. *Ann Emerg Med* 2002;39:108–22.
8. Lyrer P, Engelter S. Antithrombotic drugs for carotid artery dissection. *Cochrane Database Syst Rev* 2010;10:CD000255. doi: 10.1002/14651858.CD000255.pub2.
9. Nedeltchev K, Bickel S, Arnold M, et al. R2-re canalization of spontaneous carotid artery dissection. *Stroke* 2009;40:499–504.
10. Furie KL, Kasner SE, Adams RJ, et al. Guidelines for the Prevention of Stroke in Patients with Stroke or Transient Ischemic Attack: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association. *Stroke* 2011;42:227–76.
11. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edn (beta version). *Cephalalgia* 2013;33:629–808.
12. National Institute for Clinical Excellence (NICE) guidelines. Headaches: Diagnosis and management of headaches in young people and adults. NICE clinical guideline 150 [Issued September 2012] Available at [www.nice.org.uk](http://www.nice.org.uk)

- nice.org.uk/nicemedia/live/13901/60853/60853.pdf [Accessed 5 May 2014].
13. Verrotti A, Agostinelli S, D'Egidio C, et al. Impact of a weight-loss program on migraine in obese adolescents. *Eur J Neurol* 2013;20:394–97
  14. Halker RB, Hastriter EV, Dodick DW. Chronic daily headache: An evidence-based and systematic approach to a challenging problem. *Neurology Clin Pract* 2011;76(suppl 2):S37–43.
  15. Bigal ME, Lipton RB. Excessive acute migraine medication use and migraine progression. *Neurology* 2008;71:1821–28.
  16. Manack A, Buse DC, Serrano D, Turkel CC, Lipton RB. Rates, predictors and consequences of remission from chronic migraine to episodic migraine. *Neurology* 2011;76: 711–18.
  17. Rossi S, Editor. Migraine. In: *Australian Medicines Handbook 2014*. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
  18. Hainer BL, Matheson EM. Approach to acute headache in adults. *Am Fam Physician* 2013;87:682–87.
  19. Neurology Expert Group. Headache: migraine. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd: 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
  20. Neurology Expert Group. Headache: migraine: acute migraine attack: In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 5 May 2014].
  21. Stark RJ, Stark CD. Migraine Prophylaxis. *MJA* 2008;189:283–88.
  22. Drugs and their categories in pregnancy and breastfeeding. In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 6 May 2014].
  23. Rossi S, Editor. Migraine (cluster headache). In: *Australian Medicines Handbook 2014*. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
  24. Neurology Expert Group. Headache: cluster headache: In: eTG (Internet). Melbourne: Therapeutic Guidelines Ltd, 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 12 May 2014].
  25. Rossi S, Editor. Sumatriptan. In: *Australian Medicines Handbook 2014*. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.

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Unit 509 September 2014

## Women's health

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## Women's health

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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

## ABOUT THIS ACTIVITY

Social/cultural roles and expectations of women, as well as biological factors, can affect women's health and wellbeing.<sup>1</sup> Issues of particular concern for women's physical and mental health include perinatal problems and disorders arising from hormonal imbalances. Violence against women, a major human rights abuse that may lead to physical, mental, sexual, reproductive health and other health problems,<sup>2</sup> is also a key area of concern. Worldwide, about 35% of women have experienced intimate partner violence or non-partner sexual violence in their lifetime and about 38% of murdered women are killed by an intimate partner.<sup>2</sup> In Australia, one in three women has been exposed to physical violence, and one in five women aged over 15 years has experienced sexual violence.<sup>3</sup>

This edition of check will consider scenarios of relevance to the management of women's health in general practice.

## LEARNING OUTCOMES

At the end of this activity, participants will be able to:

- outline the management options for women with perinatal depression
- list strategies for the management of vaginismus
- summarise the diagnosis and management of polycystic ovary syndrome
- discuss useful strategies for managing women suspected of experiencing domestic violence
- describe the medical management of a woman who has been raped.

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## REFERENCES

1. World Health Organisation. Why gender and health. Geneva: WHO. Available at [www.who.int/gender/genderandhealth/en/](http://www.who.int/gender/genderandhealth/en/) [Accessed 4 August 2014].
2. World Health Organisation. Violence against women: intimate partner and sexual violence against women. Fact sheet No 239. Geneva: WHO; 2013. Available at [www.who.int/mediacentre/factsheets/fs239/en/](http://www.who.int/mediacentre/factsheets/fs239/en/) [Accessed 4 August 2014].
3. Australian Department of Social Services. The National Plan to Reduce Violence against Women and their Children 2010–2022 (the National Plan). Canberra: Australian Department of Social Services, 2011. Available at [www.dss.gov.au/our-responsibilities/women/programs-services/reducing-violence/the-national-plan-to-reduce-violence-against-women-and-their-children](http://www.dss.gov.au/our-responsibilities/women/programs-services/reducing-violence/the-national-plan-to-reduce-violence-against-women-and-their-children) [Accessed 2 June 2013].

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

BMI	body mass index	HBV	hepatitis B virus	PCOS	polycystic ovary syndrome
CAH	congenital adrenal hyperplasia	β-hCG	β-human chorionic gonadotropin	PCR	polymerase chain reaction
CBT	cognitive behaviour therapy	HCV	hepatitis C virus	SHBG	sex hormone-binding globulin
COC	combined oral contraceptive	HDL-C	high-density lipoprotein cholesterol	SSRI	selective serotonin reuptake inhibitor
DSM-5	Diagnostic and Statistical Manual of Mental Disorders 5th edition	HIV	human immunodeficiency virus	STI	sexually transmissible infection
EPDS	Edinburgh Postnatal Depression Scale	LDL-C	low-density lipoprotein cholesterol	T2DM	type 2 diabetes mellitus
FAI	free androgen index	OGTT	oral glucose tolerance test	TSH	thyroid stimulating hormone
FSH	follicle stimulating hormone	PANDA	Post and Antenatal Depression Association	VVS	vaginal vestibulitis syndrome
GDM	gestational diabetes mellitus				

**CASE 1**

**JENNY IS FINDING BEING A NEW PARENT DIFFICULT**

Jenny is a primary school teacher aged 38 years. She is married and has a daughter, Chloe, aged 2 months. She has been seeing you regularly for PAP smears and contraception, and twice for minor issues during her recent first pregnancy. She presents today with Chloe. You know that Jenny has no past history of any serious illness. Her mother had depression throughout Jenny's childhood years and her grandmother had bipolar disorder. Jenny says Chloe is not sleeping and feeds 'constantly'. She is fully breastfeeding and aspires to attachment parenting (ie understanding and responding to Chloe's emotional and physical needs and building a strong relationship with Chloe). Chloe's chart from the health centre shows she is on the 35th percentile for height and weight and has been since birth. Jenny looks tired, despite having makeup on, but says she is doing fine and that Chloe's routine is the problem.

**QUESTION 1**  

What issues need exploration in this consultation?

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**FURTHER INFORMATION**

Jenny denies being depressed but admits that her husband says she is irritable. He works long hours and doesn't provide much help. Jenny has cried on most days in the past month and says she is sleeping no more than 4 hours per night and cannot get back to sleep after Chloe wakes her. Her concentration is low and she is not enjoying anything. She says, 'motherhood is nothing like I imagined'. She worries that Chloe has allergies and has been searching the internet for causes of crying in babies. Jenny and Chloe's physical examinations are normal and Jenny denies any thoughts of harm or excessive fears. She is agreeable to your suggestion of having her mother stay with her to care for Chloe overnight so she can sleep.

At her next appointment 3 days later, which her husband does not attend because he is too busy at work, Jenny is still not sleeping, although she had an extra hour of sleep when her mother stayed over, and she feels she cannot cope. She starts crying in the consultation.

**QUESTION 2** 

What is the most likely diagnosis? What are your treatment options?

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**FURTHER INFORMATION**

Jenny agrees to see a psychologist for cognitive behaviour therapy (CBT) and her mother has agreed to look after Chloe on 3 days a week so Jenny can rest and go to yoga. When she sees you 2 weeks later, Jenny has had two CBT sessions and seems more anxious. Sleep remains a problem. She admits to having intrusive thoughts more frequently; for example, she fears that something will happen to Chloe and that it will be her fault. On closer questioning she fears she will put Chloe in the microwave by mistake or drop her in the bath. These thoughts horrify her and although she does not believe she would ever act on these thoughts, she thinks she must be the 'worst ever' mother for having them. She is getting her husband to bath the baby and has disconnected the microwave.

**QUESTION 3**  

How are you going to manage Jenny?

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**FURTHER INFORMATION**

You prescribe sertraline, up to 200 mg, after discussion with the psychiatrist to whom she has been referred. Jenny's condition improves significantly after 3 months. She is now sleeping for 6 hours per night on most nights and only occasionally has intrusive thoughts. She takes 200 mg sertraline daily. She has weaned Chloe. She has seen the psychiatrist three times in the past 2 weeks ago and has an appointment in 2 weeks. She has stopped seeing the psychologist but says she finds some of the techniques helpful. She tells you that she still does not enjoy motherhood and is thinking about going back to work. She fusses over Chloe throughout the consultation and the child is irritable. Jenny tells you Chloe is 'always like that' and at playgroups Chloe never leaves Jenny's side. She says 'it's exhausting'.

**QUESTION 4** 

Is there anything else you need to do at this time?

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**FURTHER INFORMATION**

Jenny is now 18 months postpartum and has been back to her normal self for 6 months. She never attended any extra therapy and now that Chloe is in a routine she feels everything is fine. She is working as an emergency teacher and now wants to stop her medication and have another child.

**QUESTION 5** 

What are your recommendations?

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**CASE 1 ANSWERS**

**ANSWER 1**

Chloe is the presenting issue; however, given Jenny's family history and her tired appearance there is a potential underlying issue with Jenny, although she has hinted that she is reluctant to accept that she might have any problems.<sup>1</sup> Examine Chloe first and rule out any underlying physical disorder, noting how well she is doing. At the same time, tell Jenny that the first few months of motherhood can be tough and that most women find it difficult (ie normalise). Ask Jenny if she has any breaks, how much sleep she is actually getting and if there is anyone who can care for Chloe overnight to give her a break. In particular, ask what role her husband is having and, if not much, whether he could be involved more. Suggest that he comes to the next appointment. You could also ask Jenny if her mother is able to help and whether she has other support networks, particularly other women with babies or young children. Consider any lifestyle issues, such as diet, drugs and alcohol, that might be contributing to her tiredness and suggest that some gentle exercise might help (eg walking with the pram).

You should ask if she has completed an Edinburgh Postnatal Depression Scale (EPDS)<sup>2</sup> with her child health nurse. If she has, you could ask her about her score. A score of 10 or more is suggestive of possible depression.<sup>2</sup> It is important to say how common it is for women to feel a bit down after the birth of a child and to ask Jenny how she is feeling. You could reassure Jenny that feeling tired is normal.

Jenny is still in the high-risk time frame for depression and postpartum psychosis,<sup>3</sup> both of which can be exacerbated by sleep deficit. You need to complete a psychiatric history, concentrating on depressive symptoms, psychotic symptoms, particularly related to whether her ideas about Chloe are realistic or if she has excessive concerns about her child's health, as well as undertaking a risk assessment with respect to suicide and infanticide (including neglect and distraction through tiredness).

Checking for physical issues such as mastitis, urinary tract infection and thyroid dysfunction may also be warranted.

**ANSWER 2**

If Jenny's physical tests are normal, she most probably has postnatal depression, which, according to the *Diagnostic and Statistical Manual of Mental Disorders* 5th edition (DSM-5),<sup>4</sup> may be an adjustment disorder or major depression. Psychotic depression, postpartum psychosis or a variation of bipolar disorder are diagnoses that need to be considered given her high level of anxiety, sleeplessness and her family history. The latter needs to be clarified. Her risk for major depression includes her family history, having a first baby and being an older mother.<sup>3,5</sup> She has already given you indications that she feels she has failed, that her baby is not perfect and that motherhood is not what she expected.

Reiterate that mood problems are common in the postpartum period. For example, a *beyondblue* postnatal depression screening program reported that 16% of women have depression in the postnatal period and many more have high scores on the EPDS due to adjustment difficulties.<sup>5</sup> Tell Jenny that women improve with time and treatment. Rebalancing expectations will be an ongoing part of any consultation.

Outline treatment options, starting with non-medication strategies. For example, suggest taking breaks from Chloe, perhaps by asking her mother to babysit on a regular basis or, alternatively, using paid childcare. This could be discussed with the reflection that 'it takes a village to raise a child'. Address any other lifestyle issues identified as possible contributing factors. Another option is cognitive behavioural therapy (CBT) with a psychologist or in a group setting (often run at child health centres), which may be supportive and/or therapeutic.<sup>6</sup> CBT is one of several psychological therapies reported to have moderate-quality evidence supporting improved symptoms in postnatal depression. Other options include interpersonal psychotherapy and psychodynamic therapy.<sup>7</sup> Recommend useful, vetted websites (see Resources for patients) and discourage the use of other websites. Ideally, provide Jenny with some fact sheets, for example, from *beyondblue* and the Post and Antenatal Depression Association (PANDA), and the PANDA hotline. With Jenny's permission, contact her husband if he is unable to attend an appointment and look at ways he may be able to help.

Introduce the idea of medication as another treatment possibility. Use of temazepam for a few days could be useful. Jenny is likely to be worried about addiction and transfer of medication to the baby via her breast milk; this is minimal with 10–20 mg temazepam and for 2–3 days only. Given that her current history has now persisted for more than 1 month, you could suggest a selective serotonin reuptake inhibitor (SSRI) such as sertraline, which is reported to be present in low concentrations in breast milk, has little transfer to the infant and poses few risks of side effects in the child.<sup>8</sup> The *Australian Medicines Handbook* 2014 indicates that some people consider sertraline to be one of the preferred antidepressants for use when breastfeeding<sup>9</sup> and suggests using it as an alternative to fluoxetine, which has a long half-life.<sup>10</sup> In general, SSRIs (Australian pregnancy category C), with the exception of paroxetine, are considered relatively safe for use in pregnancy and the postnatal period; paroxetine (Australian pregnancy category D) should be avoided in women of child-bearing potential.<sup>9,11</sup> Warn Jenny that the medication could make her more agitated but it will not sedate her, so she will still be able to care for Chloe. If she has bipolar disorder there is a risk that an antidepressant could cause mania if she is not on a mood stabiliser.<sup>12</sup>

### ANSWER 3

A referral to a perinatal psychiatrist is now critical and if an appointment cannot be obtained urgently then the GP should ring and get telephone advice regarding medication. An antidepressant is now likely to be effective if it has not already begun as Jenny may now have a major depressive disorder with obsessional thoughts. Obsessive-compulsive disorder is a possible differential diagnosis.<sup>13</sup> Psychosis is less likely given the intrusive thoughts are ego-dystonic

(distressing, unacceptable) and that she avoids any possibility of doing them. Given the family history, bipolar disorder is a possibility but, statistically, it is more likely in the first 3 months (particularly in the first week).<sup>14</sup>

If Jenny's anxiety continues to be a major feature (and it is often the last symptom to improve) then continuing CBT will be important; exercise and yoga might also help. She may require a higher dose of the SSRI. Often, anxious women struggle with breastfeeding and having enough milk, which in turn causes the child to be more unsettled so weaning or a supplementary night feed may be an option worth discussing with Jenny, particularly if infant weight gain or breastfeeding has been an issue for her.

If Jenny's symptoms worsen or do not begin to improve, consider a referral to a mother–baby unit if this is available.

### ANSWER 4

It seems that Jenny's depression is resolving but her lack of enjoyment may still be a feature of her illness. It may also be part of the driving stress; that is, there is an underlying attachment difficulty and Chloe's birth and their relationship have rekindled her own early attachment difficulties when her mother had depression and was emotionally unavailable during her childhood. Ask Jenny how depressed her mother was (eg was she hospitalised?) and about their relationship, particularly when Jenny was little; did she feel she could go to her mother for comfort and did her mother help her to become independent and allow her to try things on her own? Are Jenny's attachment problems with her mother being repeated with Chloe? If so, or if this lack of enjoyment persists, consider discussing with her psychiatrist about sending Jenny to a perinatal psychiatrist, mother–infant therapist or mother–infant therapy group (eg Circle of Security).<sup>15</sup>

### ANSWER 5

Jenny is at a higher risk of having another depressive episode postpartum, compared with someone who has never had postpartum depression, and her risk of having another depressive episode will be higher still (and possibly occur earlier) if she ceases her medication.<sup>16</sup> Ideally, she should be well for 12 months before ceasing her medication, but as she has been taking it for this long, and given her age, she may not want to wait any longer before trying for another baby. Planning the next pregnancy with a perinatal psychiatrist is essential.

### Table 1. Recommendations for making treatment choices for women with mental health issues who are planning a pregnancy, are pregnant or are breast feeding<sup>7,17</sup>

Health professionals should:

- choose medications with lower risk profiles for both the mother and the fetus/infant
- initiate medication at low dose and titrate slowly to the lowest effective dose (to minimise dose-related risks)
- use monotherapy in preference to combination treatment
- consider additional precautions for preterm, low birth weight or sick infants

The risks and benefits of ceasing or continuing medication need to be outlined and discussed, ideally with both Jenny and her husband. Current evidence indicates that an SSRI such as sertraline (avoid paroxetine and fluoxetine, although any increase in risk from them is small) has low risk for malformation and pulmonary hypertension of the newborn; registry studies have significant confounding variables such as higher alcohol and multiple drug use in women who take antidepressants.<sup>8,18,19</sup> As a comparison, the risk of birth defects in the general population is 2–4%.<sup>17</sup> There is a risk of slight prematurity (and possibly miscarriage), neurobehavioural delays and possibly autism but this also exists to a lesser extent in women with depression and anxiety.<sup>18,20</sup> Women who are depressed and anxious in pregnancy (and not taking SSRIs) have infants born with higher cortisol levels, and may be more likely to be at risk of later behavioural, mood and anxiety disorders, as well as poor antenatal care and suicide.<sup>21,22</sup>

If Jenny chooses to cease her medication, she should do so as slowly as possible (ideally over 6 months) and she should be monitored for symptoms of discontinuation and/or withdrawal, and signs of relapse.<sup>17</sup> Taking a reduced dose might be an option, and discussion regarding how to manage a relapse is important in advance. A refresher course of CBT and discussion of general measures, such as keeping Jenny's stress levels low, having regular sleep and engaging in regular exercise, are important regardless of whether she decides to stay on her medication or ceases using it.

## REFERENCES

1. Bilzta J, Ericksen J, Buist A. Women's experience of postnatal depression – beliefs and attitudes as barriers to care. *Aust J Advan Nurs* 2010;27:44–54.
2. Cox J, Holden J, Sagovsky R. Detection of postnatal depression: development of the 10-item Edinburgh Postnatal Depression Scale. *Brit J Psych* 1987;150:782–86.
3. O'Hara M, Swain A. Rates and risks of postpartum depression – a meta-analysis. *Int Rev Psychiatry* 1996;8:37–54.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, 5th edn (DSM-5)*. Arlington: American Psychiatric Publishing, 2013.
5. Buist AE, Austin MP, Hayes BA, et al. Postnatal mental health of women giving birth in Australia 2002–2004: findings from the *beyondblue* National Postnatal Depression Program. *Aust N Z J Psychiatry* 2008;42:66–73.
6. Goodman J, Santangelo G. Group treatment of postpartum depression: a systematic review. *Arch Womens Ment Health* 2011;14:277–93.
7. National Institute for Health and Care Excellence. Antenatal and postnatal mental health. The NICE Guideline on Clinical Management and Service Guidance. Manchester: NICE, 2007. Available at [www.nice.org.uk/guidance/CG45](http://www.nice.org.uk/guidance/CG45) [Accessed 27 June 2014].
8. Jain AE, Lacy T. Psychotropic drugs in pregnancy and lactation. *J Psychiatr Pract* 2005;11:177–91.
9. Rossi E, editor. Sertraline (special cases: pregnancy and the postnatal period). In: *Australian Medicines Handbook* 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
10. Rossi E, editor. Fluoxetine (breastfeeding). In: *Australian Medicines Handbook* 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
11. Rossi E, editor. Major depression (special cases: pregnancy and the postnatal period). In: *Australian Medicines Handbook* 2014. Adelaide: Australian Medicines Handbook Pty Ltd, 2014.
12. Ghaemi SN, Rosenquist KJ, Ko JY, et al. Antidepressant treatment in bipolar versus unipolar depression. *Am J Psychiatry* 2004;161:163–65.
13. McGuinness M, Blissett J, Jones C. OCD in the perinatal period: is postpartum OCD a distinct subtype? A review of the literature. *Behav Cogn Psychoth* 2011;39:285–310.
14. Kendall R, Chalmers J, Platz C. Epidemiology of puerperal psychosis. *Br J Psychiatry* 1987;150:662–73.
15. Marvin R, Cooper G, Hoffman K, Powell B. The Circle of Security Project: attachment-based intervention with caregivers-pre-school child dyads. *Attach Hum Dev* 2002;4:107–24.
16. Cohen LS, Altshuler LL, Harlow BL, et al. Relapse of Major Depression during pregnancy in women who maintain or discontinue antidepressant treatment. *JAMA* 2006;295:499–507.
17. *beyondblue*. Clinical practice guidelines for depression and related disorders – anxiety, bipolar disorder and puerperal psychosis – in the perinatal period. A guideline for primary care health professionals. Melbourne: *beyondblue*, 2011. Available at [www.bspg.com.au/dam/bsg/product?client=BEYONDBLUE&prodid=BL/0891&type=file](http://www.bspg.com.au/dam/bsg/product?client=BEYONDBLUE&prodid=BL/0891&type=file) [Accessed 27 June 2014].
18. Udechuku A, Nguyen T, Hill R, Szego K. Antidepressants in pregnancy: a systematic review. *Aust N Z J Psychiatry* 2010;4:978–96.
19. Buist A. Perinatal mental health: identifying problems and managing medications. *Aust Fam Physician* 2014;43:182–85.
20. Galbally M, Lewis AJ, Buist AE. Developmental outcomes of children exposed to antidepressants in pregnancy. *Aust N Z J Psychiatry* 2011;45:393–99.
21. O'Connor T, Heron J, Glover V, et al. Antenatal anxiety predicts child behavioural/emotional problems independently of postnatal depression. *J Am Acad Child Adolesc Psychiatry* 2002;41:1470–77.
22. Bonari L, Koren G, Einarson TR, Jasper JD, Taddio A, Einarson A. Use of antidepressants by pregnant women: evaluation of perception of risk, efficacy of evidence-based counselling and determinants of decision making. *Arch Womens Health* 2005;8:214–20.

## RESOURCES FOR PATIENTS

- PANDA, 1300 726 306, [www.panda.org.au](http://www.panda.org.au)
- *beyondblue*, 1300 224 636, [www.beyondblue.org.au](http://www.beyondblue.org.au)
- The Royal Women's Hospital Perinatal Psychotropic Information Centre, 03 8345 3190, [www.ppmis.org.au](http://www.ppmis.org.au)

## RESOURCES FOR DOCTORS

- The Royal Women's Hospital Perinatal Psychotropic Information Centre 0383453190, [www.ppmis.org.au](http://www.ppmis.org.au)
- *beyondblue*, 1300 224 636, [www.beyondblue.org.au](http://www.beyondblue.org.au)

**CASE 2**

**CHERYL IS FRIGHTENED OF HER HUSBAND**

Cheryl, 37 years of age, has made an urgent appointment and has specifically asked to see you. She looks white and drawn. She tells you that her husband Bob was particularly nasty and demeaning in an argument with her last night. When he came to bed she was exhausted and did not want to have sex but he was insistent. Cheryl was frightened and says she feels 'If I had said no, he would have taken it anyway.'

**QUESTION 1**  

How would you respond to Cheryl?

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**FURTHER INFORMATION**

You ask Cheryl to tell you more about her relationship with Bob. She says there has been 'trouble' in the past. When invited to say more, Cheryl tells you that Bob has episodically 'lost his cool'. It started when she was pregnant with their daughter Kate. On one occasion Bob beat her and choked her. In the past year Bob has hit out at Cheryl again, knocking her against the door and/or floor. Bob is usually very sorry after it happens. She then tells you Bob is a good father and that Kate loves her dad and is happy in her school.

**QUESTION 2** 

Is Cheryl experiencing domestic violence? How prevalent is domestic violence?

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**QUESTION 3**   

What should be documented during Cheryl's visit? Why is it important to make notes of this visit?

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**FURTHER INFORMATION**

Cheryl says she is concerned about who could access the information if it were to be recorded in her medical notes. She is worried and very embarrassed at the thought of other staff reading it when she comes back to the practice for routine medical matters.

**QUESTION 4**   

How can you protect a patient's medical records?

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**QUESTION 5** 

What are the most immediate concerns at this time? What important facts would you want to obtain from a further history?

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**FURTHER INFORMATION**

Cheryl says she wasn't aware that she has the right to refuse sex with her husband.

**QUESTION 6**  

How would you respond to Cheryl's comment?

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**QUESTION 7**   

When might you need to break the doctor–patient confidentiality?

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**FURTHER INFORMATION**

Cheryl says she really does not want to leave Bob. She does not want to break up the family, cause any distress for Kate or sell the house, but she now acknowledges she may have to leave and that all of this may indeed happen. Cheryl wants to consider her options.

**QUESTION 8** 

How can you arrange a helpful referral for Cheryl?

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**CASE 2 ANSWERS**

**ANSWER 1**

Careful thought needs to be given to the response and approach to questioning in a situation such as this where a patient has indicated possible intimate partner abuse. GPs may lack confidence in discussing issues related to domestic violence and/or perceive that they cannot help abused women.<sup>1</sup> However, studies have shown that patients want to be asked to discuss abuse and are likely to disclose abuse to their GP, particularly if asked directly and in a sensitive, empathic way.<sup>2</sup> The Royal Australian College of General Practitioners (RACGP) publication, *Abuse and violence: working with our patients in general practice*<sup>2</sup> (the white book), provides guidance about inquiry and disclosure of abuse.

Cheryl has disclosed what seems to be an abusive situation. A helpful response incorporates the following two steps from the 'nine Rs' for intervention described in the RACGP white book:<sup>2</sup>

- recognising symptoms of abuse and violence, and asking directly and sensitively
- responding to disclosures of violence with empathic listening and exploration.

It is important to acknowledge Cheryl's fears; you could respond by saying that what happened sounds very frightening for her but that she has been brave in disclosing her experience to you.

Examples of other questions or statements, which are open and likely to allow Cheryl to give a fuller history include the following:

- I am hearing you say you were frightened of what your husband would do if you did not have sex with him.
- Can you tell me more about your relationship with your partner?
- Has anything like this happened before?

The RACGP white book also suggests following up with more specific questions and statements:<sup>2</sup>

- Has your partner ever physically threatened or hurt you?
- Is there a lot of tension in your relationship? How did you resolve the argument?
- Sometimes partners react strongly in arguments and use physical force. Is this happening to you?
- Have you ever felt unsafe in the past?
- Violence is very common in the home. I ask a lot of my patients about abuse because no one should have to live in fear of their partners.

Responses that are **not** helpful include:

- What were you arguing about?

This question minimises a serious situation. It is normal for couples to argue but it is not normal or OK to feel frightened, or to be compelled to have sex. Cheryl may hear this question as blaming her, with the implication that if she had not argued the incident would not have happened.

- I don't think it is fair to talk about Bob like that. He didn't do anything to you, did he?
- You need to leave him right now.

These responses miss the point that Cheryl may be describing a situation where she is frightened of her partner and may be describing rape in marriage. Criticising her husband or telling her to leave him may provoke Cheryl to defend her husband. Family violence is disempowering.<sup>3</sup> Cheryl will benefit from support in gaining a clearer perspective and making her own decisions.

In studies assessing women's views of encounters with clinicians, consistent themes have emerged about their expectations of how GPs should respond to issues of intimate partner violence.<sup>2</sup> The main expectations are that GPs should have an understanding of the issue, be alert to signs of abuse and raise the issue in a supportive, compassionate and non-threatening way. A non-judgemental but confident approach, with assurances that confidentiality will be maintained, is essential.<sup>2</sup>

### ANSWER 2

Domestic violence is not limited to physical abuse and can include emotional, sexual, economic and social abuse, and neglect.<sup>2</sup> It can happen in any social group. The RACGP white book<sup>2</sup> provides excellent information and guidance about this subject for the GP.

From Cheryl's description of Bob's behaviour towards her, it is clear that she has experienced physical abuse. His insistence on having sex when she did not want it is sexual abuse. The demeaning nature of their argument the night before her visit to you indicates that she may also be subjected to emotional abuse.

The Australian Bureau of Statistics Personal Safety 2012 survey<sup>4</sup> found that 17% of women aged 18 years and over had experienced violence by a partner; 1.5% of women in a relationship had experienced physical violence in the previous 12 months and 25% had experienced emotional violence. The violence can continue over many years. In 20% of cases the violence commences during pregnancy. Family violence often continues for many years.

### ANSWER 3

It is important to accurately document information that Cheryl gives you at this visit, including any health complaints and symptoms, as you would at any other visit. You could also record Cheryl's descriptions of the events using quotation marks. You should document in detail your observations of Cheryl's condition, behaviour and any injuries she may have, as well a history of how the injuries were sustained.<sup>2</sup>

This situation is directly relevant to Cheryl's mental and physical health, and potentially her life and safety; it is important, therefore, to make detailed notes of the discussions.<sup>2</sup>

It is also important for Cheryl that there be a record of the alleged violence in case of any legal issues that may arise in the future. For example, Cheryl and Bob may need to separate and there may be family court orders about childcare or other legal disputes. It is invaluable for the patient to have a contemporaneous record.<sup>2</sup>

### ANSWER 4

This is a difficult issue. Some practice software allows a patient's notes to be restricted to one-doctor access. However, this would not meet Cheryl's needs as she sees other doctors in the practice from time to time. One suggestion is to write a paper medical note and scan it into the record under a title such as 'special note confidential between patient and doctor (*insert your name*)' with the date. It is worth discussing this issue at a practice meeting and confirming the suitability of any innovative arrangements with your medical defence organisation.

### ANSWER 5

Immediate concerns are assessing Cheryl's risk and safety. This should be informed by your professional judgement of Cheryl's situation, Cheryl's assessment of her risk and safety, and the presence of risk indicators such as Bob's history of violent behaviour, mental health problems, access to lethal weapons, and use of drugs and alcohol.<sup>2</sup>

You could ask the following questions to assess her safety:<sup>2</sup>

- Does Cheryl feel safe to go home? If not, you should help her to find somewhere safe where she can go. If yes, then help her to develop a safety plan (see below).<sup>2</sup>
- How safe does she feel?
- What does she need to feel safe?
- How safe is Kate?
- Is Bob obsessive about Cheryl?
- Is Bob controlling or abusive in other areas (eg financial, emotional, her relationships with friends and families, isolating her)?
- Is there a weapon in the house?
- Has Bob threatened Cheryl with a weapon?
- What does she mean by 'I can't go on like this'?
- What resources does Cheryl have to call for help or move to a place of safety (eg neighbours, family and money)?

Some women may be followed home from work or become trapped at home with the telephone disconnected and without access to money, car keys, Medicare and ATM cards or other basic means to leave. It is important to help Cheryl to consider her situation and, if necessary, to plan to be able to leave. An example of safety information for women is shown in *Table 1*.

Useful information to obtain from a further history could include:

- whether the violence is escalating now
- additional history about the choking, smothering or strangulation, which is a significant risk factor for future homicide and may often leave no visible marks or injuries<sup>5,6</sup>
- history of any past attempts to leave
- history of prior abuse in other relationships (childhood abuse is a risk factor for being a victim of abuse as an adult<sup>7,8</sup>).

**Table 1. Safety planning<sup>9</sup>**

Safety during an abusive incident	Safety at work
<ul style="list-style-type: none"> <li>• Be aware of all exit routes and safety spots</li> <li>• Have a plan that includes how to:                             <ul style="list-style-type: none"> <li>– call emergency (000)</li> <li>– safely exit the house</li> <li>– seek help</li> </ul> </li> <li>• Ask neighbours to call the police if they hear any disturbance</li> </ul>	<ul style="list-style-type: none"> <li>• Tell someone at work about the situation at home</li> <li>• Avoid using the same route each day to go to work</li> <li>• Have a plan for arriving at and leaving work</li> </ul>

**ANSWER 6**

In all Australian jurisdictions rape in marriage is a crime. In this case, however, Cheryl explains that Bob did not specifically threaten her about the sex and she cannot be sure of what would have happened if she had refused to have sex. Cheryl does not think what took place was rape but it was the trigger that has caused her to seek your help. This case illustrates the important concept that where there is violence with fear and control it is not possible to freely consent to sexual intimacy.

**ANSWER 7**

The laws for reporting domestic violence vary in different states. However, it may be necessary to break doctor–patient confidentiality under the following circumstances when dealing with cases of domestic violence:<sup>2</sup>

- You believe that the patient is in imminent danger.  
Ideally, you should seek the patient's consent to report the matter to the police; however, if they are unable to give consent (eg they are cognitively impaired) or if they have been threatened with weapons such as guns and knives, the patient's safety overrides doctor–patient confidentiality. The NSW Department of Health recommends notifying the police if the patient has serious injuries. In the Northern Territory reporting to the police is mandatory if a person has been, or is likely to be, seriously physically harmed from family violence.
- There are children involved  
If there is any risk to a child, whether direct or indirect (eg the child witnesses the abuse of a parent), the situation must be reported to the child protection authorities. Northern Territory law also requires all adults to report to the police if a child is likely to be at risk of any type of harm, including sexual offences.

If there is any doubt about the need to break doctor–patient confidentiality, it is advisable to consult your medical defence organisation.<sup>2</sup>

**ANSWER 8**

There are domestic violence support services in every state/territory. The following resources could be helpful for Cheryl:

- Access to a list of local counselling services and shelters with expertise in managing domestic violence is very helpful. Each

Medicare Local and/or practice can ensure that the list is kept up to date.

- The telephone service 1800RESPECT offers 24/7 phone counselling. The 1800RESPECT website has online counselling and information for victims, professionals, and friends and family.
- Local police or emergency services (000) should be contacted if Cheryl and/or Kate are in danger. In some states there are specialist police domestic violence liaison officers. The police can provide information on court orders to restrain or apprehend violent offenders. These orders can exclude a violent person from a home or another location but do not necessarily mean that the couple cannot continue to live together.
- In some states there are programs, such as *Staying home leaving violence*,<sup>10</sup> where the victim and the children stay in their home. The perpetrator is mandated to leave the house and the locks are changed.

Any past history of violence or sexual abuse is relevant and should be addressed when Cheryl is ready. Domestic violence or counselling services can assist with this if and when Cheryl is ready to be referred to the appropriate health professional.

Cheryl may need to be very careful with any material she takes home as it may trigger further abuse if her husband finds it.

**SUMMARY**

The above discussion illustrates the following nine steps to intervention (the nine Rs), recommended by the RACGP white book:

1. Role with patients who are experiencing abuse and violence
2. Readiness to be open to
3. Recognise symptoms of abuse and violence, ask directly and sensitively and
4. Respond to disclosures of violence with empathic listening and explore
5. Risk and safety issues
6. Review the patient for follow-up and support
7. Refer appropriately and also
8. Reflect on our own attitudes and management of abuse and violence
9. Respect for our patients, our colleagues and ourselves is an overarching principle of this sensitive work.

**CONCLUSION**

Cheryl decides that she and Kate will stay with her sister for a couple of days so she can think about what she should do. She agrees to phone 1800RESPECT from her sister's house today. She thanks you for your help. She tells you this is the most she has ever told anyone about the violence. You again acknowledge her bravery. You offer to continue to see her and make a follow-up appointment.

## REFERENCES

1. Hegarty K, Taft A. Overcoming the barriers to disclosure and inquiry of partner abuse for women attending general practice. *Aust N Z J Public Health* 2001;25:443–47.
2. Royal Australian College of General Practitioners. Abuse and violence. Working with our patients in general practice. 4th edn. Melbourne: RACGP; 2014. Available at [www.racgp.org.au/your-practice/guidelines/whitebook/](http://www.racgp.org.au/your-practice/guidelines/whitebook/) [Accessed 27 June 2014].
3. McLennan W. Women's safety in Australia. Canberra: Commonwealth of Australia; 1996. Available at [www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/F16680629C465E03CA256980007C4A81/\\$File/41280\\_1996.pdf](http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/F16680629C465E03CA256980007C4A81/$File/41280_1996.pdf) [Accessed 27 June 2014].
4. Australian Bureau of Statistics. Personal safety. Measuring the prevalence of violence. Canberra: Commonwealth of Australia; 2012. Available at [www.abs.gov.au/ausstats/abs@.nsf/Lookup/4906.0Chapter2002012](http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/4906.0Chapter2002012) [Accessed 11 July 2014].
5. Block CR, Devitt CO, Fonda D, et al. (2000). The Chicago Women's Health Study: risk of serious injury or death in intimate violence: a collaborative research project. Washington, DC: US Department of Justice; 2000.
6. Evelyn Jacobs Ortner Unity Center on Family Violence. Fact sheet: Strangulation assaults in domestic violence cases. Philadelphia: Evelyn Jacobs Ortner Unity Center on Family Violence; 2009. Available at [www.sp2.upenn.edu/ortner/docs/factsheet\\_strangulation.pdf](http://www.sp2.upenn.edu/ortner/docs/factsheet_strangulation.pdf) [Accessed 30 July 2014].
7. Bensley L, van Eenwyk J, Simmons KW. Childhood family violence history and women's risk for intimate partner family violence and poor health. *Am J Prevent Med* 2003;25:38–44.
8. Classen CC, Palesh OG, Aggarwal R. Sexual revictimization: a review of the empirical literature. *Trauma Violence Abuse* 2005;6:103–29.
9. Wimlah Specialist Domestic Violence Service. Charmed and dangerous. A woman's guide to reclaiming a healthy relationship. Available at [www.wimlah.org.au/wp-content/uploads/2013/03/CharmedDangerous\\_wimlah-draft3-1.pdf](http://www.wimlah.org.au/wp-content/uploads/2013/03/CharmedDangerous_wimlah-draft3-1.pdf) [Accessed 30 July 2014].
10. NSW Government Family and Community Services. Staying home leaving violence. Available at [www.community.nsw.gov.au/docs\\_menu/for\\_agencies\\_that\\_work\\_with\\_us/our\\_funding\\_programs/shlv.html](http://www.community.nsw.gov.au/docs_menu/for_agencies_that_work_with_us/our_funding_programs/shlv.html) [Accessed 22 July 2014].

**CASE 3**

**JACKIE FEARS SHE WAS RAPED**

Jackie, aged 24 years, comes to see you late on Saturday morning, teary and distressed. She last saw you 9 months ago for a Pap smear. She is in good health. She tells you that she thinks she was drugged the night before and woke up with a man on top of her who was having sex with her. She is worried she may catch something.

**QUESTION 1**  

What is the most strategic approach for managing this consultation?

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**QUESTION 2**  

What messages are therapeutic for someone disclosing rape?

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**QUESTION 3** 

What are some common emotional responses to rape?

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**FURTHER INFORMATION**

Following discussion, Jackie provides a more detailed history. Jackie has been in a new job at an advertising agency for 2 weeks. On Friday night Jackie and her colleagues had a few drinks after work and then some colleagues invited her to go with them to a party. She felt a bit tipsy in the taxi. At the party, she had one more drink and then felt dizzy and unwell. She stumbled and a man helped her up the stairs and into a bedroom to lie down. She woke up with a man on top of her, having vaginal sex with her. She felt weak and sick and tried to tell him to go away and that she did not want to do this. He did not stop. After he left, she slowly recovered, found her clothes and got dressed. She phoned her sister who came and picked her up and brought her to your clinic this morning.

You are concerned that this consultation should ideally take longer than you can manage on a busy Saturday.

**QUESTION 4**   

As your time is limited on this occasion, what issues should be a priority at this stage?

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**QUESTION 5**   

What care and options are open to an adult who has been raped? What help is available to you to manage Jackie's immediate needs?

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**FURTHER INFORMATION**

The nearest sexual assault centre with a forensic facility is 3 hours drive away from your clinic. Jackie wants to talk about risks of STIs and then go home and rest before she does anything else. After further discussion, Jackie insists on going home and says she is not ready to talk to the police at this time.

**QUESTION 6**  

What will you do now? Would you prescribe anything?

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**QUESTION 7**    

What advice can you give a patient, to assist in preserving evidence, if the patient has not yet decided whether to report the rape to the police?

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**FURTHER INFORMATION**

Jackie went to the police, who are investigating her case, and she had the forensic examination.

You see Jackie at 2 weeks and 3 months for her sexual health follow-up. She tells you that her flashbacks are subsiding, her sleep is improving and she is beginning to feel normal again. The police have laid charges against a man who was at the party. She is aware that recovery may be a long process and that her appearance in court could be traumatic, but she has decided to go ahead with it and has decided to continue with the support of her sexual assault counsellor.

At her visit today, she has a question to ask you. She has just heard from the police that her urine and blood tests did not show the presence of any drugs.

**QUESTION 8** 

Jackie wants to know how it is possible there was nothing in her system?

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**FURTHER INFORMATION**

Jackie attends your surgery about a year later on an unrelated issue. You have not seen her since some time ago when she told you she was attending counselling.

**QUESTION 9**  

Is it helpful to ask her how she is after the rape?

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## CASE 3 ANSWERS

## ANSWER 1

A helpful approach might be to:

- Respond first to Jackie's distress and the initial concerns she has expressed, which might be considered as providing 'psychological first aid'. This includes making a statement to the effect that sex without consent is a crime.
- Obtain sufficient history to assess the situation.
- Consider what options you can offer Jackie for immediate support when she leaves your surgery today.

The above approach could be achieved using the following examples:

**Respond to the sexual assault:** 'Jackie I am very sorry to hear that. It is terrible that this has happened to you. From what you have told me it sounds like a man had sex with you without your consent. This is a crime. Drugging is also a crime.'

**Respond to her stated concerns:** 'I hear what you are saying about the risk of sexually transmissible infection (STI) and we will come back to that.' Jackie has highlighted her concern that she may 'catch something' and she should be reassured that you will address this issue. It is very possible that the stated concern that she may contract an STI is her 'ticket' to see the doctor in a situation where she feels overwhelmed and her life has been completely disrupted.

**Obtain a fuller history:** 'First can you tell me some more about what happened?'

## ANSWER 2

The initial words and response made by a doctor or people close to the victim can have a significant effect. Examples of statements that may be considered therapeutic for someone who discloses rape include:<sup>1</sup>

- I am sorry for what has happened (this is heard as 'I believe you').
- This is a crime (this is heard as 'this is not your fault').
- I will do what I can to help (this is heard as 'you are not alone').

It is important to listen to the patient, believe their story and be non-judgmental and supportive.<sup>2</sup>

## ANSWER 3

In the period after a sexual assault, victims may experience a wide range of feelings including fear, anxiety, numbness, disbelief, panic, anger, shame, loneliness, embarrassment, irritability, guilt, powerlessness, loss of control, vulnerability, distress and confusion.<sup>3</sup>

## ANSWER 4

The following issues should be addressed:

- Her STI risk

- Pregnancy prevention with postcoital contraception
- Assessing for injury (most people who are raped do not have injuries requiring treatment)
- Expressing support and affirmation for Jackie
- Her social safety:
  - Does she have somewhere safe to go and where she can be supported on leaving the surgery?
  - Will she be physically and emotionally safe at work?
  - What is her emotional/psychological situation (mental health risk assessment)?
- The legal/police situation (Jackie may have been drugged and raped).

Ideally, she should be referred today to a sexual assault service with a counsellor and doctor for psychosocial support, medical care and, if she chooses, a forensic examination, which is best done as early as possible and within 72 hours.

## ANSWER 5

The following care and legal options might be available to an adult who has been raped.

## Care

This depends on where you practice. New South Wales, Victoria, Adelaide, Perth and other locations have specialised sexual assault services where a sexual assault counsellor and a specially trained doctor offer a coordinated psychosocial and medical response and a forensic medical examination if the patient chooses this. The counsellor will also support the patient during any legal process, if one is undertaken.

## Legal

The patient can report the crime to the police for investigation and, where appropriate, for police assistance in promoting the patient's safety (eg to facilitate a court order to prevent future violence or stalking). Police receive training in how to respond to victims of sexual assault and most members of the police force will respond in a sensitive and supportive manner. The police will investigate and, if they can gather sufficient evidence, press charges. This does not depend on whether the patient has physical injuries. In general terms, victims of rape retain control of whether the matter goes to court and in most cases they can withdraw from the police process at any time, up to when the case goes to court.

The police will request that Jackie has a forensic medical examination. This is performed by a forensically trained doctor or nurse, who takes a history, carefully and sensitively examines and documents any injury, and collects swabs from relevant areas to test for the offender's DNA, as well as blood and urine for toxicology testing. The process will be carefully explained to Jackie and she would then have the option to decline any part of the examination (eg speculum use). In dedicated sexual assault centres, the counsellor and doctor work together to provide a holistic service.

It is very valuable to have a local list of services available in your practice, including 24/7 for sexual assault, family violence, etc. This could be prepared and kept up to date by each Medicare Local or by practice staff. You could refer Jackie to the:

- 24/7 telephone counselling service: Rape and Domestic Violence Services Australia (previously known as the Rape Crisis Centre), 1800RESPECT, [www.rape-dvservices.org.au](http://www.rape-dvservices.org.au)
- nearest hospital social worker or emergency department
- police.

Police websites also have useful information on sexual assault (refer to Resources for patients and doctors).

### ANSWER 6

At this stage, the next steps must be tailored to the patient's history, local STI risk and their likelihood of returning for adequate follow-up. The latest guidelines are available in the *National Management Guidelines for Sexually Transmissible Infections*.<sup>4</sup> The RACGP white book<sup>2</sup> and Yarrowplace Rape and Sexual Assault Service<sup>5</sup> also provides some guidance. The Royal Prince Alfred (RPA) Sexual Assault Service provides the following protocol:<sup>6</sup>

At presentation consider prophylaxis for:

- pregnancy prevention
  - levonorgestrel 1500 mg, taken as soon as possible or up to 120 hours later (as its risks are minimal)<sup>7</sup>
- chlamydia, gonorrhoea and syphilis
  - azithromycin 1 g or follow local guidelines, especially if follow-up is not likely
- hepatitis B virus (HBV) infection
  - commence vaccination if not immunised
- human immunodeficiency virus (HIV) infection
  - assess whether post-exposure prophylaxis might be required.

It is important to advise the patient that at this stage it is too early to test for most STIs and that follow-up is essential to ensure that appropriate testing takes place.

At 2 weeks follow-up:

- check blood for HBV, hepatitis C virus (HCV), syphilis, HIV (timing of HIV testing may vary to 4 weeks, depending on the laboratory capability) and  $\beta$ -human chorionic gonadotropin ( $\beta$ -hCG)
- test for chlamydia/gonorrhoea by polymerase chain reaction (PCR) at potentially exposed sites.

At 3 months follow-up:

- check blood for HBV, HCV, HIV and syphilis.

In cases where there might be an increased risk of HCV transmission (eg infected needles used during the assault), a 6-month HCV test will be required.

As Jackie insists on going home, a suitable plan needs to be discussed with her. It is agreed that her sister will stay with her today. Jackie also agrees that they will telephone the rape crisis counsellor (1800RESPECT) when they go home for advice about her options for reporting to the police and for counselling and care.

### ANSWER 7

The following information and advice could be provided to Jackie:<sup>4</sup>

- Do not shower or wash.
- Place the clothes she was wearing in a bag (preferably in individual paper bags). Also advise Jackie to preserve any panty liners, tampons, etc, if relevant.
- If she needs to use the toilet she should press her panties into the vulva before urinating or opening the bowels. Jackie should save these panties in a bag.

There is no approved method of preserving any evidence of drugs that may have been given to her until she can have blood or urine collected. You could consider offering to take samples for your pathology service to screen and hold.

### ANSWER 8

Several possibilities could account for the fact that no drugs were identified in her urine and blood samples. These include the following:<sup>8</sup>

- Many drugs are eliminated very rapidly from the body.<sup>4</sup>
- Some of the new designer drugs may not be detected at all with current testing methods.
- A common method for spiking of drinks is the use of 'extra' alcohol.<sup>8</sup>
- It is possible for a person to lose track of how many alcoholic drinks they have had or not realise that the same number of drinks can have a variable response depending on other factors such as fatigue, other medications or drugs.

Discussion of the last point needs to be handled very sensitively, as victims of rape often tend to blame themselves and tend to very easily hear blame from others. The victim being intoxicated does not excuse the perpetrator of a crime, or mean that the victim is to blame. However, if history shows that Jackie does drink to a dangerous level, it may be helpful to discuss the many health risks of excessive alcohol consumption with Jackie.

### ANSWER 9

It is helpful to ask how she is and give her an opportunity to talk if she wishes to do so. Research shows rape survivors find 'a wall of silence', where the opportunity to talk is limited or absent, and this does not promote recovery. You also do not want to give the impression that today or every time she sees you she is required to think or talk about the rape. It may be helpful to ask her if she wants to fill you in on how she is going, while reassuring her that it is not necessary to talk about it if she does not want to.<sup>9</sup>

### CONCLUSION

Jackie is glad you asked. She is proceeding with the police and court case despite the date of the court case being postponed twice due to legal delays. The counsellor is available on the telephone to support her in this. She is rarely having flashbacks but has occasional anxiety. She is still fearful of meeting the man and had considered moving

interstate. She has decided to leave the advertising world and has commenced retraining as a nurse. She is grateful for your support to her and her sister on that busy Saturday morning and this has influenced her decision to consider a career in health.

## REFERENCES

1. Rape and Domestic Services Australia. Available at [www.rape-dvservices.org.au](http://www.rape-dvservices.org.au) [Accessed 24 June 2014].
2. Royal Australian College of General Practitioners. Abuse and violence. Working with our patients in general practice. 4th edn. Melbourne: RACGP, 2014. Available at [www.racgp.org.au/your-practice/guidelines/whitebook/](http://www.racgp.org.au/your-practice/guidelines/whitebook/) [Accessed 22 July 2014].
3. New South Wales Police Force. Adult sexual assault. Available at [www.police.nsw.gov.au/community\\_issues/adult\\_sexual\\_assault](http://www.police.nsw.gov.au/community_issues/adult_sexual_assault) [Accessed 24 June 2014].
4. Sexual Health Society of Victoria. National management guidelines for sexually transmissible infections. Melbourne: Sexual Health Society of Victoria, 2008. Available at <http://mshc.org.au/Portals/6/NMGFSTI.pdf> [Accessed 1 August 2014].
5. Yarrowplace Rape and Sexual Assault Service. Information for health/welfare professionals. Available at [www.yarrowplace.sa.gov.au/healthprof\\_assess.htm](http://www.yarrowplace.sa.gov.au/healthprof_assess.htm) [Accessed 25 August 2014].
6. Induction Manual RPA and LIVERPOOL Sexual Assault and Clinical Forensic Medicine. 2013.
7. Rossi S, editor. Levonorgestrel. In: Australian Medicines Handbook. Adelaide: Medicines Handbook Pty Ltd, 2014.
8. Drummer O, Odell M. The Forensic Pharmacology of Drugs of Abuse. London: Arnold Publishers, 2001.
9. Lievore D. No longer silent. A study of women's help-seeking decisions and service responses to sexual assault. Canberra: Australian Institute of Criminology, 2005. Available at [www.aic.gov.au/media\\_library/publications/other/2005-06-noLongerSilent.pdf](http://www.aic.gov.au/media_library/publications/other/2005-06-noLongerSilent.pdf) [Accessed 1 August 2014].

## RESOURCES FOR PATIENTS AND DOCTORS

- The Better Health Channel. Sexual assault. [www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Sexual\\_assault?open](http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Sexual_assault?open)
- New South Wales Police. Adult sexual assault. [www.police.nsw.gov.au/community\\_issues/adult\\_sexual\\_assault](http://www.police.nsw.gov.au/community_issues/adult_sexual_assault)
- Queensland Police. Adult sexual assault. [www.police.qld.gov.au/programs/adultassault/adultasslt.htm](http://www.police.qld.gov.au/programs/adultassault/adultasslt.htm)
- Victoria Police. Crime prevention and community safety. Sexual assault. [www.police.vic.gov.au/content.asp?Document\\_ID=10904](http://www.police.vic.gov.au/content.asp?Document_ID=10904)
- Western Australia Police. Sexual assault. [www.police.wa.gov.au/Yoursafety/Sexualassault/tabid/1607/Default.aspx](http://www.police.wa.gov.au/Yoursafety/Sexualassault/tabid/1607/Default.aspx)
- Australian Institute for Family Studies. Crisis support. [www.aifs.gov.au/acssa/crisis.html](http://www.aifs.gov.au/acssa/crisis.html)

**CASE 4**

**EMILY'S SECRET PAIN**

Emily is an art teacher aged 30 years and has been in a relationship for 3 years with Jason, an engineer. She has a 2-year history of increasing pain with sex and with Pap smears. Emily and Jason have not had sex for the past 6 weeks, as Emily cannot bring herself to have sex any more. This situation has precipitated her appointment today.

**QUESTION 1**  

What questions would you ask Emily to collect a relevant history?

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**FURTHER INFORMATION**

On questioning, it transpires that Emily enjoyed her sexual relationship with Jason in the first 6 months, before they moved in together. However, since then there had been a number of stresses and Emily was scared that the relationship would be at risk if something did not change soon.

Vaginal examination and Pap smears used to be fine until her last Pap smear. On that occasion there was a different doctor and he needed to repeat the Pap smear because he did not get enough cells the first time. Emily felt quite tense because he seemed rushed but she said nothing, even though the procedure hurt her a lot. The doctor said that the Pap smear would be over soon and she knew it would be, but found the pain excruciating while it was being performed.

**QUESTION 2**  

Emily asks 'What's wrong with me?' How would you respond?

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**QUESTION 3**  

Emily asks 'Am I the only one? I've never heard of anyone else having this.' How would you answer?

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**FURTHER INFORMATION**

Emily is due for a Pap smear but is terrified it will hurt.

**QUESTION 4**  

How would you discuss this with her and how would you proceed?

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**QUESTION 5** 

What are the options and rationales for the treatment of vaginismus?

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**QUESTION 6** 

What can doctors do to help identify and treat vaginismus?

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**CASE 4 ANSWERS**

**ANSWER 1**

A psychosexual history helps us understand what life circumstances, including health issues, may have contributed to the patient's symptom(s). Relevant questions for a psychosexual history include questions regarding Emily's personal, medical, sexual and psychosocial history (Table 1).

**Table 1. Obtaining a psychosexual history<sup>1</sup>**

History	Possible questions/discussions points
Personal history (including family's attitudes and beliefs about sex and a brief outline of life history, including school, friendships and work)	What was it like growing up in your family? Was sex talked about or was it unmentionable? Were parents physically affectionate? How did you find out about sex? What were your ideas about it before you got started? How did you find out about periods? Any pain with periods? Tampon use?
Medical and surgical history	Ask especially about history of pelvic surgery, infections (eg recurrent thrush), episiotomies, etc
Relationship history	Relationships with previous partners Feelings of self-worth and desirability Relationship with current partner
Sexual history	Contraception Past-traumatic experiences: physical/sexual abuse, sexual assault or painful vaginal examination
Psychosexual history	Describe the problem in detail Has the problem always been there? Has this been a problem in any previous relationship? The reason for coming now

**ANSWER 2**

On the basis of her presenting and psychological history, Emily has vaginismus. She has had severe pain with penetration until penetration felt impossible and pain with Pap smears.

Vaginismus is newly classified in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5),<sup>2</sup> as part of a genitopelvic pain disorder/penetration disorder. The DSM-5 states that the most frequent clinical presentation is one in which both psychological and physiological factors contribute. Associated features are described as partner factors, relationship factors, individual vulnerability factors, cultural or religious factors, as well as medical factors.

There has been a fundamental shift over the past two decades from a dualistic view of the aetiology of painful and/or difficult vaginal penetration as being due to either psychological or physiological causes to the current multifactorial perspective, with

a biopsychosocial lens, which sees a complex interplay between physiological, psychological and social factors.<sup>3</sup>

Women with vaginismus often avoid sexual intercourse, experience involuntary muscle contraction and anticipate, fear or experience pain or burning with penetration. The degree of distress is more central to the diagnosis than muscle tone.<sup>4</sup>

**ANSWER 3**

Women feel isolated and ashamed when penetrative sex is painful or impossible. Although the population prevalence of vaginismus remains unknown, it has been reported to be 5–17% in clinical settings.<sup>5</sup> It might also be helpful to explain that vaginismus may be primary (ie pain at first intercourse) or secondary (ie developing after pain-free intercourse, as in Emily's case following a painful Pap smear).<sup>4</sup>

**ANSWER 4**

It is important to conduct an examination to diagnose organic pathology,<sup>4</sup> but the psychosexual examination, like a Pap smear, is not urgent and can wait until Emily feels ready. She needs to know that she can speak up if she is uncomfortable and trust that her doctor will listen to her and respect her wishes. Avoidance of Pap smears or pain with Pap smears can be indicators of past trauma.<sup>6</sup> Given that the biggest risk factor for cervical cancer is not being screened regularly, it is important to ask women if they have had their routine health checks and if not, why not. Ensuring that women get the help they need to deal with painful or stressful feelings that have resulted in pain or 'tight muscles' will enable them to have normal preventive healthcare when they feel ready.

Several visits may be required before a woman is ready to be examined and any examination should be approached gently.<sup>4</sup> For example, when she is ready to be examined, you could ask Emily to tell you if there is any pain or discomfort, or if she wants to stop the examination at any time. You should be watching for any signs of distress, but you should avoid inadvertently causing pain (iatrogenic pain). Describe in detail everything that you intend to do. Inviting her to contract then relax her pelvic muscles or instructing her to take a deep breath in then exhale slowly, are two physical ways to help her vaginal muscles relax and allow a finger to slide in.

The clinical examination can be used as a learning tool to educate patients on performing self-examinations at home. As Emily familiarises herself with this hidden part of her body in the privacy of her home, fantasies or fears that she has with penetration may emerge that can be discussed as part of her therapy. This helps connect the physical (how it feels) and emotional (what it's like for her) when talking about painful feelings in her body and her life.

**ANSWER 5**

Some doctors and patients, think that if penetrative sex and pelvic examination were possible (ie symptoms were 'fixed') the patient's problems would be solved; hence the offer of surgery, 'stretching' under anaesthesia, dilators, biofeedback, oral and intravaginal anxiolytics, botox injections, lignocaine gel and nitroglycerin

ointment.<sup>5</sup> The rationale for these treatments is that the problem is 'spastic' vaginal muscles. There is very limited evidence from controlled trials concerning their effectiveness.<sup>7</sup>

The ethics of prescribing medication or performing surgery, which have potential side effects on patients whose problems are not organically based, has not received much attention. This is because of the value placed on 'normality'.<sup>8</sup>

Often, doctors and patients believe that 'getting a Pap smear over quickly' will shorten the duration of pain for a frightened patient and therefore be helpful. However, this can inadvertently re-traumatise a patient who has a history of physical/sexual abuse or sexual assault (iatrogenic traumatic examination).<sup>9–11</sup>

The DSM-5 consensus that vaginismus has biopsychosocial origins<sup>2</sup> suggests that we should treat the patient holistically, not just treat the symptom. Psychosexual therapy is the only therapy to adopt a biopsychosocial model of diagnosis and treatment. In this model, vaginismus is conceptualised as a psychosomatic symptom: the symptom is the result of a complex interplay between physiological, psychological and interpersonal factors, expressed in the body (soma) as a painful symptom, which alerts us to painful feelings. An integrated approach to management is offered on the basis of this understanding. The aim of treatment is not to achieve tolerance of penetration but to encourage participation in sex only when it is pleasurable and pain-free. Individual and relationship issues are important, as is the experience of sex for the patient.<sup>12</sup>

### Elements of treatment

The psychosexual history and examination (when the patient is ready) gives the doctor information needed for understanding the patient's problem in the context of her life and diagnoses any physical causes that need treatment (*Tables 2, 3*). It also begins the process of treatment where the woman, with or without her partner, can begin to make connections between what has happened in her life and how she has been affected by it. It requires a factual, non-judgemental attitude by the doctor.

**Table 2. Physical/medical causes of pain that may be contributory factors in vaginismus<sup>4,5</sup>**

- Recurrent genital tract infections (eg thrush) and topical treatments
- Vulvovestibulitis
- Oestrogen deficiency (peri/post-menopause, following oophorectomy (surgical menopause))
- Prolonged use of depot medroxyprogesterone contraception
- Trauma associated with childbirth (including episiotomy)
- Genital surgery, radiotherapy and irritation caused by douches, spermicides or latex in condoms
- Diabetes, multiple sclerosis or spinal cord injury (patients may experience pain with penetration because of poor lubrication)

There is controversy around the differentiation between vulvar vestibulitis syndrome (VVS) and vaginismus because patients with vaginismus may also show signs of allodynia on the cotton bud test.

**Table 3. Understanding and addressing features of vaginismus**

- Many women are unfamiliar with this hidden part of their body (ie the vagina)
- There may be an associated fantasy that the vagina is too small to accommodate a penis when the patient doesn't feel 'big enough' for the adult world of sex
- Their bodies harbour painful feelings (physical and psychological)
- The unresolved issues that lead to pain and tightness may be unconscious. Patients are fearful and the symptom is self-protective. Some may feel disgust<sup>13</sup> with the sexual part of their body
- Address pain and tightness in vaginal muscles (specialist physiotherapy) and address painful feelings (psychosexual therapy)
- The patient needs to feel in control and they need to develop trust, slowly and gently

Patients should be encouraged to talk about their feelings, and their fears, genital pain, pelvic floor muscle tension and issues of sexual pleasure.<sup>5,14</sup> Treatment should be individualised for each woman, with or without her partner.

Encouraging non-penetrative sex is important at times when penetrative sex would interfere with progress of treatment.

Specially trained physiotherapists with skills in patient education (anatomy, physiology of sexual response) and gentle examination are helpful in a multidisciplinary approach. Initially, the woman is encouraged to look at and self-touch in a nonsexual manner, moving on to the insertion of a finger, then a small tampon. This can be combined with pelvic floor physiotherapy. Whilst the use of dilators has been a standard approach for physiotherapist-led treatment, this focus on tolerating increasingly sized dilators can make self-examination at home feel more like stretching exercises than exploration to see how the vagina and the examining finger feel, what that's like for the woman, and what feelings are aroused, increasing her sense of mastery of her own body as she gets in touch with her feelings around sex.

### Recommendations on sexual activity

Women often feel guilty and men may feel frustrated by the idea of non-penetrative sex for a period of time. However, with penetrative sex being off the agenda, there may be more experimentation with non-penetrative sex (foreplay). This may lead to an increase in trust, arousal and natural lubrication, and improvement in sexual connection, especially when penetrative sex has been the primary mode of sexual expression. It will also help differentiate between patients for whom penetrative sex is the primary problem (individual issues) and those for whom there are additional relationship difficulties to be addressed, as the latter patients tend to continue to avoid sex.

**ANSWER 6**

Many women who have sexual pain do not discuss their problems with health providers. A Swedish study reported that only 28% of approximately 3000 women aged 20–60 years consulted their doctor regarding prolonged and severe dyspareunia.<sup>15</sup>

Doctors can take a leading role in identifying vaginismus and ensuring that patients have regular Pap smears by making Pap smears a positive experience for their patients (Table 4), as well as offering education, counselling and other support as required.<sup>4</sup>

**Table 4. What can doctors do?**

- Ask all female patients if they have had routine Pap smears
- Refer patients who have pain with sex or Pap smears for psychosexual therapy with or without pelvic floor physiotherapy
- Ensure there is no pain with Pap smears; Pap smears should be done when the patient feels ready, under her direction
- Ensure that patients understand that they can stop an examination at any time, as many patients have difficulty saying 'no'

**CONCLUSION**

Jason accompanied Emily to her second appointment. At this visit, information and education on vaginismus were provided. The problem was discussed, as well as how it affected each of them. The importance of a period of non-penetrative sex until Emily was ready was also discussed. Jason said it would be hard for him, as they were already doing the 'no sex' thing and they had not had sex for at least 6 weeks. It was explained that non-penetrative sex did not mean 'no sex' and that their sex life was important. Other forms of sexual activity could continue as long as Emily felt comfortable. Emily confirmed that she had been avoiding anything more than a hug because of her fear that it would lead to attempts at penetrative sex. She knew it wasn't good for their relationship but she didn't know what else to do.

Emily thought it would be helpful for them to continue to attend sessions together to deal with tensions that had built up in the relationship. She was pleased to be referred to a specialist physiotherapist to help at the physical level. After 6 sessions together, Emily came by herself to sort out her feelings about her body and sexuality. She now trusted Jason to go no further than she was comfortable with. Both were finding their (non-penetrative) sexual relationship rewarding. Within 6 months Emily felt ready to guide Jason's finger into her vagina in a way where she felt in control. She was then ready to guide him towards penetrative sex, happy to feel that this was something she wanted and felt ready for, and confident that she could also say 'no' if she didn't want to participate for whatever reason.

**REFERENCES**

1. Goodwach R. Sex therapy: historical evolution, current practice. Part 2. ANZJFT 2005;26:178–83.
2. American Psychiatric Association. Diagnostic criteria and codes: sexual dysfunctions: genito-pelvic pain/penetration disorder. In: Diagnostic and Statistical Manual of Mental Disorders, 5th edn. Arlington: American

Psychiatric Publishing, 2013. Available at <http://dsm.psychiatryonline.org/content.aspx?bookid=556&sectionid=41101779&resultclick=24> [Accessed 24 July 2014].

3. Boyer SC, Goldfinger C, Thibault-Gagnon S, Pukall CF. Management of female sexual pain disorders. *Adv Psychosom Med* 2011;31:83–104.
4. Crowley T, Goldmeier D, Hiller J. Clinical review: diagnosing and managing vaginismus. *BMJ* 2009;338:225–29.
5. Lahaie M, Boyer C, Amsel R, Khalife S, Binik Y. Vaginismus: a review of the literature on the classification/diagnosis, etiology and treatment. *Womens Health* 2010;6:705–19.
6. Farley M, Golding JM, Minkoff JR. Is a history of trauma associated with a reduced likelihood of cervical cancer screening. *J Fam Pract* 2002;51:827–31.
7. Melnik T, Hawton K, McGuire H. Interventions for vaginismus. *Cochrane Database Syst Rev* 2012;12:CD001760. doi: 10.1002/14651858.CD001760.pub2.
8. Moynihan R, Cassels A. Selling sickness: how the world's biggest pharmaceutical companies are turning us all into patients. Sydney: Allen & Unwin, 2005.
9. Australian Women's Coalition, Australian Federation of Medical Women, Victorian Medical Women's Society. Happy healthy women not just survivors. Consultation report: advocating for a long-term model of care for survivors of sexual violence. Ingleburn: Australian Women's Coalition, 2010. Available at [www.awcausa.org.au/resources/documents/HHW\\_Consultation\\_Report.pdf](http://www.awcausa.org.au/resources/documents/HHW_Consultation_Report.pdf) [Accessed 30 June 2014].
10. Taylor SC, Pugh J, Goodwach R, Coles J. Sexual trauma in women. The importance of identifying a history of sexual violence. *Aust Fam Physician* 2012;41:538–41.
11. Pedersen B, Mohl B. Vaginismus. Iatrogenic precipitation and maintenance. *Acta Obstet Gynaecol Scand* 1992;71:525–28.
12. Goodwach R. Sex therapy: historical evolution. *Curr Pract Part 1 Aust N Z J Fam Therapy* 2005;26:155–64.
13. de Jong PJ, Peters ML, Olatunji BO, McKay D. Sex and the sexual dysfunctions: the role of disgust and contamination sensitivity. Washington DC: American Psychological Association, 2009. p253–70. doi: 10.1037/11856-012.
14. Binik YM. The DSM diagnostic criteria for vaginismus. *Arch Sex Behav* 2010;39:278–91.
15. Danielsson I, Sjöberg I, Stenlund H, Wikman M. Prevalence and incidence of prolonged and severe dyspareunia in women: results from a population study. *Scand J Public Health* 2003;31:113–18.

**RESOURCES FOR PATIENTS**

- The Jean Hailes Foundation, <http://jeanhailes.org.au/health-a-z/sex-sexual-health/painful-sex-dyspareunia>
- The Society of Obstetricians and Gynaecologists of Canada, <http://sogc.org/publications/when-sex-hurts-vaginismus>
- Sexual Health Australia, [www.sexualhealthaustralia.com.au/page/vaginismus.html](http://www.sexualhealthaustralia.com.au/page/vaginismus.html)

**RESOURCES FOR DOCTORS**

- Crowley T, Richardson D, Goldmeier: Recommendations for the management of vaginismus: BASHH Special Interest Group for Sexual Dysfunction Int J STD & AIDS 2006;17:14–18.
- Lahaie M, Boyer C, Amsel R, Khalife S, Binik Y. Vaginismus: A review of the literature on the classification/diagnosis, etiology and treatment. *Womens Health* 2010; 6:705–19.
- McGuire H, Hawton K. Interventions for vaginismus. *Cochrane Database Syst Rev* 2012;12:cd001760.
- Taylor CS, Pugh J, Goodwach R, Coles J. Sexual trauma in women. The importance of identifying a history of sexual violence. *Aust Fam Phys* 2012;41:538–41.

**CASE 5**

**JESSICA HAS HAD NO PERIODS FOR 4 MONTHS**

Jessica is a beautician aged 26 years. She is concerned about oligomenorrhoea, which she has had for the last 4 months. She is not taking any medications. Jessica had menarche at the age of 14 years and her cycles have always been irregular (45–60 days). She is currently sexually active and her partner uses condoms for contraception. They do not have any plans for pregnancy. There is a strong family history of type 2 diabetes (T2DM) and myocardial infarction on Jessica's father's side. Jessica has gained 7 kg over the past 2 years, which she is struggling to lose. She has a sedentary lifestyle as she works full time and has little time for exercise.

On examination she is not cushingoid. She is normotensive and has a body mass index (BMI) of 28 kg/m<sup>2</sup> (weight: 79 kg, height: 168 cm) and central adiposity with a waist circumference of 89 cm. On further questioning, Jessica describes excessive facial hair growth, which requires regular waxing. She gives herself a score of 11 on the Ferriman-Gallway (FG) scoring system for hirsutism.<sup>1</sup>

**QUESTION 1** 

What are the key issues in Jessica's history?

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**QUESTION 2** 

What is the most likely diagnosis? What investigations would you perform to confirm the diagnosis?

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**FURTHER INFORMATION**

The results of Jessica's investigations reveal the following:

- serum testosterone: 2.0 nmol/L (normal: 0.1–1.7)
- sex hormone-binding globulin (SHBG): 15 nmol/L (normal: 18–136)
- free androgen index (FAI): 13% (normal: 0.7–10.9)
- thyroid stimulating hormone (TSH), prolactin, follicle stimulating hormone (FSH), luteinising hormone (LH) and β-human chorionic gonadotropin (hCG): normal
- transvaginal ultrasound: multiple follicles consistent with polycystic ovary syndrome (PCOS).

**QUESTION 3** 

What is the diagnosis?

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**QUESTION 4** 

What are the key health challenges for Jessica? What general approach should you take to manage her key issues?

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**QUESTION 5** 

What therapeutic options for oligomenorrhoea should be considered and discussed with Jessica?

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**QUESTION 6** 

What role would weight management have in improving Jessica's metabolic and reproductive features and how will you facilitate this?

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**FURTHER INFORMATION**

Jessica's oral glucose tolerance test (OGTT) shows normal results and her fasting lipids show mild dyslipidaemia with normal total cholesterol and low-density lipoprotein-cholesterol (LDL-C) levels, and a low level of high-density lipoprotein cholesterol (HDL-C). Jessica chooses to start a COC for regulation of her periods and considers laser therapy for hirsutism. She starts seeing a dietician, as well as attending regular aerobic exercise sessions at the local gym to achieve weight loss.

Four years later, Jessica visits you, after presenting to the emergency department the day before for vaginal bleeding and abdominal pain. She was found to be pregnant and was referred for an ultrasound, which confirmed a live pregnancy at 8 weeks of gestation. The bleeding stopped and Jessica was reassured that the pregnancy was still viable at this stage.

On examination, her weight is 73 kg, BMI is 26 kg/m<sup>2</sup> and she is normotensive. Jessica tells you that she stopped taking her COC about 6 months ago with the aim of getting pregnant.

Knowing her previous history of PCOS, you referred Jessica for an early OGTT to screen for gestational diabetes. Her fasting plasma glucose was 5.1 mmol/L and 2-hour plasma glucose was 9 mmol/L.

**QUESTION 7** 

How would you interpret this result? What would be your approach?

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**QUESTION 8** 

What information would you give Jessica regarding postpartum screening for diabetes?

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**QUESTION 9** 

How would you advise Jessica about her risk of T2DM? How should this be managed?

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**QUESTION 10** 

What is the risk of gestational diabetes mellitus (GDM) with Jessica's future pregnancies? What would you advise her about pre-pregnancy screening for dysglycaemia in women with PCOS?

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## CASE 5 ANSWERS

## ANSWER 1

The key issues from Jessica's history include the following:

- oligomenorrhoea and history of irregular menses
- increased body weight with a BMI of 28 kg/m<sup>2</sup>
- sedentary lifestyle and difficulty losing weight
- family history of T2DM and cardiovascular disease
- hirsutism.

## ANSWER 2

The most likely diagnosis is PCOS. This is the most common endocrine condition in women of reproductive age, affecting 7–28% of women and increasing in frequency with increasing weight.<sup>2</sup> PCOS occurs in 7–12% of lean women and 20–28% of those who are overweight. Women at risk of PCOS include Aboriginal and Torres Strait Islander and Asian women, or women with a family history of PCOS or T2DM.<sup>2</sup>

Insulin resistance and hyperandrogenism are the key hormonal disturbances underpinning PCOS and are independent of, but exacerbated by, weight gain (Figure 1).<sup>3</sup>

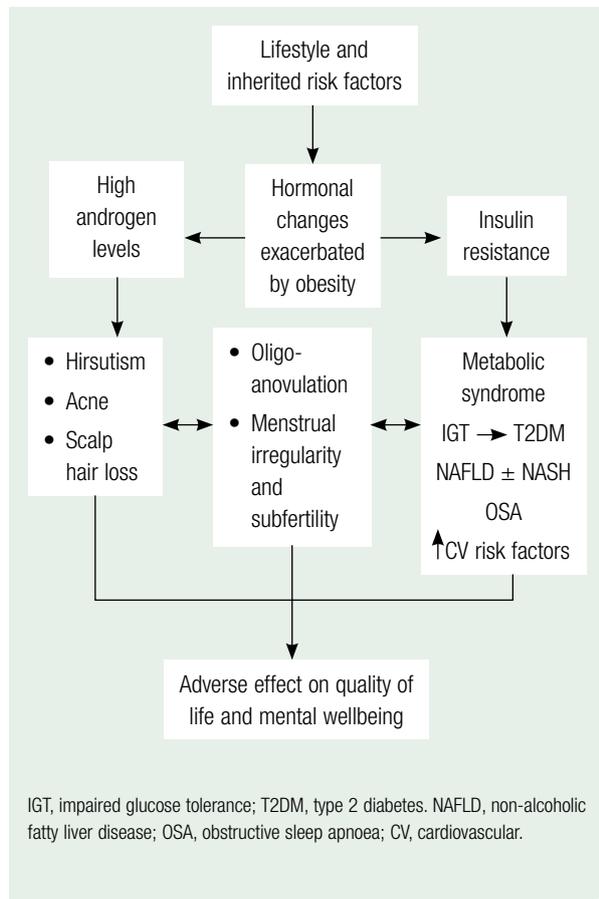


Figure 1. Aetiological, hormonal and clinical features of PCOS.<sup>2</sup>

Diagnosis of PCOS requires two of the three Rotterdam criteria, which are the internationally accepted diagnostic criteria for PCOS, as well as exclusion of other aetiologies, such as hypothyroidism, Cushing's syndrome, late-onset congenital adrenal hyperplasia (CAH), androgen-secreting tumours and hyperprolactinemia.<sup>2</sup> The Rotterdam criteria are:

- oligo- or anovulation
- clinical and/or biochemical signs of hyperandrogenism
- polycystic ovaries on ultrasound.

Recommended investigations to confirm the diagnosis include:

- Measurement of testosterone, SHBG and free androgen index (FAI) or calculated free testosterone. Total testosterone alone or additional androgens, including androstendione and dehydroepiandrosterone sulphate, are not recommended in routine investigation of PCOS.<sup>2</sup>
- A pelvic ultrasound could be performed to assess ovarian morphology, if required for diagnosis, and should be performed in the follicular phase of the menstrual cycle where possible. Pelvic ultrasound is also useful for measurement of endometrial thickness in the setting of oligo-/amenorrhoea. However, it is not required in all cases (it should be performed per vaginal and by an experienced gynaecological ultrasonographer to increase accuracy where possible), and is not recommended in adolescents, especially if they are not yet sexually active; 60–80% of adolescents will meet PCOS criteria as they reach reproductive maturity.<sup>2</sup>
- Measurement of TSH, serum prolactin, 17(OH)-progesterone (in the follicular phase to exclude CAH) and  $\beta$ -hCG should be undertaken to exclude other causes.<sup>2</sup>

It is difficult to assess androgen status in women who are on the combined oral contraceptive (COC) pill as its effects include oestrogen-mediated increases in SHBG and reduction in androgens; therefore, COCs should be withdrawn, ideally for 3 months prior to reliable hormonal investigation.<sup>4</sup>

Referral to an endocrinologist is necessary for work-up if other rare causes, including Cushing's syndrome, non-classical CAH or rare androgen-secreting tumours, are suspected clinically (ie rapidity and severity of onset, hypertension, virilisation, markedly elevated androgen levels or cushingoid features).

## ANSWER 3

On the basis of irregular menses, clinical and biochemical hyperandrogenism and follicles on pelvic ultrasound, Jessica has PCOS. Note that other causes were excluded on the basis of biochemistry and the absence of clinical suspicion of more severe conditions.

## ANSWER 4

Women with PCOS may present with a range of features, including reproductive (hyperandrogenism, hirsutism, anovulation, infertility), metabolic (insulin resistance, impaired glucose tolerance, GDM, T2DM, dyslipidaemia, obstructive sleep apnoea) and psychological (increased anxiety, depression and worsened quality of life) features.<sup>5–8</sup> Women with PCOS are at a higher risk of developing

pre-diabetes, GDM and T2DM, and have higher cardiovascular risk factors, all with onset at an earlier age and all affecting lean as well as overweight women.<sup>6</sup> It is increasingly recognised that PCOS is not only a reproductive issue, but also a metabolic disease that carries important health risks from a young age.

A multidisciplinary approach including evaluation, patient education and consideration of treatment for each of the reproductive, metabolic and psychosocial areas is necessary.<sup>2</sup> Targeted treatment options are summarised in *Table 1*.

<b>Table 1. Targeted treatment options for polycystic ovary syndrome</b>
<b>Oligomenorrhoea/amenorrhoea</b>
<ul style="list-style-type: none"> <li>• Lifestyle change (5–10% weight loss + structured exercise)</li> <li>• Combined oral contraceptive pill (low oestrogen doses, eg 20 µg may have less impact on insulin resistance)</li> <li>• Cyclic progestins (eg 10 mg medroxyprogesterone acetate 10–14 days every 2–3 months)</li> <li>• Metformin (improves ovulation and menstrual cyclicity)</li> </ul>
<b>Hirsutism</b>
<ul style="list-style-type: none"> <li>• Self-administered and professional cosmetic therapy is first line (laser is recommended)</li> </ul>
<b>Pharmacological therapy</b>
<ul style="list-style-type: none"> <li>• Consider if there is patient concern or if cosmetic treatment is ineffective/inaccessible/unaffordable</li> <li>• Should be trialled for at least 6 months before making changes in dose or medication</li> <li>• Primary therapy is the COCP (monitor glucose tolerance in those at risk of diabetes)</li> <li>• Anti-androgen monotherapy (eg spironolactone) should not be used without adequate contraception</li> <li>• Combination therapy – if 3–6 months of COCP is ineffective, add anti-androgen to COCP (daily spironolactone, and if &gt;50 mg twice daily)</li> </ul>
<b>Infertility</b>
<ul style="list-style-type: none"> <li>• Advise smoking cessation, optimal weight, exercise and folate supplementation</li> <li>• Advise regarding the age-related decline in fertility to allow optimal timing of family planning</li> <li>• Infertility therapies may include clomiphene, metformin, gonadotropins, aromatase inhibitors, surgery and <i>in vitro</i> fertilisation</li> </ul>
<b>Cardiometabolic risk</b>
<ul style="list-style-type: none"> <li>• Lifestyle change with a &gt;5% weight loss in those who are overweight reduces diabetes risk by ~50–60% in high risk groups</li> <li>• Metformin reduces the risk of diabetes by ~50% in adherent high risk groups*</li> </ul>
*Prescribed for this indication by specialists only.
Reproduced with permission from the RACGP from Boyle J, Teede H. <i>Aust Fam Physician</i> 2012;41:752–56.

Lifestyle intervention is recognised as the first step in management of women with PCOS.<sup>2</sup> A single or combined approach of diet, exercise and/or behavioural interventions is required. Weight loss (in women

with a BMI ≥25 kg/m<sup>2</sup> [overweight/obese]) and prevention of weight gain (in women with a BMI 18.5–24.9 kg/m<sup>2</sup> [lean]) should be actively encouraged lifelong, as there is documented evidence that women who are insulin-resistant gain weight at a faster rate than unaffected women. Prevention of weight gain through regular monitoring, lifestyle efforts and support should be encouraged in all women with PCOS and requires far less lifestyle modification than weight loss.<sup>2</sup>

For women who are overweight, a 5–10% loss of current body weight is an achievable realistic goal to set in the short term and has significant benefits across metabolic and reproductive features including pregnancy. Weight loss can be achieved through moderate reduction of energy intake, and introduction of moderate physical activity including structured exercise of at least 150 minutes per week with 90 minutes of this exercise being aerobic activity at moderate-to-high intensity.<sup>2</sup> A large randomised controlled trial reported weight reductions of 5–10% over 2 years for a range of energy-reduced diets with different macronutrient content. It seems that caloric (energy) restriction per se, rather than macronutrient composition, is effective for weight loss and clinical benefits.<sup>9</sup> Physical activity, even in the absence of weight loss, improves a range of factors including hypertension, insulin resistance, impaired glucose tolerance and ovulatory function, and should be encouraged in women with PCOS.<sup>10</sup>

**Management of reproductive features**

Initial steps in management include planning early for family initiation, prevention of weight gain and intensive lifestyle programs, all of which are important in the management of PCOS in primary care. COCs are effective in achieving menstrual cycle regularity, providing contraception and controlling hirsutism; however, there may be a negative influence on insulin resistance, and a low-dose COC may be preferred.<sup>4</sup> Fertility is not necessarily impaired in all patients with PCOS and, depending on the severity of their condition, some women conceive without medical intervention. Contraception, therefore, is still relevant. Smoking history, age, weight, metabolic risk and thromboembolism need to be considered when prescribing COCs.<sup>2</sup>

If infertility is an issue, pharmacological options include ovulation induction with clomiphene citrate, metformin, gonadotropins and, more recently, aromatase inhibitors. Additional options include surgery (laparoscopic ovarian drilling) and, if ovulation induction is unsuccessful or there are other infertility factors, *in vitro* fertilisation.<sup>2</sup> Steps to optimise fertility, including lifestyle changes, can be undertaken at the primary care level; however, once infertility (12 months of failure to conceive) is established, referral to a fertility specialist is recommended. In older women referral should not be delayed if infertility is suspected. Additionally, patients should be advised that a history of PCOS increases the risk for GDM, necessitating earlier screening during pregnancy.<sup>11</sup>

When managing hirsutism the choice of therapeutic agents depends on patient preference, impact on wellbeing, access and affordability, and includes first-line cosmetic therapy (laser and electrolysis) and medical options (eg COCs with potential addition of an anti-androgen in generalised hirsutism if required).

Women with PCOS are at increased risk of developing endometrial

cancer due to anovulation with unopposed uterine exposure to oestrogen. In this setting, intermittent progestin every 3 months may be used to induce a withdrawal bleed and protect the endometrium from hyperplasia if the COC is not desired or tolerated.<sup>2,12</sup>

**Management of metabolic features**

National and international guidelines recommend the following screening program for metabolic risk management in PCOS:<sup>2,12</sup>

- Screen for pre-diabetes (impaired fasting glucose and impaired glucose tolerance) and diabetes
  - Start screening from a young age, especially preconception and early in pregnancy.\*
- Encourage smoking cessation.
- Monitor anthropometric factors including weight, BMI and waist circumference at most visits.
- Measure fasting lipids every 2 years if normal and every year if abnormal and/or the patient is overweight or obese. The most common abnormalities are low levels of high-density lipoprotein cholesterol (HDL-C) and high levels of triglycerides.
- Measure blood pressure annually if BMI is <25 kg/m<sup>2</sup>, or at every visit if BMI is >25 kg/m<sup>2</sup>.

\*An OGTT test is recommended every 2 years in all women and every year in those with additional risks for diabetes (age, ethnicity, parental history of diabetes, history of high glucose levels, smoking and use of COCs or antihypertensive medications, physical inactivity and waist circumference >80 cm). Note, even lean and younger women often have impaired glucose tolerance, providing opportunities for prevention; this is missed in 60–80% of cases with fasting glucose alone in this population.

**Management of psychosocial features**

Anxiety and depression are far more common in patients with PCOS, as is poorer quality of life. Mood disorders warrant screening and need to be addressed if present, to optimise engagement

and adherence to lifestyle interventions. The national guideline recommends emotional health screening, especially for depression and anxiety, and provides an evidence-based, simple screening tool to facilitate this in women with PCOS.<sup>2</sup>

**Therapeutic benefits of metformin in PCOS**

Metformin is not first-line treatment in PCOS and its role in PCOS remains controversial.<sup>2</sup> Nevertheless, large diabetes prevention trials that included women with PCOS have shown that metformin can prevent weight gain, but it is not a substitute for lifestyle interventions, nor will it further reduce weight in patients using effective lifestyle programs.<sup>13,14</sup> Hence, metformin may remain a treatment consideration for prevention of weight gain, and management and prevention of impaired glucose tolerance and diabetes in women with PCOS, if diet and exercise are ineffective.<sup>4,12</sup>

Metformin also has a role in regulation of menses and ovulation, especially where contraception is not desired, as it has been shown to improve menstrual regularity and ovulation rate.<sup>12,15,16</sup> Studies on metformin have not been sufficiently powered to study its effect on acne and hirsutism; some studies show benefit, but it is likely to be less effective than the COC.<sup>4</sup>

**ANSWER 5**

A stepwise approach to Jessica's health issues is summarised in *Table 2*. Note that lifestyle intervention is always the core part of management.

**ANSWER 6**

Obesity exacerbates severity of PCOS.<sup>18</sup> It is important, therefore, to explain to Jessica that moderate weight loss of 5–10% will result in:

- improvement of menstrual regularity, improved ovulation and

Table 2. A stepwise approach to Jessica's health issues	
<b>Reproductive features</b>	<p><b>Irregular menstruation</b></p> <p>Low dose COC or cyclic progesterone to induce withdrawal bleed (2–3 monthly)</p> <p>Discuss potential risk for infertility and emphasise prevention of weight gain and weight loss 5–10% goal and confirm the value of early family initiation</p> <p>Metformin could be a potential option for restoration of menstrual regularity, and ovulation particularly if contraception is not desired or if she fails to lose weight with lifestyle interventions (start metformin at 500 mg of slow release daily to enhance GI tolerance and maximise the dose in weeks to months to reach 2 g/day)</p> <p><b>Hirsutism</b></p> <p>Laser therapy was discussed given the concern it caused for Jessica. Other options including the COC pill were also discussed.</p>
<b>Metabolic features</b>	<p><b>Obesity and cardiovascular risk factors</b></p> <p>Aim for weight loss of 5–10% of current body weight</p> <p>Refer to a dietician</p> <p>Encourage regular exercise</p> <p>Regularly test markers of glucose metabolism, and cardiovascular risk (generally 2 yearly) including:</p> <ul style="list-style-type: none"> <li>• 75 g OGTT</li> <li>• Fasting lipids</li> </ul> <p>Discuss importance of preconception screening for diabetes, and early antenatal screening for gestational diabetes</p>
<b>Psychosocial features</b>	<p>Screen for current features of depression or anxiety</p>

reduced pregnancy complications<sup>19</sup>

- risk reduction for development of impaired glucose tolerance and T2DM<sup>10</sup>
- improvement in cardiovascular risk factors including dyslipidaemia and hypertension<sup>2</sup>
- improvement in psychological health.<sup>2</sup>

### ANSWER 7

GDM is defined as glucose intolerance of variable severity with onset or first recognition during pregnancy.<sup>11</sup> It can be associated with increased risk of adverse pregnancy outcomes affecting both mother and fetus. History of PCOS is among the risk factors that necessitate early screening for GDM.<sup>11</sup>

A diagnosis of GDM is made if one or more of the following is present:<sup>11, 20</sup>

- Fasting glucose  $\geq 5.1$  mmol/L
- 1-hour glucose  $\geq 10.0$  mmol/L
- 2-hour glucose  $\geq 8.5$  mmol/L.

Given her screening results, Jessica is diagnosed with early GDM.

### Management<sup>20</sup>

Referral to an antenatal centre where multidisciplinary care is provided, including access to a dietician, diabetes educator and an endocrinologist, is recommended. The initial step is patient education on how to self-monitor pre- and postprandial blood glucose levels. Initial treatment should consist of lifestyle interventions including nutrition therapy and moderate daily exercise for 30 minutes or more.

Pharmacological therapy is recommended for women in whom lifestyle therapy is ineffective.

Regular weight monitoring and recommendations for healthy weight gain in pregnancy should also be discussed with Jessica. According to the 2009 Institute of Medicine recommendations, the total recommended weight gain for Jessica's BMI of 26 kg/m<sup>2</sup> is 7–11 kg.<sup>21</sup>

### ANSWER 8

Any blood glucose-lowering therapy being used during pregnancy will be stopped after delivery in women with GDM, unless overt diabetes is suspected. Women are screened for diabetes again at 6–12 weeks after delivery.<sup>20</sup> Jessica will have her blood glucose measured by midwives for the first 48–72 hours after birth to exclude ongoing hyperglycaemia, and thereafter her follow-up will be as explained above. Given that Jessica was diagnosed early in her pregnancy, before conventional pregnancy-related insulin resistance occurs, it is highly likely she will remain with impaired glucose intolerance postpartum and she will have a high risk of early progression to T2DM.

### ANSWER 9

Women with a previous history of GDM are at higher risk of developing diabetes in the future. Approximately 50% of these women develop diabetes within 10–20 years.<sup>22</sup> Such women, therefore, require lifelong screening for the development of diabetes

or pre-diabetes. Prevention of excess gestational weight gain and postpartum weight retention via lifestyle changes including healthy eating, regular exercise and maintaining a healthy weight is the key component to lowering this risk in the future.

As development of GDM is considered an additional risk factor to PCOS for development of diabetes, Jessica is considered to be at a higher risk and will require annual screening with OGTT.<sup>2</sup>

### ANSWER 10

There is 2–3-fold increase in the risk of GDM in women PCOS<sup>7</sup> and, therefore, assessment of BMI, blood pressure and an OGTT prior to conception is recommended in these women.<sup>12</sup> If no evidence of glycaemic abnormalities prior to conception, women with PCOS should undergo early screening for GDM with an OGTT at their first antenatal visit and this needs to be repeated at 24–28 weeks gestation if normal in early pregnancy.<sup>11</sup>

There is also approximately a 30% chance of a recurrent GDM in a subsequent pregnancy in women with a history of previous GDM.<sup>23</sup>

Given the combined increase in risk, Jessica should be advised that planning of future pregnancies is essential. Screening with 75 g OGTT preconception should be discussed with her. Contraception should be encouraged to avoid unplanned pregnancy and, in Jessica's case, the history of GDM should not affect the choice of contraceptive method.<sup>20</sup>

### REFERENCES

1. Ferriman D, Gallway JD. Clinical assessment of body hair growth in women. *J Clin Endocrin Metab* 1961;21:1440–47.
2. Teede HJ, Misso ML, Deeks AM, et al. Assessment and management of polycystic ovary syndrome: summary of an evidence-based guideline. *Med J Aust* 2011;195:S65–112.
3. Stepto NK, Cassar S, Joham AE, et al. Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemic–hyperinsulaemic clamp. *Hum Reprod* 2013;28:777–84.
4. Meyer C, McGrath BP, Teede HJ. Effects of medical therapy on insulin resistance and the cardiovascular system in polycystic ovary syndrome. *Diabetes Care* 2007;30:471–78.
5. Teede H, Deeks A, Moran L. Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. *BMC Med* 2010;8:41.
6. Moran LJ, Misso ML, Wild RA, Norman RJ. Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. *Hum Reprod Update* 2010;16:347–63.
7. Boomsma CM, Eijkemans MJ, Hughes EG, Visser GH, Fauser BC, Macklon NS. A meta-analysis of pregnancy outcomes in women with polycystic ovary syndrome. *Hum Reprod Update* 2006;12:673–83.
8. Bajuk Studen K, Jensterle Sever M, Pfeifer M. Cardiovascular risk and subclinical cardiovascular disease in polycystic ovary syndrome. *Front Horm Res* 2013;40:64–82.
9. Sacks FM, Bray GA, Carey VJ, et al. Comparison of weight-loss diets with different compositions of fat, protein and carbohydrates. *N Engl J Med* 2009;360:859–73.
10. National Health and Medical Research Council. Clinical practice guideline for the management of overweight and obesity in adults. Canberra: NHMRC, 2003. Available at [http://obesityconsortium.unimelb.edu.au/news\\_events/Report&Media/clinical\\_management\\_adults.pdf](http://obesityconsortium.unimelb.edu.au/news_events/Report&Media/clinical_management_adults.pdf) [Accessed 30 June 2014].

11. Nankervis A, McIntyre D, Moses R, et al. ADIPS consensus guidelines for the testing and diagnosis of gestational diabetes mellitus in Australia. Sydney: Australasian Diabetes in Pregnancy Society, 2013. p1–8. Available at [www.bhs.org.au/airapps/Services/au/org/bhs/govdoc/files/references/12042.pdf](http://www.bhs.org.au/airapps/Services/au/org/bhs/govdoc/files/references/12042.pdf) [Accessed 30 June 2014].
12. Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R, Welt CK. Diagnosis and treatment of polycystic ovary syndrome: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab* 2013;98:4565–92.
13. Ladson G, Dodson WC, Sweet SD, et al. The effects of metformin with lifestyle therapy in polycystic ovary syndrome: a randomized double-blind study. *Fertil Steril* 2011;95:1059–66.
14. Nieuwenhuis-Ruifrok AE, KuchenbeckerWK, HoekA, Middleton P, Norman RJ. Insulin-sensitizing drugs for weight loss in women of reproductive age who are overweight or obese: systematic review and meta-analysis. *Hum Reprod Update* 2009;15:57–68.
15. Tang T, Lord JM, Norman RJ, Yasmin E, Balen AH. Insulin-sensitising drugs (metformin, rosiglitazone, pioglitazone, D-chiroinositol) for women with polycystic ovary syndrome, oligo amenorrhoea and subfertility. *Cochrane Database Syst Rev* 2010;1:CD003053.
16. Nestler JE, Jakubowicz DJ, Evans WS, Pasquali R. Effects of metformin on spontaneous and clomiphene-induced ovulation in the polycystic ovary syndrome. *N Engl J Med* 1998;338:1876–80.
17. Boyle J, Teede HJ. Polycystic ovary syndrome – an update. *Aust Fam Physician* 2012;41:752–56.
18. Teede HJ, Joham AE, Paul E, et al. Longitudinal weight gain in women identified with polycystic ovary syndrome: results of an observational study in young women. *Obesity* 2013;21:1526–32.
19. Clark AM, Thornley B, Tomlinson L, et al. Weight loss in obese infertile women results in improvement in reproductive outcome for all forms of fertility treatment. *Human Reprod* 1998;13:1502–05.
20. Blumer I, Hadar E, Hadden DR, et al. Diabetes and pregnancy: an Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab* 2013;98:4227–49.
21. Institute of Medicine. Weight gain during pregnancy: reexamining the guidelines. Washington DC: National Academy of Sciences, 2009. Available at [www.iom.edu/pregnancyweightgain](http://www.iom.edu/pregnancyweightgain) [Accessed 14 August 2014].
22. Centres for Disease Control and Prevention: National diabetes fact sheet: general information and national estimates on diabetes in the United States, 2011. Atlanta: US Department of Health and Human Services, Centres for Disease Control and Prevention, 2011.
23. Moses RG. The recurrence rate of gestational diabetes mellitus in subsequent pregnancies. *Diabetes Care* 1996;19:1348–50.

### RESOURCES FOR PATIENTS AND DOCTORS

- The Jean Hailes Foundation, [www.jeanhailes.org.au](http://www.jeanhailes.org.au)
- The Jean Hailes Foundation. Management tool, <http://jeanhailes.org.au/health-professionals/tools/>

### Women's health

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 <http://gplearning.racgp.org.au>
- log into the *gplearning* website at <http://gplearning.racgp.org.au> and answer the following 10 multiple choice questions (MCQs) online
- 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 <http://gplearning.racgp.org.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.**

#### CASE 1 – JANE

Jane, aged 39 years, is a stay-at-home mum with a 2-year-old toddler. She had postnatal depression and was on antidepressant medication, which she ceased 6 months ago. She remains well. Jane and her husband are planning a second pregnancy and she is concerned about a repeat episode of depression during or after the pregnancy. She has a family history of depression.

#### QUESTION 1

Which of the following statements is the most correct regarding Jane's situation?

- Jane's risk for postnatal depression with a second pregnancy is no greater than the background population risk for postnatal depression.
- If Jane required an antidepressant during her second pregnancy she should be prescribed paroxetine.
- If Jane developed postnatal depression and wanted to continue breastfeeding, use of a selective serotonin reuptake inhibitor (SSRI) would be acceptable.
- SSRI use during pregnancy poses unacceptable risks to the fetus and should be avoided.
- The background risk of birth defects in the general population is 0.2–0.4%.

#### QUESTION 2

Jane, now 41 years, comes to see you with 5-week-old James. Her husband travels extensively for work and is unable to provide much support. She has been mainly on her own for the past 3 weeks. A teary Jane tells you that she feels flat, irritable, tired and is not sleeping well. She says she cannot cope with two children on her own. She thinks she may have postnatal depression again. Which statement is the most correct?

- Given her prior history of postnatal depression, Jane should be prescribed an SSRI immediately.
- A score of 10 or more for Jane using the Edinburgh Postnatal Depression Scale (EPDS) suggests possible depression.
- Jane has no risk factors for depression in her history.
- Non-medication strategies are not appropriate for Jane given her history and symptoms.
- Jane should be referred immediately to a perinatal psychiatrist.

#### CASE 2 – PENNY

Penny, a university student aged 20 years, has come to see you. She is dating a postgraduate student. When they try to have penetrative sex she feels extreme pain. They have never managed to have 'full sex' while dating. She had the same problem with her first 'real' boyfriend, leading to the break up of the relationship. She likes her current boyfriend and does not want to lose him.

#### QUESTION 3

Which of the following statements is correct with regard to obtaining a history from Penny?

- You can assume that Penny has had two relationships.
- Given her age, asking Penny questions about her medical and surgical history is not relevant.
- Asking Penny questions about her family's attitudes and beliefs towards sex may provide useful information regarding contributing factors to her current problem.
- Asking Penny whether her parent's relationship was physically affectionate is not useful.
- Past traumatic experiences are unlikely to have contributed to Penny's current problem.

#### QUESTION 4

Regarding Penny's situation, which of the following statements is the most correct?

- Given her history, Penny probably has secondary vaginismus.
- Vaginismus does not appear in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition (DSM-5), 2013.
- Precise details of the population prevalence of vaginismus are unknown, but studies suggest a prevalence of 0.5–1.7%.
- Penny should be referred for management of her 'spastic vaginal muscles', which may require surgery for stretching under anaesthesia.

**CASE 3 – LUCIA**

Lucia, a university student aged 19 years, presents to discuss her infrequent periods, weight gain and increasing facial and body hair. Her periods, which have always been irregular, are becoming more infrequent. She had four periods last year and none so far this year. She has never been sexually active. She has a strong family history of type 2 diabetes and cardiovascular disease. Her blood pressure is normal, her body mass index is 30 kg/m<sup>2</sup> and she has central adiposity with a waist circumference of 91 cm. She has mild hirsutism and otherwise looks normal.

**QUESTION 5**

Which statement describes the most likely diagnosis for Lucia?

- A. Polycystic ovary syndrome (PCOS)
- B. Hypothyroidism
- C. Cushing syndrome
- D. Late-onset congenital adrenal hyperplasia
- E. Androgen-secreting tumour

**QUESTION 6**

Which one of the following statements is the most correct regarding management options for women with PCOS?

- A. A multidisciplinary management approach is recommended.
- B. Management of psychosocial features is the key priority.
- C. Management of reproductive features is the key priority.
- D. Management of metabolic features is the key priority.
- E. Management priorities should be based on the patient's key concerns.

**CASE 4 – EMILY**

Emily, aged 26 years, presents in a distressed state. She attended a party in a city apartment last night and got a bit drunk. She met a man she liked, who suggested they go into a bedroom to talk. She recalls kissing the man but when he began to undress her she said she was not interested in sex. He ignored her wishes, became forceful and angry, tore her clothes off and had sexual intercourse with her. He then left the apartment. She was in tears and called her mother. They came to see you right away.

**QUESTION 7**

Which statement below most correctly outlines appropriate next steps you should take following Emily's disclosure of rape?

- A. Careful consideration should be given to the initial words offered to Emily as these words may have a significant impact on her.
- B. You should obtain a fuller history by gently asking her questions to further assess the situation.
- C. Offer support and empathy.

- D. You could refer Emily to the nearest sexual assault centre with a forensic facility.
- E. All of the above.

**QUESTION 8**

Emily reports that she is very concerned about 'catching HIV or some other disease'. She is also alarmed at the possibility of a pregnancy. Which statement below best describes how these concerns should be managed?

- A. The key consideration is prophylaxis for chlamydia, gonorrhoea and syphilis.
- B. Prevention of an unwanted pregnancy is the most important consideration.
- C. The most important consideration is management of viral risk.
- D. Management should be tailored to the patient's history, risk of sexually transmissible infection (STI) and the likelihood of them returning for adequate follow-up; following a sexual assault protocol, such as that offered by the Royal Prince Alfred Hospital, or your local sexual health clinic is recommended.
- E. Prophylaxis is required for chlamydia, gonorrhoea and syphilis, medication for pregnancy prevention, as well as management of viral risks; however follow-up is not required.

**CASE 5 – SALLY**

Sally, 29 years of age, attends your practice. She looks pale and gets teary and shaky when she tells you about her weekend. Sam, her partner came home late and was very drunk and disorderly after drinking with the boys. An argument ensued about his drinking, culminating in Sam pushing and slapping Sally. She fell down several times. It continued for 5–10 minutes. Sam later apologised and made her a cup of tea. She feels achy, tired and unwell, and cannot stop crying when on her own. Sally has bruising on her limbs and torso. She has not gone to work for the past two days.

**QUESTION 9**

Which of the following questions/statements is the best way of responding to Sally?

- A. Has anything like this happened before?
- B. Can you tell me more about your relationship with Sam?
- C. I always thought Sam was a nice guy.
- D. I can't believe he could do something like this.
- E. You need to leave Sam right now.

**QUESTION 10**

In assessing Sally's situation and formulating a plan to help her, which one of the statements below summarises the best action plan?

- A. Report the incident to the police.

- B. You should obtain additional history and ask Sally what she would like to do and provide her with resources to help her (details of local shelters and counselling services etc).
- C. Assume that Sally is safe.
- D. Break doctor–patient confidentiality by contacting Sam, whom you also see, to discuss the situation.
- E. None of the above.













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Unit 510 October 2014

## Diabetes and obesity

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## Diabetes and obesity

Unit 510 October 2014

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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

## ABOUT THIS ACTIVITY

The incidence of diabetes has doubled in the last two decades and in 2006–2007 diabetes accounted for 2.5% of all GP consultations in Australia.<sup>1</sup> Diabetes is predicted to be the seventh leading cause of death by 2030.<sup>2</sup> The global prevalence of obesity has also almost doubled since 1980.<sup>1</sup> Excess body weight is a major risk factor for non-communicable diseases,<sup>3</sup> including diabetes mellitus, cardiovascular disease, musculoskeletal disorders and certain cancers (eg endometrial, breast and colon).<sup>3</sup> This edition of *check* will consider the management of diabetes and obesity in general practice.

## LEARNING OUTCOMES

At the end of this activity, participants will be able to:

- describe the diagnosis and management of type 1 diabetes
- outline the management of type 2 diabetes
- describe the management of vision loss in people with type 2 diabetes
- discuss palliative care for patients with diabetes
- summarise the potential benefits of weight loss and exercise for people who are obese and/or have diabetes
- discuss the risks and benefits of laparoscopic adjustable gastric banding surgery.

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### REFERENCES

1. Australian Institute of Health and Welfare. Australia's health 2008. Cat. no. AUS 99. Canberra: AIHW. Available at [www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442453674](http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442453674) [Accessed 3 September 2014].
2. World Health Organization. Global status report on noncommunicable diseases 2010. Geneva: WHO, 2011. Available at [www.who.int/nmh/publications/ncd\\_report\\_full\\_en.pdf](http://www.who.int/nmh/publications/ncd_report_full_en.pdf) [Accessed 3 September 2014].
3. World Health Organisation. Obesity and overweight. Fact sheet N°311 [Updated August 2014]. Available at [www.who.int/mediacentre/factsheets/fs311/en](http://www.who.int/mediacentre/factsheets/fs311/en) [Accessed 3 September 2014].

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

ACEI	angiotensin converting enzyme inhibitor	EDIC	Epidemiology of Diabetes Interventions and Complications	OCT	optical coherence tomography
ADA	American Diabetes Association	ENDIT	European Nicotinamide Diabetes Intervention Trial	OGTT	oral glucose tolerance test
BMI	body mass index	GAD	glutamate decarboxylase	QoL	quality of life
COPD	chronic obstructive pulmonary disease	GLP-1	glucagon-like peptide-1	PBS	Pharmaceutical Benefits Scheme
CORE	Centre for Obesity Research and Education	HbA1c	glycated haemoglobin	SGLT2	sodium-glucose transporter 2
CrCl	creatinine clearance	HLA	human leukocyte antibody	T1DM	type 1 diabetes mellitus
DCCT	Diabetes Control and Complications Trial	IA2	tyrosine phosphatase	T2DM	type 2 diabetes mellitus
DMP	diabetic macular oedema	LAGB	laparoscopic adjustable gastric banding	TFT	thyroid function test
DPP4	dipeptidyl peptidase-4	LFT	liver function test	TGA	Therapeutics Goods Administration
DPT	Diabetes Prevention Trial	NHMRC	National Health and Medical Research Council	VEGF	vascular endothelial growth factor

**CASE 1**

**ANNIE HAS SIGNS OF DIABETES**

Annie is 15 years of age. She presents with a 3-week history of polyuria and polydipsia. Annie is obese and you are concerned that she has developed diabetes. You tell Annie that you suspect a diagnosis of diabetes mellitus. Her mother, who has accompanied Annie, asks if you think Annie has type 1 (T1DM) or type 2 diabetes mellitus (T2DM).

**QUESTION 1** 

What would you tell her?

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**QUESTION 2** 

What features of Annie's history are important?

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**FURTHER INFORMATION**

On further questioning, Annie reveals that she is pleased she lost about 5 kg recently 'without even trying'.

Annie's parents' families were originally from the Indian subcontinent and there is a strong family history of diabetes on both sides. Her mother was diagnosed with gestational

diabetes in her first pregnancy, with Annie's older brother. Her father and paternal grandmother were diagnosed with T2DM at the age of 45 years. Several other family members on Annie's father's side have T2DM, but Annie and her mother are unsure of the details. All family members are managed with either oral hypoglycaemic agents or diet, except for Annie's paternal grandmother who was commenced on subcutaneous insulin at the age of 70 years.

Annie has been overweight or obese since late childhood. This has been attributed to a combination of inappropriate food choices in the family home and lack of physical activity. This became more of an issue after the time of menarche, which occurred when Annie was 12 years of age. Annie's periods have been regular since that time. Annie's body mass index (BMI) is now greater than the 97th centile for her age, again raising your suspicions of T2DM, given Annie's family history. Neither acanthosis nigricans nor hirsutism was present.

**QUESTION 3** 

Which test is the most appropriate first-line investigation?

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**FURTHER INFORMATION**

Annie's random blood glucose level is 15 mmol/L and her blood ketones are 0.2 mmol/L.

**QUESTION 4** 

What is the appropriate next step?

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**FURTHER INFORMATION**

Annie was appropriately referred to the local hospital endocrinology service for assessment. On presentation to the emergency department, Annie's venous pH was normal at 7.34 (7.35–7.45) and her ketones were borderline positive at 0.6 mmol/L (normal <0.6 mmol/L). She was therefore not in diabetic ketoacidosis and was only mildly dehydrated. She was commenced on subcutaneous basal bolus insulin (1 unit/kg/day) with oral rehydration. Blood samples were sent for autoantibody testing, thyroid function tests (TFTs), screening for coeliac disease, liver function tests (LFTs) and cholesterol. All tests were normal except for those shown in *Table 1*. Measurement of C-peptide was not included as it is not part of the diagnostic workup for diabetes in childhood.<sup>1</sup>

Table 1. Abnormal blood test results	
Test	Result
GAD antibodies	20 U/mL (normal <5 U/mL)
HbA1c	10.5% (91.3 mmol/mol)
GAD, glutamate decarboxylase; HbA1c, glycated haemoglobin	

Annie and her parents were counselled that this was most probably T1DM and should be managed as such. Annie and her family received comprehensive education on diabetes aetiology and pathogenesis, insulin treatment, practical aspects of self-management and dietary advice. She was discharged 2 days later, when she was comfortable with self-administration of insulin, and was given a plan for regular monitoring of diabetes control at medical specialist appointments every 3–4 months.<sup>2</sup>

After commencing basal bolus insulin, Annie returns to you for review 3 weeks later. She is adjusting to the diagnosis of diabetes but is concerned that she is the only one in her immediate family who has to use insulin. She asks if she can take tablets instead of injections.

**QUESTION 5** 

Do oral hypoglycaemic agents have a role in the management of Annie's diabetes?

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**QUESTION 6** 

What advice/information would you provide to help Annie adjust to her diagnosis?

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**CASE 1 ANSWERS**

**ANSWER 1**

Statistically, people of Annie's age are more likely to develop T1DM than T2DM regardless of body size/weight and family history. The incidence of T1DM is increasing<sup>3</sup> and now affects 500,000 children worldwide.<sup>4</sup> T1DM accounts for >90% cases of childhood and adolescent diabetes in most western countries.<sup>5</sup> In Australia, 14–23 per 100,000 children (0–14 years) are diagnosed with T1DM annually.<sup>6,7</sup> Despite the increasing prevalence of overweight and obesity,<sup>8,9</sup> T2DM is still uncommon in the Australian paediatric group.<sup>10</sup>

There is no feature that can definitively discriminate between the two types of diabetes at diagnosis and, therefore, Annie must be assumed to have T1DM until proven otherwise, as misdiagnosis of T1DM may result in clinical deterioration within hours, which is potentially life-threatening.<sup>11,12</sup> Missed cases and delayed referral for insulin commencement are the most common reasons for diabetic ketoacidosis in patients newly diagnosed with T1DM,<sup>13,14</sup> which is a major cause for morbidity and mortality in this population.<sup>11,12</sup> The osmotic symptoms that Annie describes should increase your suspicion of T1DM.<sup>1</sup>

**ANSWER 2**

The following points are important:

- **Presenting features:** the classical osmotic symptoms of hyperglycaemia (polyuria, polydipsia, weight loss and fatigue) strongly favour a diagnosis of T1DM.<sup>1</sup>
- **Family history:** family history is not usually positive in a child with T1DM but a personal or family history of autoimmune conditions (thyroid disease, coeliac disease, vitiligo, pernicious anaemia, inflammatory bowel disease, rheumatological conditions) may increase the suspicion for a primary autoimmune disorder.<sup>15</sup> Family

history is often contributory in T2DM<sup>15</sup> and although it is important to ask about it, family history does not help to delineate between T1DM and T2DM diabetes in the first instance.

- **Features of the metabolic syndrome:** the presence of acanthosis nigricans in the neck creases, axillae, antecubital fossae, groin and inframammary areas is suggestive of insulin resistance but does not definitively confer a diagnosis of T2DM.<sup>15,16</sup> Other features, such as the presence of non-alcoholic fatty liver disease, hypertension or manifestations of polycystic ovarian syndrome, may also increase your suspicion of T2DM, but may also co-exist in an individual with T1DM.
- **Ethnicity:** ethnicity is important but does not supersede the clinical presentation. Aboriginal and Torres Strait Islander peoples, people from the Pacific Islands, Indian subcontinent and China are at higher risk of T2DM, compared with other groups<sup>15</sup> but even in these populations T1DM should still be the first consideration in childhood.

### ANSWER 3

In the primary care setting, if there is a suspicion of diabetes in a child, the first-line investigation should be a random blood glucose level on fingerprick point-of-care testing. As per the American Diabetes Association (ADA) position statement, a diagnosis of diabetes is made where there is a random blood glucose level of  $\geq 11.1$  mmol/L or a fasting plasma glucose of  $\geq 7.0$  mmol/L, with typical symptoms of hyperglycaemia,<sup>17</sup> which should then be confirmed by a laboratory sample. Note that the ADA diagnostic criteria are recognised worldwide. A fingerprick assessment of ketones using a point-of-care meter should also be done where available. Frequently, children present to the emergency department with typical symptoms of diabetes and are referred for outpatient phlebotomy, with no immediate follow-up of abnormal results. This is concerning as clinical deterioration of these children can lead to diabetic ketoacidosis, a potentially life-threatening situation. Indeed, up to one-third of children who present in diabetic ketoacidosis have had at least one medical contact in the week before presentation.<sup>18</sup>

The use of an oral glucose tolerance test (OGTT) or fasting blood glucose levels are generally not recommended as part of the diagnostic workup for suspected T1DM in childhood.<sup>1</sup> As second-line investigations, these should be reserved for a child with no osmotic symptoms and a normal blood sugar on random testing, when there is a high suspicion of T2DM. When an OGTT or fasting plasma glucose is requested, there should be immediate notification of abnormal results to the requesting physician, with same-day specialist referral for any positive results.

If the above results are equivocal, there should be discussion about further options with a paediatric endocrinologist, who is likely to advise referral for inpatient blood glucose monitoring given the nature of this presentation.

### ANSWER 4

If a random blood glucose level is diagnostic or strongly indicative of diabetes, same-day referral for specialist assessment is warranted,

as any delay in confirmation of the diagnosis may increase the risk of diabetic ketoacidosis and associated complications.<sup>13</sup> Additional investigations are unnecessary at this time, except to confirm the findings in a laboratory sample.

High levels of blood ketones are indicative of severe insulin deficiency.<sup>19</sup> Recall that ketones are a toxic by-product arising from the degradation of fat stores for energy in the absence of insulin<sup>20</sup> which can lead to the development of diabetic ketoacidosis, a potentially fatal medical emergency. At this time Annie's blood ketone level is within range.

### ANSWER 5

Annie's clinical history and laboratory results are suggestive of T1DM and therefore subcutaneous insulin is the appropriate first-line treatment for her.<sup>1</sup> Emphasis should be placed on the underlying diagnosis with its associated insulin deficiency, and insulin therapy should be optimised in the first instance. Insulin resistance in T1DM increases during adolescence<sup>21</sup> and may be a contributing factor to the suboptimal glycaemic control frequently seen in this population. If Annie's insulin requirement approaches 2 units/kg/day, this should be discussed with her endocrinologist as an apparently large insulin requirement may in fact reflect insulin omission (intentionally omitting or reducing the dose of insulin).<sup>22</sup>

In families where there is a strong history of T2DM, managed with either diet alone or oral hypoglycaemic agents, there may be some resistance to insulin therapy. Annie may feel unsupported in her home environment and appropriate education of her family members may be required. Tools are available to assess diabetes-related distress and depression.<sup>15</sup>

### ANSWER 6

It is important to emphasise to Annie that T1DM requires lifelong medical management with insulin therapy. Education and psychological support will be essential in helping Annie to adjust to the diagnosis. These should be provided in a culturally, developmentally and age-appropriate manner.<sup>19</sup> You should advise Annie to maintain regular contact with her diabetes healthcare team and ensure that she receives information about:<sup>19</sup>

- preventive interventions at key developmental stages: these interventions emphasise appropriate family involvement and support for Annie in managing her diabetes
- access to mental health professionals for psychological support
- flexible insulin therapy programs.

Education should also include nutritional/dietary and lifestyle advice, encouraging healthy lifelong eating habits, which includes advice about:<sup>19</sup>

- carbohydrate quantification and insulin-to-carbohydrate ratios
- the benefits of low-glycaemic index food choices
- avoiding high protein, low carbohydrate diets
- reducing intake of saturated fats and substitution of saturated fats with monounsaturated and polyunsaturated fats.

## REFERENCES

1. Global IDF/ISPAD Guideline for diabetes in childhood and adolescence. International Diabetes Federation 2011. Available at [www.idf.org/sites/default/files/Diabetes-in-Childhood-and-Adolescence-Guidelines.pdf](http://www.idf.org/sites/default/files/Diabetes-in-Childhood-and-Adolescence-Guidelines.pdf) [Accessed 9 July 2014].
2. Rewers M, Pihoker C, Donaghue K, Hanas R, Swift P, Klingensmith GJ. Assessment and monitoring of glycemic control in children and adolescents with diabetes. *Pediatric Diab* 2009;10 (Suppl 12):71–81.
3. Dabelea D. The accelerating epidemic of childhood diabetes. *Lancet* 2009;373:1999–2000.
4. Patterson C, Guariguata L, Dahlquist G, Soltesz G, Ogle G, Silink M. Diabetes in the young – a global view and worldwide estimates of numbers of children with type 1 diabetes. *Diabet Res Clin Pract* 2014;103:161–75.
5. Craig ME, Hattersley A, Donaghue KC. ISPAD Clinical Practice Consensus Guideline 2009 Compendium. Definition, epidemiology and classification of diabetes in children and adolescents. *Pediatr Diab* 2009;10(Suppl 12):3–12.
6. Chong JW, Craig ME, Cameron FJ, et al. Marked increase in type 1 diabetes mellitus incidence in children aged 0–14 yr in Victoria, Australia, from 1999–2002. *Pediatr Diab* 2007;8:67–73.
7. Haynes A, Bower C, Bulsara MK, Jones TW, Davis EA. Continued increase in the incidence of childhood Type 1 diabetes in a population-based Australian sample (1985–2002). *Diabetologia* 2004;47:866–70.
8. Olds TS, Tomkinson GR, Ferrar KE, Maher CA. Trends in the prevalence of childhood overweight and obesity in Australia between 1985 and 2008. *Int J Obes (Lond)* 2010;34:57–66.
9. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA* 2004;291:2847–50.
10. Ruhayel SD, James RA, Ehtisham S, Cameron FJ, Werther GA, Sabin MA. An observational study of type 2 diabetes within a large Australian tertiary hospital pediatric diabetes service. *Pediatr Diab* 2010;11:544–51.
11. Dunger DB, Sperling MA, Acerini CL, et al. European Society for Paediatric Endocrinology/Lawson Wilkins Pediatric Endocrine Society consensus statement on diabetic ketoacidosis in children and adolescents. *Pediatrics* 2004;113:e133–40.
12. O’Grady MJ, Delaney J, Jones TW, Davis EA. Standardised mortality is increased three-fold in a population-based sample of children and adolescents with type 1 diabetes. *Pediatric Diab* 2013;14:13–17.
13. Sabin MA CF, Cameron FJ, Werther GA. Type 1 diabetes: still the commonest form of diabetes in childhood. *Aust Fam Phys* 2009;38:695–97.
14. Sundaram PC, Day E, Kirk JM. Delayed diagnosis in type 1 diabetes mellitus. *Arch Dis Childhood* 2009;94:151–52.
15. The Royal Australian College of General Practitioners and Diabetes Australia. General practice management of type 2 diabetes – 2014–2015. Melbourne: RACGP and Diabetes Australia; 2014.
16. Brickman WJ, Huang J, Silverman BL, Metzger BE. Acanthosis nigricans identifies youth at high risk for metabolic abnormalities. *J Pediatr* 2010;156:87–92.
17. American Diabetes Association: Diagnosis and classification of diabetes mellitus. *Diab Care* 2014;37:S81–90.
18. Usher-Smith JA, Thompson MJ, Sharp SJ, Walter FM. Factors associated with the presence of diabetic ketoacidosis at diagnosis of diabetes in children and young adults: a systematic review. *BMJ* 2011;343:d4092.
19. Craig ME, Twigg, Donaghue KC, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidenced-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing; 2011. Available at [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/ext004\\_type1\\_diabetes\\_children\\_adolescents\\_adults.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ext004_type1_diabetes_children_adolescents_adults.pdf) [Accessed 25 July 2014].
20. Laffel L. Ketone bodies: a review of physiology, pathophysiology and application of monitoring to diabetes. *Diab Metab Res Rev* 1999;15:412–26.
21. Amiel SA, Sherwin RS, Simonson DC, Lauritano AA, Tamborlane WV. Impaired insulin action in puberty. A contributing factor to poor glycemic control in adolescents with diabetes. *N Engl J Med* 1986;315:215–19.
22. Wiegand S, Raile K, Reinehr T, et al. Daily insulin requirement of children and adolescents with type 1 diabetes: effect of age, gender, body mass index and mode of therapy. *Europ J Endocrinol* 2008;158:543–49.

## RESOURCES FOR PATIENTS

- The Better Health Channel provides a fact sheet on type 1 diabetes, [www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes\\_Type\\_1\\_or\\_juvenile\\_diabetes](http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes_Type_1_or_juvenile_diabetes) [Accessed 27 August 2014].
- NPS Medicinewise provides information on diabetes for patients, [www.nps.org.au/](http://www.nps.org.au/)

## RESOURCES FOR DOCTORS

- Craig ME, Twigg, Donaghue KC, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidenced-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing, 2011, [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/ext004\\_type1\\_diabetes\\_children\\_adolescents\\_adults.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ext004_type1_diabetes_children_adolescents_adults.pdf) [Accessed 25 July 2014].
- NPS Medicinewise provides information on diabetes for health professionals, [www.nps.org.au](http://www.nps.org.au)

**CASE 2**

**JACK HAS BEEN WETTING HIS BED**

Jack is 7 years of age and his mother, Sarah, has brought him to see you as he has recently started wetting his bed at night. She thinks the bedwetting is associated with Jack being thirsty all the time for the past few weeks and needing to drink lots of water. She is also worried because Jack has lost weight.

**QUESTION 1** 

What differential diagnoses would you consider?

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**QUESTION 2** 

Sarah asks if Jack's diabetes could have been prevented. How would you respond?

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**QUESTION 3** 

What is the likelihood of Jack's siblings developing diabetes?

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**FURTHER INFORMATION**

After confirming the diagnosis of type 1 diabetes mellitus (T1DM) and starting insulin therapy, Sarah calls you to say 'Jack's diabetes has gone away'.

**QUESTION 4** 

What would you say to her?

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**QUESTION 5** 

What are the long-term consequences of T1DM? What routine screening for complications is recommended in T1DM?

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**QUESTION 6** 

Jack's mother asks you if he will lead a 'normal' life.

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## CASE 2 ANSWERS

## ANSWER 1

Jack is a child who presents with a classical history of increasing polyuria, polydipsia and weight loss over a few weeks. This presentation should not usually pose a diagnostic difficulty for T1DM.<sup>1</sup> As in Case 1, if there is a suspicion of diabetes in a child in the primary care setting, the first-line investigation should be a random blood glucose level on fingerprick point-of-care testing and, where available, a fingerprick assessment of ketones. If a random blood glucose level is diagnostic or strongly indicative of diabetes, same-day referral for specialist assessment, management and initiation of therapy is warranted, as any delay in confirmation of the diagnosis may increase the risk of diabetic ketoacidosis and associated complications.<sup>2</sup>

It is important to be aware of the various non-emergency presentations of diabetes. As in Jack's case, a recent onset of secondary enuresis and polyuria in a previously toilet-trained child should raise suspicions of T1DM. However, it could be misdiagnosed as a urinary tract infection<sup>1</sup> or the result of excessive fluid ingestion. Note that it is possible to have a urinary tract infection in addition to T1DM. Similarly, polydipsia may be thought to be psychogenic.<sup>1</sup> Vomiting, if present, may be misdiagnosed as gastroenteritis or sepsis.<sup>1</sup> Chronic weight loss or failure to gain weight in a growing child is worrying, as are recurrent skin infections or vaginal candidiasis, especially in prepubertal girls. It is always important to enquire about irritability and decreasing performance at school.

Consider differential diagnoses carefully as some situations may result in a late diagnosis of diabetic ketoacidosis. For example, the hyperventilation of ketoacidosis (Kausmaul breathing) may be misdiagnosed as pneumonia or asthma, although cough and breathlessness distinguish these conditions from diabetic ketoacidosis. In addition, the abdominal pain associated with ketoacidosis may simulate an acute abdomen and lead to referral to a surgeon.<sup>1</sup> An Australian study found that a population awareness campaign was effective in reducing the number of children who presented in diabetic ketoacidosis by 64%. In the intervention group, all childcare centres, schools and doctors' offices in the region received an educational poster illustrating four common signs of diabetes, including weight loss, increased thirst, increasing urination and fatigue. The early recognition of symptoms, prompted diagnosis and treatment helped avoid diabetic ketoacidosis in the majority of children.<sup>3</sup>

We know that T1DM is a chronic autoimmune disease in the majority of patients and accounts for over 90% of childhood and adolescent diabetes in Australia.<sup>4</sup> T-cell-mediated destruction of the pancreatic beta cells leads to insulin deficiency.<sup>1,5</sup> T1DM includes those cases attributable to an idiopathic cause for which neither an aetiology nor a pathogenesis is known. Regardless of its aetiology, the clinical staging of T1DM reflects the new concept that diabetes progresses

through several clinical stages during its natural history. These stages are characterised by preclinical, clinical, partial remission and chronic phases;<sup>1,6</sup> that is, normoglycaemia, preclinical diabetes (impaired glucose regulation, which includes impaired fasting glycaemia or impaired glucose tolerance) and then overt presentation of T1DM.<sup>1,7</sup> The important point to note is that individuals may move from stage to stage in either direction, and understanding this time continuum of presentation can aid diagnosis. In patients with classical T1DM, such as Jack, reversion to more normal glucose levels is not possible without insulin therapy.

## ANSWER 2

Current guidelines do not recommend any interventions for use in clinical practice to delay or prevent T1DM as, to date, all clinical trials attempting to prevent or delay the onset of T1DM in those at high risk have been unsuccessful.<sup>1</sup> The most important of these intervention studies were the European Nicotinamide Diabetes Intervention Trial (ENDIT), which showed that nicotinamide did not delay or prevent the onset of T1DM in high-risk first-degree relatives,<sup>8</sup> and the Diabetes Prevention Trial (DPT), in which low-dose subcutaneous insulin therapy did not delay or prevent the onset of clinical diabetes in first-degree relatives.<sup>9</sup> Screening of any population or intervention in the preclinical phase should not occur outside the context of defined clinical studies and research settings.<sup>10</sup> Individuals who test positive for genetic or immunological markers of T1DM should have access to appropriate counselling and to centres participating in intervention and other defined studies.

In T1DM, progressive destruction of beta cells occurs at a variable rate and diabetes becomes clinically symptomatic when approximately 90% of the pancreatic beta cells are destroyed.<sup>11</sup> Insulin deficiency then manifests clinically as blood glucose levels rise to pathological levels. It is important to explain to the patient and the family our current understanding about the multifactorial pathogenesis of diabetes whereby T1DM results from an interplay between genetic predisposition and environmental factors.<sup>1</sup> Many parents experience feelings of guilt, assuming that T1DM occurred as a result of allowing their child to have a high-sugar diet. These misconceptions need to be addressed early on.

## ANSWER 3

Families need to be aware that there is no recognisable pattern of T1DM inheritance. Susceptibility to autoimmune T1DM is determined by the interaction between multiple genes, with HLA genes having the strongest known association. These genetic markers can confer either an increased or decreased risk.<sup>1</sup> However, several studies have provided valuable insight in further characterising aspects of the disease. The overall risk for first-degree relatives in singleton families is 4–6%<sup>12</sup> and rises to 15% if two first-degree relatives are affected. Alternatively, concordance for T1DM between monozygotic twins is around 36%.<sup>13</sup> Hence, the genetic load on risk is relatively low. Interestingly, T1DM is transmitted more frequently to the offspring of diabetic men than those of diabetic women (6.1%, compared with 1.3% of offspring).<sup>14</sup> In summary, Jack's siblings have a 95%

chance of not developing T1DM. However, as with everyone else in the population, their chances of developing type 2 diabetes mellitus (T2DM) diabetes at some time in their lives is greater than 1 in 3.<sup>15</sup> Although we do not fully understand the multifactorial pathological processes leading to T1DM, we understand that these processes can start several months to years before clinical symptoms are manifested.<sup>1</sup> The most important information to convey about sibling risk is that although parents would not be able to alter the course of disease progression, if they notice any early symptoms, Jack's siblings should be investigated early and referred to a specialist diabetes centre for management, as discussed earlier. Prospective follow-up of high-risk individuals shows that diagnosis of T1DM can be made in asymptomatic individuals in the majority of cases, as shown in the DPT: when high-risk individuals were followed up, 73% of participants who were diagnosed with diabetes were asymptomatic.<sup>9</sup>

The onset of the disease is predictable, especially in the relatives of affected individuals, using a combination of auto-antibody measurements, glucose tolerance testing and genotyping.<sup>16</sup> The parameters currently helping to define the preclinical phase include the islet cell autoantibodies – glutamic acid decarboxylase (GAD) autoantibodies, IA2 (tyrosine phosphatase) autoantibodies, insulin autoantibodies – and human leukocyte antibody (HLA) typing.<sup>1</sup> Relatives with at least two of the autoantibodies were found to have a risk of 39% (95% CI, 27–52) and 68% (95% CI, 52–84) of developing diabetes within 3 and 5 years, respectively. For those with all three autoantibodies the risk of developing diabetes within 5 years was estimated to be 100%.<sup>17</sup> These findings suggest that the presence of two or more of the autoantibodies (IAA, GAAs and ICA512bdcAAs) is predictive of T1DM developing in relatives of affected individual.<sup>17</sup>

We also know that environmental triggers (chemical and/or viral) that start the process of autoimmune destruction of the pancreatic beta cells are yet to be fully described.<sup>1</sup> Congenital rubella<sup>18</sup> and other potential environmental triggers like enteroviral infections<sup>19</sup> (particularly coxsackie and ECHO viruses<sup>20,21</sup>), casein/cow's milk proteins<sup>22</sup> and gluten<sup>23</sup> have all been implicated. There is an international race on with trials that are investigating potential triggers, describing seasonal variation and researching protective factors in an attempt to understand the multifactorial nature of T1DM.

#### ANSWER 4

In approximately 80% of children and adolescents, insulin requirements decrease transiently following initiation of treatment.<sup>24</sup> This is known as the partial remission or honeymoon phase.<sup>1,24</sup> Most studies define a partial remission phase when the patient requires less than 0.5 units/kg/day of insulin and has an HbA1c <7%.<sup>24</sup> The honeymoon phase can commence within days or weeks of starting insulin therapy and may last for weeks to months.<sup>10</sup> During this phase, blood glucose levels are frequently stable within the normal range, despite fluctuations in diet and exercise, and insulin therapy may even be ceased.

Patients and families should be advised of the transient nature of the honeymoon period, to avoid the false hope that the diabetes has spontaneously gone into remission.<sup>10</sup> There is no evidence that any intervention has any effect on the length of the honeymoon period.<sup>25,26</sup> As the chronic phase of the lifelong dependence on insulin gradually occurs, it is important to support the patient and family as the only treatment for managing T1DM is insulin. There is no alternative and insulin is essential for survival.

#### ANSWER 5

Death of people with T1DM is usually due to diabetes-related complications.<sup>1</sup> The main long-term microvascular complications of diabetes can cause blindness due to diabetic retinopathy, renal failure due to diabetic nephropathy, and diabetic neuropathy which can be both peripheral and autonomic. Hypertension, dyslipidaemia and smoking influence the development of both microvascular and macrovascular complications.

Current Australian guidelines for routine screening or monitoring for complications in patients with T1DM are shown in *Table 1*.<sup>1</sup>

**Table 1. NHMRC guidelines for screening and monitoring for complications in T1DM<sup>1</sup>**

Complication	Commence screening	Monitoring frequency
Retinopathy	After 2 years duration in adolescents and adults, and after 5 years duration or from age 9 in children	Every second year or annually for select groups (eg high-risk groups)
Nephropathy	After 2 years duration in adolescents and adults, and after 5 years duration or from age 9 in children	Annually
Neuropathy	Annually	Annually
Lipids	At diagnosis if there is a family history or from 12 years	Every 5 years until puberty and then annually
Blood pressure	At diagnosis	At least annually
Macrovascular disease	In adulthood	At least annually

#### ANSWER 6

Some of the fundamentals of successful diabetes management, which can help patients lead a normal, healthy life, are discussed below.

T1DM is a chronic disease among children and adolescents and its diagnosis and management usually involves a significant burden on the patient and their families, as they must change various aspects of their lifestyle to fulfil the demands of treatment.

Clinicians need to be aware that psychological disorders commonly co-exist in those with T1DM and that validated screening tools for psychological disorders in T1DM are available.<sup>1</sup> They should be able to provide appropriate support to the patient and family for a healthy

adjustment to the diagnosis of T1DM or referral to other providers as necessary.<sup>27</sup> Psychological interventions have been shown to improve psychosocial outcomes and have an effect on HbA1c reduction, although modest. A pooled analysis of 10 studies found a 0.5% reduction in HbA1c following psychological interventions.<sup>1</sup> Although there was significant variability in these reports on the benefits of psychological interventions on metabolic control, distress was significantly reduced. Results of a 2012 systematic review reported that generic quality of life (QoL) of children and adolescents with T1DM was not impaired when compared with healthy peers, despite having to live with a demanding treatment regime. Nevertheless, disease-specific QoL problems such as a negative impact of diabetes on daily functioning and diabetes-related worries were present.<sup>27</sup>

Diabetes education and access to care by a multidisciplinary team trained in childhood diabetes is essential.<sup>7</sup> Education provision should be adapted to each individual's age, maturity, stage of diabetes, lifestyle and culture. Any insulin regimen should be considered in the wider context of a total diabetes management package, which must include dietary management, exercise and physical activity, blood glucose monitoring, initial and ongoing education, regular medical follow-up and psychological care.<sup>7</sup>

Intensive management (including multiple daily injections or pump therapy, education, intensive monitoring and psychosocial support) of T1DM in adolescents improves metabolic control and reduces the risk of microvascular complications.<sup>28</sup> However, the long-term benefits of intensifying management need to be weighed against the risks of frequency and severity of hypoglycaemia. This is because severe hypoglycaemia has been associated with lower verbal and full-scale intelligence quotient scores; attention, processing speed and executive skills were mostly affected in children with younger-onset of diabetes.<sup>29</sup> To this end, current guidelines recommend making every effort to achieve glycaemic targets to help minimise the potential impact of diabetes on cognitive function. The guidelines also recommend minimising acute episodes of hypoglycaemia and hyperglycaemia to help maintain optimal cognitive performance.<sup>1</sup>

The Diabetes Control and Complications Trial (DCCT) confirmed the benefit of maintaining near normal glycaemia in reducing the development and progression of diabetic microvascular complications in adolescents and adults.<sup>30,31</sup> The Epidemiology of Diabetes Interventions and Complications (EDIC) study reported that people with T1DM (aged 13–39 years at baseline) receiving intensive diabetes treatment had decreased progression of intima–media thickness, a marker for coronary and cerebrovascular disease, compared with patients receiving conventional therapy, over the mean follow-up period of 6.5 years.<sup>32</sup> In older children and adolescents, the target HbA1c should be <7.5%.<sup>33</sup>

In summary, T1DM in childhood imposes a number of psychological stresses on both the child and the family. However, open and frequent communication will help everyone to continue to work towards the primary goals of treatment, which include maintaining near-normal glycaemia through intensive insulin therapy, avoiding acute and long-term complications through regular screening, while balancing these needs to maintain as close to a normal, fulfilled life as possible

for the person with diabetes. General practice management plans and team care arrangements could also be considered to help improve clinical outcomes for patients.

## REFERENCES

1. Craig ME, Twigg, Donaghue KC, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidenced-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing; 2011. Available at [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/ext004\\_type1\\_diabetes\\_children\\_adolescents\\_adults.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ext004_type1_diabetes_children_adolescents_adults.pdf) [Accessed 25 July 2014].
2. Dunger DB, Sperling MA, Acerini CL, et al. European Society for Paediatric Endocrinology/Lawson Wilkins Pediatric Endocrine Society consensus statement on diabetic ketoacidosis in children and adolescents. *Pediatrics* 2004;113:e133–40.
3. King BR, Howard NJ, Verge CF, et al. A diabetes awareness campaign prevents diabetic ketoacidosis in children at their initial presentation with type 1 diabetes. *Paediatr Diabetes* 2012;13:647–51.
4. Craig ME, Hattersley A, Donaghue KC. ISPAD Clinical Practice Consensus Guideline 2009 Compendium. Definition, epidemiology and classification of diabetes in children and adolescents. *Pediatr Diabetes* 2009;10(Suppl 12):3–12.
5. World Health Organization. Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications. Report of a WHO Consultation. Part 1: Diagnosis and Classification. WHO/NCD/NCS/99.2. Geneva:WHO; 1999. Available at [http://whqlibdoc.who.int/hq/1999/who\\_ncd\\_ncs\\_99.2.pdf](http://whqlibdoc.who.int/hq/1999/who_ncd_ncs_99.2.pdf) [Accessed 25 July 2014].
6. Kuzuya T, Matsuda A. Classification of diabetes on the basis of etiologies versus degree of insulin deficiency. *Diabetes Care* 1997;20:219–20.
7. International Diabetes Federation. Global IDF/ISPAD Guideline for Diabetes in Children and Adolescence. Brussels: IDF; 2011. Available at [www.ispad.org/sites/default/files/resources/files/idf-ispad\\_diabetes\\_in\\_childhood\\_and\\_adolescence\\_guidelines\\_2011\\_0.pdf](http://www.ispad.org/sites/default/files/resources/files/idf-ispad_diabetes_in_childhood_and_adolescence_guidelines_2011_0.pdf) [Accessed 25 July 2014].
8. Gale EA, Bingley PJ, Emmett CL, Gillier T. European Nicotinamide Diabetes Intervention Trial (ENDIT) Group: European Nicotinamide Diabetes Intervention Trial (ENDIT): a randomised controlled trial of intervention before the onset of type 1 diabetes. *Lancet* 2004;363:925–31.
9. Diabetes Prevention Trial-Type 1 Diabetes Study Group: Effects of insulin in relatives of patients with type 1 diabetes mellitus. *N Eng J Med* 2002;346:1685–91.
10. Couper J, Donaghue KC. ISPAD Clinical Practice Consensus Guidelines 2009 Compendium: phases of diabetes in children and adolescents. *Pediatr Diabetes* 2009;10:13–16.
11. Gepts W. Pathologic anatomy of the pancreas in juvenile diabetes mellitus. *Diabetes* 1965;14:619–33.
12. Lorenzen T, Pociot F, Hougaard P, Nerup J. Long-term risk of IDDM in first-degree relatives of patients with IDDM. *Diabetologia* 1994;37:321–27.
13. Olmos P, A'Hern R, Heaton DA, et al. The significance of the concordance rate for type 1 (insulin-dependent) diabetes in identical twins. *Diabetologia* 1998;31:747–50.
14. Warram JH, Krolewski AS, Gottlieb MS, Kahn CR: Differences in risk of insulin-dependent diabetes in offspring of diabetic mothers and diabetic fathers. *N Eng J Med* 1984;311:149–52.
15. Magliano DJ, Shaw JE, Shortreed SM et al. Lifetime risk and projected population prevalence of diabetes. *Diabetologia* 2008;51:2179–86.
16. Kulmala P, Savola K, Reijonen H, et al. Genetic markers, humoral autoimmunity, and prediction of type 1 diabetes in siblings of affected children. Childhood Diabetes in Finland Study Group. *Diabetes* 2000;49:48–58.
17. Verge CF, Gianani R, Kawasaki E, et al. Prediction of type I diabetes in first-degree relatives using a combination of insulin, GAD and ICA512bdc/IA-2 autoantibodies. *Diabetes* 1999;48:926–33.

18. McIntosh ED, Menser MA. A fifty-year follow-up of congenital rubella. *Lancet* 1992;340:414–15.
19. Hyoty H, Hiltunen M, Knip M et al. A prospective study of the role of coxsackie B and other enterovirus infections in the pathogenesis of IDDM. Childhood Diabetes in Finland (DiMe) Study Group. *Diabetes* 1995;44:652–57.
20. Yeung WCG, Rawlinson WD, Craig ME. Enterovirus infection and type 2 diabetes mellitus: systematic review and meta-analysis of observational molecular studies. *BMJ* 2011;342:d35 Doi:10.1136/bmj.d35.
21. Schulte BM, Lanke KHW, Piganelli JD et al. Cytokine and chemokine production by human pancreatic islets upon enterovirus infection. *Diabetes* 2012;61:2030–36.
22. Akerblom HK, Virtanen SM, Ilonen J et al. Dietary manipulation of beta cell autoimmunity in infants at increased risk of type 1 diabetes: a pilot study. *Diabetologia* 2005;48:829–37.
23. Norris JM, Barriga K, Klingensmith G et al. Timing of initial cereal exposure in infancy and risk of islet autoimmunity. *JAMA* 2003;290:1713–1720.
24. Lombardo F, Valenzise M, Wasniewska M, et al. Two-year prospective evaluation of the factors affecting honeymoon frequency and duration in children with insulin dependent diabetes mellitus: the key-role of age at diagnosis. *Diabetes Nutr Metabol* 2002;15:246–51.
25. Neylon OM, White M, O'Connell MA, Cameron FJ. Insulin-dose-adjusted HbA1c-defined partial remission phase in a paediatric population – when is the honeymoon over? *Diabet Med* 2013;30:627–28.
26. Ludvigsson J. Immune intervention at diagnosis – should we treat children to preserve beta-cell function? *Pediatr Diabetes* 2007;8(Suppl 6):34–39.
27. Nieuwesteeg A, Pouwer F, van der Kamp R, van bakel H, Aanstoot HJ, Hartman E. Quality of life of children with type 1 diabetes: a systematic review. *Curr Diabetes Rev* 2012;8:434–43.
28. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Eng J Med* 1993;329:977–86.
29. Northam EA, Anderson PJ, Jacobs R, Hughes M, Warne GL, Werther GA. Neuropsychological profiles of children with type 1 diabetes 6 years after disease onset. *Diabetes Care* 2001;24:1541–46.
30. Australasian Paediatric Endocrine Group for the Department of Health and Ageing. Clinical practice guidelines: type 1 diabetes in children and adolescents. Canberra: Commonwealth of Australia; 2005. Available at [www.chw.edu.au/prof/services/endocrinology/apeg/apeg\\_handbook\\_final.pdf](http://www.chw.edu.au/prof/services/endocrinology/apeg/apeg_handbook_final.pdf) [Accessed 25 July 2014].
31. The Diabetes Control and Complications Trial Research Group. Effect of intensive diabetes treatment on the development and progression of long-term complications in adolescents with insulin-dependent diabetes mellitus: Diabetes Control and Complications Trial. *J Pediatr* 1994;125:177–88.
32. Nathan DM, Lachin J, Cleary P, et al. Intensive diabetes therapy and carotid intima–media thickness in type 1 diabetes mellitus. *N Eng J Med* 2003;348:2294–303.
33. National Institute for Health and Care Excellence. Type 1 diabetes: diagnosis and management of type 1 diabetes in children, young people. Key priorities for implementation: children and young people. NICE Guidelines [CG15]; 2014. Available at: [www.nice.org.uk/guidance/CG15/chapter/Key-priorities-for-implementation-children-and-young-people](http://www.nice.org.uk/guidance/CG15/chapter/Key-priorities-for-implementation-children-and-young-people) [Accessed 25 July 2014].

## RESOURCES FOR PATIENTS

- The Better Health Channel provides a fact sheet on type 1 diabetes, [www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes\\_Type\\_1\\_or\\_juvenile\\_diabetes](http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes_Type_1_or_juvenile_diabetes) [Accessed 27 August 2014].
- NPS Medicinewise provides information on diabetes for patients, [www.nps.org.au](http://www.nps.org.au)

## RESOURCES FOR DOCTORS

- Craig ME, Twigg, Donaghue KC, et al. for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidenced-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing, 2011, [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/ext004\\_type1\\_diabetes\\_children\\_adolescents\\_adults.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/ext004_type1_diabetes_children_adolescents_adults.pdf) [Accessed 25 July 2014].
- NPS Medicinewise provides information on diabetes for health professionals, [www.nps.org.au](http://www.nps.org.au)

**CASE 3**

**ERICA IS STRUGGLING WITH BLOOD SUGAR CONTROL AND HER WEIGHT**

Erica, a retired schoolteacher aged 62 years, was diagnosed with type 2 diabetes (T2DM) 3 years ago. She has been struggling with glycaemic control and her weight. At her last visit she stated that her 'sugars' must be bad as she is up all night urinating. She is on metformin slow release 2 g/day and gliclazide slow release 120 mg at night. Her last HbA1c was 7.6% (60 mmol/mol) 1 month ago. She has come to see you today for her diabetes review and states that her main concerns are her weight and frequent urination.

**QUESTION 1** 

What clinical possibilities need to be considered at this time?

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**QUESTION 2**  

Does ethnicity affect the classification of her obesity?

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**FURTHER INFORMATION**

Erica is often hungry in the mornings and therefore eats a large breakfast. She has gained weight since she started gliclazide. You advise Erica that she may have some hypoglycaemia leading to compensatory 'defensive snacking', which could be contributing to her weight gain. Erica does not self-monitor her glucose levels.

**QUESTION 3** 

What are the current recommendations for self-monitoring of blood glucose for patients using oral medications?

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**QUESTION 4** 

Which glucose-lowering agents commonly cause hypoglycaemia? Which agents have low rates of hypoglycaemia?

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**FURTHER INFORMATION**

Erica would like to lose some weight and would like to know your thoughts on this. Her body mass index (BMI) is 34 kg/m<sup>2</sup> and her waist circumference is 90 cm.



**ANSWER 4**

Insulin and sulphonylureas can cause hypoglycaemia. Of the sulphonylureas, gliclazide and glipizide are least likely to cause hypoglycaemia.<sup>1</sup> Other agents including metformin, acarbose, glitazones, glucagon-like peptide-1 (GLP-1)-mimetics and dipeptidyl peptidase-4 (DPP4) inhibitors will not cause hypoglycaemia when used as monotherapy.<sup>1</sup>

The risks of hypoglycemia may escalate when dual or triple combination glucose-lowering therapy is used.

**ANSWER 5**

A weight management plan should incorporate general advice about weight loss and physical activity and provide dietary advice. Weight management has been shown to be more difficult in people with T2DM.<sup>4-6</sup>

**Weight loss**

Weight loss in people with T2DM often results in improved glycaemic control, blood pressure and lipid profiles. A sustained weight reduction of about 5 kg has been associated with a reduction in HbA1c of 0.5–1%. In adults with a BMI <35 kg/m<sup>2</sup> and dysglycaemic states or hypertension, weight loss of at least 2–3 kg achieved with lifestyle interventions may result in clinically meaningful systolic blood pressure reductions (an average of 4.5 mmHg systolic and 3–3.5 mmHg diastolic).<sup>2</sup> Any level of weight loss should be encouraged and even losses of 5–10% will improve glycaemic control.<sup>1,7,8</sup>

**Physical activity**

The greatest health benefits attributable to physical activity are seen in those who change from doing no physical activity, or very little, to doing more.<sup>9</sup> Table 1 shows the different intensities of physical activity, which is useful information that could be discussed with patients.

Table 1. Levels of intensity of physical activity <sup>2</sup>		
Intensity	Description	Examples
Sedentary	Activities that involve sitting or lying down, with little energy expenditure	Occupational (eg sitting at work) Leisure (eg watching TV, reading, sewing, computer use for games, social networking) Transport (eg sitting in a car, train, bus or tram)
Light	Activities that require standing up and moving around in the home, workplace or community	Housework (eg hanging out washing, ironing, dusting) Working at a standing workstation
Moderate	Activities are at an intensity that requires some effort, but allow a conversation to be held	Brisk walking Gentle swimming Social tennis
Vigorous	Activities that lead to harder breathing or puffing and panting (depending on fitness)	Aerobics Jogging Some competitive sports

Any commencement of increased activity should be supported and reinforced by clinicians. Health benefits are achieved with 150–300 minutes of moderate-intensity activity or 75–150 minutes of vigorous activity (or a combination of moderate-intensity and vigorous activity), accumulated over a week.<sup>9</sup> Current evidence suggests that physical activity has little effect on weight loss unless it is combined with a reduced energy intake.<sup>10-12</sup> Even so, increased physical activity has been associated with a range of health benefits, including improved cardiovascular disease risk factors, even in the absence of weight loss.<sup>11,12</sup> Note that initial weight gain, due to an increase in muscle mass, has been associated with the commencement of muscle strengthening exercises.<sup>2</sup>

**Dietary advice**

Aiming for a reduction of 2000–2500 kilojoules (478–598 calories or 10%) in total daily energy intake per day should result in a weight loss of about 0.5 kg per week.<sup>1</sup>

Practical advice in constructing a weight management plan for adults who are obese can be found in the National Health and Medical Research Council (NHMRC) *Summary guide for the management of overweight and obesity in primary care 2013*,<sup>13</sup> which is based on the 5As approach (ask and assess, advise, assist, arrange).

**Ask and assess**

- Measure waist circumference and calculate BMI.
- Discuss readiness to change lifestyle behaviours as well as other psychological factors, such as comorbid depression, using primary practice tools such as *The Patient Health Questionnaire-2* (PHQ2) and *Problem Areas in Diabetes Questionnaire* (PAID).

**Advise**

- Convey the message that even small amounts of weight loss improve health and wellbeing.

**Assist**

- Use multicomponent approaches as these work better than single interventions.
- Refer appropriately to assist people to make lifestyle changes or for further intervention.

**Arrange**

- Support a self-management approach and provide ongoing monitoring.
- Use a multidisciplinary approach to support.

**ANSWER 6**

In a meta-analysis of 27 randomised controlled trials of about 11,200 participants, the addition of sulphonylureas and thiazolidinediones to maximal doses of metformin was associated with weight gain of 2.06 kg and 2.08 kg respectively. No weight change was reported with DPP4 inhibitors, whereas GLP1 agonists were associated with weight loss of 0.74 kg. All of the drugs had a similar effect on glycaemic control.<sup>14</sup> In a systematic review of sodium-glucose transporter 2 (SGLT2) receptor inhibitors available on the Pharmaceutical Benefits Scheme (PBS), dapagliflozin was associated

with weight loss of 1.81 kg and canagliflozin with a loss of 2.3 kg compared to placebo.<sup>3</sup>

There is no correct answer regarding the use of diabetes medications for Erica. If she can use lifestyle interventions to achieve weight loss, this may lead to improvements in her glycaemic control and she may not need to increase the doses of any of her medications or to use alternative or additional medications. Additionally, depending on her weight loss, her medication needs may decrease.

Diabetes often requires combination therapy to allow patients to remain at their glycaemia targets as the disease progresses. Metformin remains the drug of first choice for initial drug therapy in T2DM,<sup>1,15</sup> but decisions regarding which drug to add next and combination therapy are hampered by a lack of long-term, high-quality outcome data, particularly for cardiovascular endpoints. To date the most studied agents include metformin and sulphonylureas.<sup>1</sup>

If Erica's blood glucose levels remain uncontrolled and above target (>7% or 53 mmol/mol) it may be worth considering a combination of metformin and a GLP1 agonist or metformin and an SGLT2 receptor inhibitor; triple combination of metformin, sulphonylurea and GLP1 agonist (or alternatively an SGLT2 receptor agonist) may also be an option. These combinations may not all be supported by reimbursement under the PBS, so prescribers would need to check current PBS indications for available prescribing choices.

It is also important to consider the potential side effects of any additional agents that may be prescribed. For example, there is a risk of nausea or vomiting with use of GLP1 agonists, which occurs in up to 50% of patients but, in most cases, disappears after 1–2 weeks of continued treatment.<sup>16</sup> There is an increased risk of urogenital infections and possible urinary tract infections for the SGLT2 receptor inhibitors.<sup>1</sup> Newer agents such as these do not as yet have high-quality, long-term prospective cardiovascular safety trials. This should be discussed with patients when considering their clinical use.

Renal dysfunction may require a change in medication and/or dosage:

- Metformin: reduce maximum dose when creatinine clearance (CrCl) is <90 mL/minute<sup>17</sup>
  - 2 g recommend daily for when CrCl is 60–90 mL/minute
  - 1 g daily when CrCl is 30–60 mL/minute
  - contraindicated when <30 mL/minute.
- SGLT 2 receptor inhibitors:<sup>18,19</sup>
  - dapagliflozin is contraindicated if CrCl <60 mL/minute
  - canagliflozin is contraindicated if CrCl <45 mL/minute)
  - care in combination with loop diuretics.

## REFERENCES

1. The Royal Australasian College of General Practice and Diabetes Australia. General practice management of type 2 diabetes 2014–15. Melbourne: RACGP and Diabetes Australia; 2014. Available at [www.racgp.org.au/your-practice/guidelines/diabetes](http://www.racgp.org.au/your-practice/guidelines/diabetes) and [www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf](http://www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf) [Accessed 21 July 2014].
2. National Health and Medical Research Council. Clinical practice guidelines for the management of overweight and obesity in adults,

adolescents and children in Australia. Melbourne: National Health and Medical Research Council; 2013. Available at [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/n57\\_obesity\\_guidelines\\_131204\\_0.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57_obesity_guidelines_131204_0.pdf) [Accessed 25 July 2014].

3. Clar C, Gill JA, Court R, Waugh N. Systematic review of SGLT2 receptor inhibitors in dual or triple therapy in type 2 diabetes. *BMJ Open* 2012;2:e001007. doi:10.1136/bmjopen-2012-001007.
4. Mavian AA, Miller S, Henry RR. Managing type 2 diabetes: balancing HbA1c and body weight. *Postgrad Med* 2010;122:106–17.
5. Khaodhlar L, Cummings S, Apovian CM. Treating diabetes and prediabetes by focusing on obesity management. *Curr Diab Rep* 2009;9:348–54.
6. Pi-Sunyer FX. Weight loss in type 2 diabetic patients. *Diabetes Care* 2005;28:1526–27.
7. Bazzano LA, Serdula M, Liu S. Prevention of type 2 diabetes by diet and lifestyle modification. *J Am Coll Nutr* 2005;24:310–19.
8. Anderson JW, Kendall CW, Jenkins DJ. Importance of weight management in type 2 diabetes: review with meta-analysis of clinical studies. *J Am Coll Nutr* 2003;22:331–39.
9. Powell KE, Paluch AE, Blair SN. Physical activity for health: What kind? How much? How intense? On top of what? *Ann Rev Public Health* 2011;32:349–65.
10. Shea MK, Houston DK, Nicklas BJ et al. The effect of randomization to weight loss on total mortality in older overweight and obese adults: the ADAPT Study. *J Gerontol A Biol Sci Med Sci* 2010;65:519–25.
11. Witham MD, Avenell A. Interventions to achieve long-term weight loss in obese older people: a systematic review and meta-analysis. *Age Ageing* 2010;39:176–84.
12. Shaw KA, Gennat H, O'Rourke P, Del Mar C. Exercise for overweight or obesity. *Cochrane Database of Systematic Reviews*; 2006;4:CD003817.
13. National Health and Medical Research Council. Summary guide for the management of overweight and obesity in primary care. Melbourne: National Health and Medical Research Council; 2013. Available at [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/n57b\\_obesity\\_guidelines\\_summary\\_guide\\_131219.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57b_obesity_guidelines_summary_guide_131219.pdf) [Accessed 25 July 2014].
14. Phung O, Scholle J, Talwar M, Coleman C. Effect of Non-insulin antidiabetic drugs added to metformin therapy on glycemic control, weight gain, and hypoglycemia in type 2 diabetes. *JAMA* 2010;303:1410–18.
15. Endocrinology Expert Group. Choice of antiglycaemic drug and combination therapy in adults with type 2 diabetes. In: eTG Complete [Internet] Melbourne: Therapeutic Guidelines Ltd; 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 21 July 2014].
16. Rossi S, editor. Glucagon-like peptide-1 analogues. In: *Australian Medicines Handbook* 2014. Adelaide: Australian Medicines Handbook Pty Ltd; 2014.
17. Rossi S, editor. Metformin. In: *Australian Medicines Handbook*. Adelaide: Australian Medicines Handbook Pty Ltd; 2014.
18. Rossi S, editor. Dapagliflozin. In: *Australian Medicines Handbook*. Adelaide: Australian Medicines Handbook Pty Ltd; 2014.
19. Rossi S, editor. Canagliflozin. In: *Australian Medicines Handbook*. Adelaide: Australian Medicines Handbook Pty Ltd; 2014.

## RESOURCES FOR PATIENTS

- Diabetes Australia provides resources including guidance on diet and physical activity, [www.diabetesaustralia.com.au/Living-with-Diabetes/Eating-Well](http://www.diabetesaustralia.com.au/Living-with-Diabetes/Eating-Well)

## RESOURCES FOR DOCTORS

- [www.healthactive.gov.au](http://www.healthactive.gov.au)
- [www.health.gov.au/internet/main/publishing.nsf/Content/Nutrition+and+Physical+Activity-1](http://www.health.gov.au/internet/main/publishing.nsf/Content/Nutrition+and+Physical+Activity-1)

- General Practice management of type 2 diabetes 2014-15, [www.racgp.org.au/your-practice/guidelines/diabetes](http://www.racgp.org.au/your-practice/guidelines/diabetes)
- NHMRC summary guide for the management of overweight and obesity in primary care 2013, [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/n57b\\_obesity\\_guidelines\\_summary\\_guide\\_131219.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57b_obesity_guidelines_summary_guide_131219.pdf)
- NHMRC clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia, [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/n57\\_obesity\\_guidelines\\_131204\\_0.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57_obesity_guidelines_131204_0.pdf) P25
- Guidelines for preventive activities in general practice 8th edition Section 7.2, overweight, [www.racgp.org.au/your-practice/guidelines/redbook/prevention-of-chronic-disease/overweight/](http://www.racgp.org.au/your-practice/guidelines/redbook/prevention-of-chronic-disease/overweight/)
- Diabetes Australia Position paper on bariatric surgery. Diabetes Australia 2011; [www.diabetesaustralia.com.au/PageFiles/3/BariatricSurgery\\_PositionStatement\\_211211.pdf](http://www.diabetesaustralia.com.au/PageFiles/3/BariatricSurgery_PositionStatement_211211.pdf)
- Indigenous focussed healthy lifestyle practice resources, [www.aihw.gov.au/uploadedFiles/ClosingTheGap/Content/Publications/2012/ctgc-rs09.pdf](http://www.aihw.gov.au/uploadedFiles/ClosingTheGap/Content/Publications/2012/ctgc-rs09.pdf)

**CASE 4**

**FRANK IS SHORT OF BREATH**

Frank is 86 years of age and is one of your long-term nursing home patients. He has been deteriorating slowly over the past 12 months and is known to the local palliative care team. He has become increasingly frail, has been less mobile and has a decreased appetite. Recently he has had difficulty swallowing some of his medication. He has a past history of end-stage chronic obstructive pulmonary disease (COPD), type 2 diabetes mellitus (T2DM), several strokes and dementia. His medications include metformin, glicazide, tiotropium bromide, salbutamol, aspirin, a statin and an angiotensin converting enzyme inhibitor (ACEI).

Today when you visit he is very quiet and withdrawn. Increasingly, he experiences shortness of breath but his temperature is normal and his heart rate and blood pressure are stable. He has decreased air entry at the base of both lungs but normal percussion. His abdomen is normal and a urine dipstick test shows 2+ glucose, but is otherwise negative. A chest X-ray shows COPD but no evidence of consolidation. His blood sugar level is 19 mmol/L.

**QUESTION 1** 

Why is diabetes mellitus important when considering the needs of a patient requiring palliative care?

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**QUESTION 2** 

What are appropriate blood glucose readings at the end of life?

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**QUESTION 3** 

What factors affect glycaemic control in patients with diabetes at the end of life?

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**QUESTION 4** 

How can poor glycaemic control negatively impact on a patient's quality of life at end of life?

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**QUESTION 5** 

How would you manage Frank's diabetes now?

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## CASE 4 ANSWERS

## ANSWER 1

Diabetes mellitus is a common condition and its prevalence is increasing. Globally it has been estimated that from 2010 to 2030 there will be a 69% increase in adults aged 20–79 years with diabetes in developing countries, and a 20% increase in developed countries.<sup>1</sup> At the current rate of growth, it is estimated that the number of Australians with T2DM will increase from about 950,000 to over 2.5 million in 20 years.<sup>2</sup> As the population ages, there will be more patients with diabetes who will require palliative care. Diabetes is also one of the most common comorbidities in cancer patients. It is associated with increased risk of death from several forms of cancer including liver, pancreas, endometrial, colon and breast cancer in women, and breast, liver, oral cavity/pharynx, pancreas, bladder and colon cancer in men.<sup>3</sup>

The management of diabetes in palliative care settings aims to give the best quality of life to the patient, while aiming to achieve glucose readings in a range that do not cause symptoms. In particular, the avoidance of metabolic decompensation and diabetes-related emergencies (eg frequent and unnecessary hypoglycaemia, persistent symptomatic hyperglycaemia, diabetic ketoacidosis) are key principles of care. In most cases, tight glycaemic control is no longer appropriate in patients nearing the end of life.<sup>4</sup>

## ANSWER 2

The consensus of several guidelines suggest a range of 6–15 mmol/L is an appropriate blood glucose target for most patients in palliative care.<sup>4–6</sup> Readings at this level are optimal for maintaining patient wellbeing and cognitive function.<sup>7,8</sup> As patients will be aware of the blood glucose targets usually set in diabetes and those set for them previously, explanation and reassurance may be required.<sup>4</sup> If patients are stable and are not in the terminal phase of care, maintaining HbA1c at no lower than 59 mmol/mol (7.5%) when taking oral hypoglycaemic medication will avoid hypoglycaemia.<sup>6</sup> Hypoglycaemia is always more of a risk if the patient is taking insulin or sulphonylureas and these medications should be reviewed and the dose changed or stopped if appropriate.<sup>4</sup> Frank's blood sugar level is currently 19 mmol/L, despite being on metformin and gliclazide treatment.

There is minimal evidence for managing diabetes at the end of life, including information about an optimal blood glucose range, as patients at the end of life are a vulnerable and heterogenic group, and recruitment to studies is difficult.<sup>4,5</sup> Moreover, tight glycaemic control is unlikely to be of benefit in people with major life-limiting comorbidities, given that diabetic complications take years to develop.<sup>9</sup>

## ANSWER 3

Factors that can affect blood glucose levels at the end of life include the stress response to sustained illness, organ failure leading to renal

or liver dysfunction, the malignancy, chemotherapy, use of steroids for symptom control, frequent infections, anorexia and cachexia, nausea and vomiting, dehydration, swallowing problems and weight loss.<sup>6</sup>

## ANSWER 4

Hyperglycaemia may worsen confusion, thirst and incontinence, and impair cognitive function.<sup>7,8</sup> Blood glucose levels above 15 mmol/L may cause polyuria and an increased risk of infections. Diabetic ketoacidosis can mimic terminal illness and if not recognised and treated it can be fatal.<sup>6</sup>

Hypoglycaemia can cause discomfort, confusion and impaired cognitive function.<sup>6</sup>

## ANSWER 5

Frank's blood glucose, which is 19 mmol/L, is above the recommended optimal level, despite his being on treatment. This may be attributable to his decreased mobility, decrease in appetite or deterioration of his clinical condition.<sup>6</sup> However, hyperglycaemia can negatively impact on Frank's cognition and comfort. Depending on Frank's prognosis, additional treatment of his diabetes may be warranted. If Frank has weeks or months to live then his hyperglycaemia could be treated while rationalising his medication (*Figure 1*). If, however, Frank is in his final days of life, then the focus of his diabetes care should be ensuring his comfort (*Figure 2*).

When rationalising medication, several key points should be considered. Changes in appetite or difficulty with swallowing require a review of tablet dose and frequency. Metformin in particular can cause gastrointestinal symptoms and worsen appetite, so review of its use may be warranted.<sup>4</sup> Glucagon-like peptide 1 (GLP-1) agonists can cause nausea, vomiting, weight loss and reduced appetite.<sup>4,5</sup> Avoidance of dietary sugars may no longer be appropriate as food choices become limited,<sup>4</sup> and so involving a dietitian may be useful in addition to adjusting therapy. Avoiding long-acting sulphonylurea preparations can decrease the risk of hypoglycaemia in patients with poor dietary intake, and renal or hepatic failure.<sup>4,6</sup> Low-dose insulin may be the only option for patients whose glucose levels are high and are symptomatic despite significantly reduced dietary intake.<sup>4</sup> Patients who were on insulin for type 1 (T1DM) or T2DM previously will need lower doses if dietary intake is poor and a daily long-acting insulin may be a more appropriate regime.<sup>4,6</sup> In complex cases, liaison with the local palliative care team or diabetes team is recommended.

It is important to discuss any changes to a patient's diabetic management with the patient where possible, as well as family members or carers. Managing diabetes during the end of life can be challenging for the practitioner, patient and family.<sup>10</sup> For some people the change or even withdrawal of medication, especially insulin, can be distressing as some may view it as life-sustaining,<sup>10</sup> whereas other people may feel that managing diabetes in addition to their terminal illness is 'pointless'.<sup>6</sup> Palliative Care Australia has information sheets that can be accessed via their website that can help with patient or carer education.

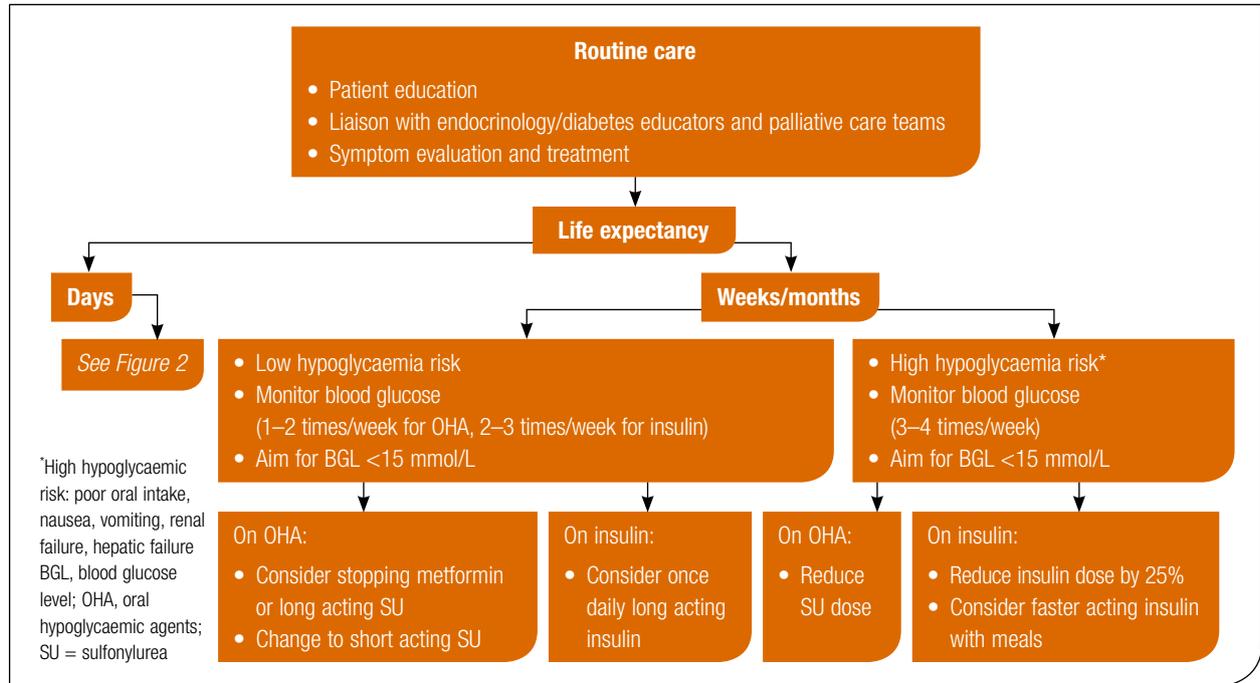


Figure 1. Algorithm for management of patients with diabetes with weeks or months to live<sup>11</sup>

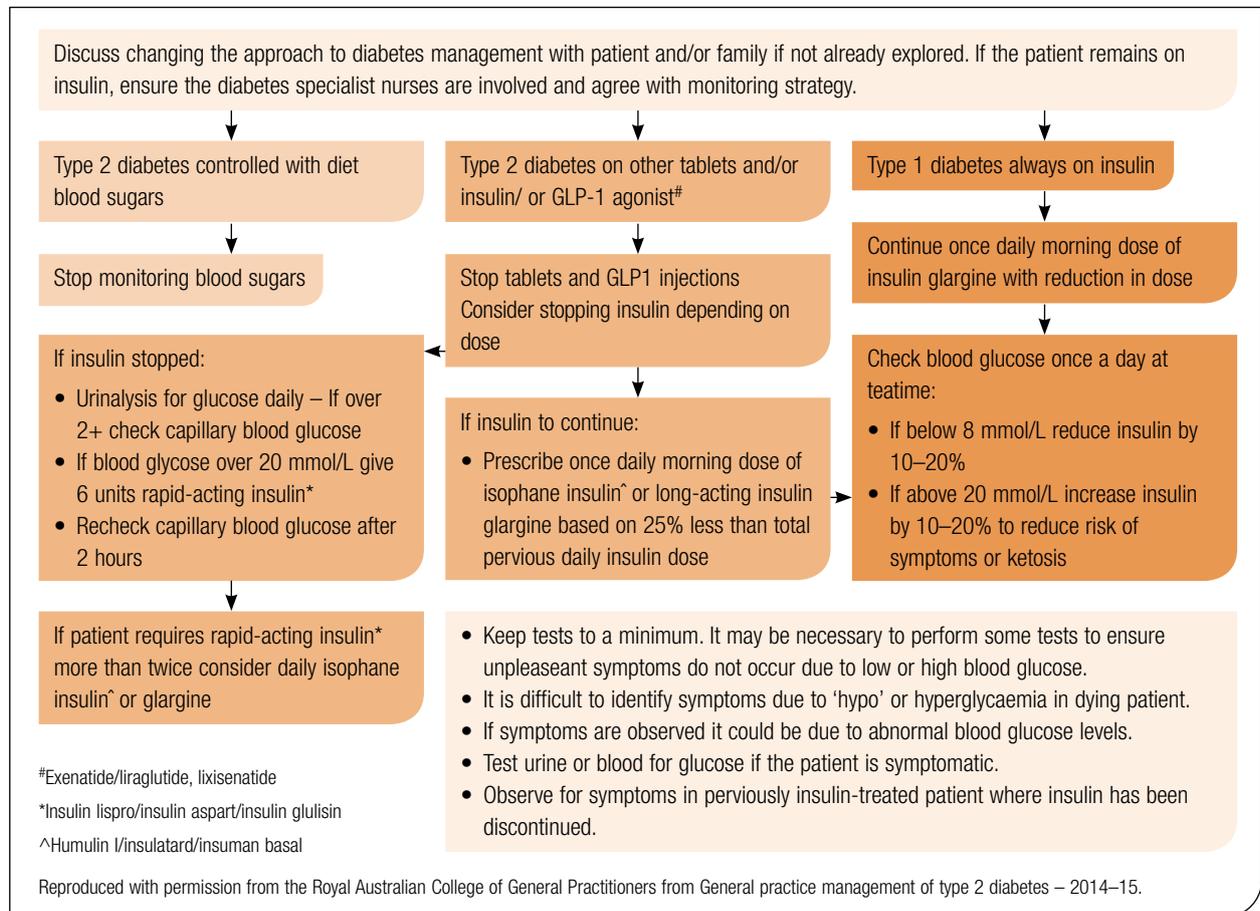


Figure 2. Algorithm for an end-of-life diabetes care management strategy for a patient with days to live<sup>7</sup>

**REFERENCES**

1. Shaw J, Tanamas S, editors. Diabetes: the silent pandemic and its impact on Australia. Melbourne: Baker IDI Heart and Diabetes Institute, 2012.
2. Shaw J, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diab Res Clin Prac* 2010;87:4–14.
3. Campbell P, Newton C, Patel A, Jacobs E, Gapstur S. Diabetes and cause-specific mortality in a prospective cohort of one million U.S. adults. *Diabetes Care* 2012;35:1835–44.
4. Diabetes UK. End of life diabetes care: clinical care recommendations. 2<sup>nd</sup> edn; 2013. Available at [www.trend-uk.org/documents/End\\_of\\_Life%20clinical%20recommendations.pdf](http://www.trend-uk.org/documents/End_of_Life%20clinical%20recommendations.pdf) [Accessed 8 July 2014].
5. Dunning T, Martin P, Savage S, Duggan N. Guidelines for managing diabetes at the end of life. Melbourne: Deakin, 2010.
6. The Royal Australian College of General Practitioners and Diabetes Australia. General practice management of type 2 diabetes – 2014–15. Melbourne: RACGP; 2014. Available at [www.racgp.org.au/your-practice/guidelines/diabetes](http://www.racgp.org.au/your-practice/guidelines/diabetes) and [www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf](http://www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf) [Accessed 8 July 2014].
7. Sommerfield AJ, Deary IJ, Frier BM. Acute hyperglycemia alters mood state and impairs cognitive performance in people with type 2 diabetes. *Diabetes Care* 2004;27:2335–40.
8. Cox DJ, Kovatchev BP, Gonder-Frederick LA, et al. Relationships between hyperglycemia and cognitive performance among adults with type 1 and type 2 diabetes. *Diabetes Care* 2005;28:71–77.
9. Cheung NW et al. Position statement of the Australian Diabetes Society: individualisation of glycosylated haemoglobin targets for adults with diabetes mellitus. *Med J Aust* 2009;191:339–44.
10. Quinn K, Hudson P, Dunning T. Diabetes management in patients receiving palliative care. *J Pain Symptom Manage* 2006;32:275–86.
11. King EJ, Haboubi H, Evans D, Baker I, Bain SC, Stephens JW. The management of diabetes in terminal illness related to cancer. *QJM* 2012;105:3–9.

**RESOURCES FOR PATIENTS AND CARERS**

- Diabetes and Palliative Care. Information for people with diabetes and family members of people with diabetes, [www.palliativecare.org.au/Resources/Consumerresources.aspx](http://www.palliativecare.org.au/Resources/Consumerresources.aspx)

**RESOURCES FOR DOCTORS**

- The Royal College of General Practitioners and Diabetes Australia. General practice management of type 2 diabetes 2014–2015. Melbourne: RACGP and Diabetes Australia, [www.racgp.org.au/your-practice/guidelines/diabetes](http://www.racgp.org.au/your-practice/guidelines/diabetes) and [www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf](http://www.diabetesaustralia.com.au/PageFiles/763/RACP%20Guidelines%20PDF.pdf)
- Diabetes UK. End-of-life diabetes care: clinical care recommendations, [www.trend-uk.org/documents/End\\_of\\_Life%20clinical%20recommendations.pdf](http://www.trend-uk.org/documents/End_of_Life%20clinical%20recommendations.pdf)

**CASE 5**

**FRED IS WORRIED ABOUT HIS WEIGHT**

Fred is a busy professional aged 48 years. Recently he was diagnosed with type 2 diabetes (T2DM), which is being controlled through dietary intervention. He currently weighs 118 kg and has a body mass index (BMI) of 38 kg/m<sup>2</sup>. His weight has steadily increased over the past 20 years. He has tried various diets, including commercial weight loss programs, slimming programs from the chemist, duromine and medication bought over the internet. None of these approaches had led to a sustainable and substantial weight loss, despite some initial successes. His wife has been urging him to do something about his weight for several years. He wants to do something definitive and with his busy work and family schedule does not feel another diet will be of any value. He would like information about adjustable gastric band surgery and requests a referral to see a surgeon.

**QUESTION 1**  

How would you respond, before discussing surgery with Fred?

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**QUESTION 2** 

What would you tell Fred about weight loss surgery in general and laparoscopic adjustable gastric banding (LAGB) surgery more specifically?

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**QUESTION 3** 

What are the patient selection criteria for bariatric surgery in Australia?

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**QUESTION 4** 

What is the mechanism of action of LAGB surgery?

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**FURTHER INFORMATION**

Several months later, Fred has lost 4 kg, but regained 2 kg. Having thought objectively about his options, he has decided that LAGB is his only alternative. He elects to undergo surgery. Placement of a LAGB is performed uneventfully as a day procedure and he is back working 4 days later. As he is well educated about the process and has realistic expectations, Fred adapts well. He achieves a loss of 18 kg in the first 12 months. His diabetes remits. He feels much better and feels energetic enough to go to the gym three times per week and now regularly plays 18 holes of golf. He attends for routine review.

**QUESTION 5** 

What is the role of the GP in managing a patient following LAGB surgery?

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**FURTHER INFORMATION**

Fred returns 4 years later. After losing a peak of 26 kg, he has regained 4 kg. He is worried that he has developed a complication. He has found that he is eating only softer (high caloric) foods and is suffering from heartburn. He is struggling to swallow most solid foods and is regurgitating every second day. He had some saline added to the LAGB eight months ago, but has not returned to the bariatric surgical follow-up team since then.

**QUESTION 6** 

What complications can arise after LAGB?

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**CASE 5 ANSWERS**

**ANSWER 1**

Although it is likely that bariatric surgery will be beneficial to Fred, several things can be considered prior to surgery to help address his weight problem. For example:

- Encourage Fred to pursue substantial weight loss.
- Gently discuss weight-loss approaches (eg reduced energy diets, increased physical activity) recommended by the National Health and Medical Research Council (NHMRC) guidelines.<sup>1</sup> These approaches include dietary intervention (reduced energy intake), increased physical activity and lifestyle. Specifically, mention that even a small amount of weight loss will have substantial benefits to his overall health.
- Develop a weight management plan for Fred, which could include referral to other health professionals (eg dietitian).
- Advise Fred that even if he undergoes surgery, he will still need to continue to make substantial efforts to control his weight and improve his overall health for the remainder of his life.
- Use this visit as an opportunity to discuss and screen for other medical problems such as complications of his diabetes, obstructive sleep apnoea, hypercholesterolaemia, hypertension, non-alcoholic fatty liver disease and related conditions, as well as

psychosocial and psychiatric issues; where appropriate referrals and review should be arranged.

- Emphasise the three key benefits of weight loss, which include improved physical quality of life, improvements in medical problems and long-term reduction of cardiovascular and mortality risks.
- Ensure that Fred has adequate information to make a decision about surgery. Suggest that he look at reputable websites such as Obesity Surgeons Society of Australia and New Zealand (refer to *Resources for doctors*) and obtain further information, for instance by reading the *LAP-BAND Solution* book.<sup>2</sup>
- Schedule a follow-up appointment to review Fred's progress and discuss surgery at that time if appropriate.

**ANSWER 2**

The term bariatric surgery covers a range of surgical procedures that aim to reduce weight and to maintain weight loss. Techniques include LAGB. Another procedure is laparoscopic sleeve or tube gastrectomy, where most of the fundus and gastric body are removed, leaving an approximately 200-ml capacity residual gastric sleeve or tube. Gastric bypass (Roux-en-Y or RYGB) involves dividing the upper stomach, creating a 30-mL proximal gastric pouch to which the jejunum is anastomosed, thereby bypassing the remainder of the stomach. All bariatric procedures induce weight loss by either reducing caloric intake or limiting caloric absorption. Depending on the surgical technique used, weight loss may also be achieved via additional mechanisms (eg delayed digestion, changes in the levels of hormones that control hunger).<sup>3</sup>

Different procedures carry differing risk profiles and follow-up requirements. All procedures have been shown to be highly effective at inducing substantial weight loss, inducing remission of comorbidities, improving quality of life and reducing long-term mortality.<sup>4</sup>

The risk and benefits of weight loss surgery should be explained to Fred. LAGB is a very effective weight loss procedure. It has the advantages of safety, efficacy, adjustability and reversibility. It is often performed as a day-case procedure.<sup>5</sup> *Figure 1* shows the LAGB device and port with the balloon inflated and deflated. *Figure 2* shows the band correctly positioned at the very top of the stomach around the cardia. There are several different devices in use, which are generally similar in design.

LAGB requires lifelong follow-up. This usually involves at least 6–8 visits in the first year, 4–6 visits in the second year and then review every 3–6 months in the long term. Follow-up involves discussion about food intake, nutrition, physical activity, understanding the effects of the band, ensuring the band is adjusted appropriately, and monitoring for complications.<sup>6</sup>

LAGB requires major behavioural change, an alteration in the approach to eating and continued efforts to lose and control weight, paying attention to food selection, quantity and portion control, as well as ensuring that regular exercise (20–30 minutes) is incorporated into the daily schedule.<sup>2</sup>

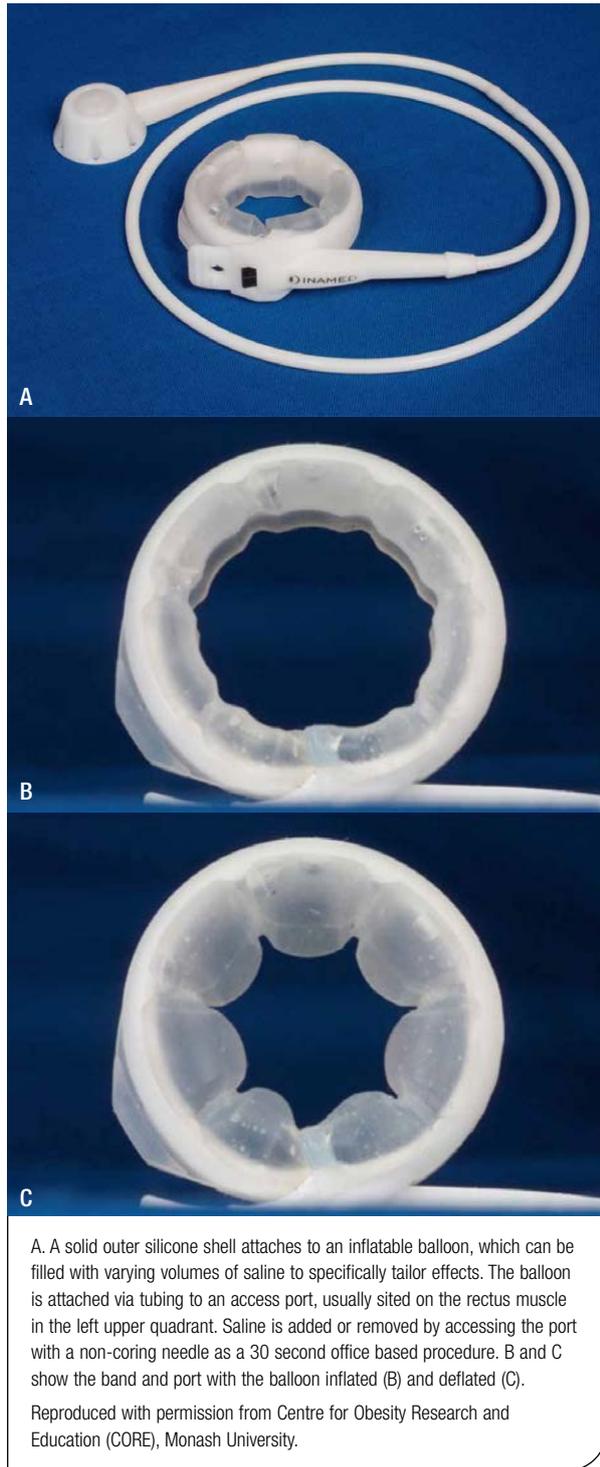


Figure 1. Laparoscopic adjustable gastric band

Weight loss of 1 kg/week is possible in the first year following bariatric surgery but a more realistic weight loss is around 0.5 kg/week. Depending on the procedure undertaken, a steady weight loss is achieved within 12–24 months post surgery.<sup>3</sup> There is a high chance of achieving substantial weight loss (about 50% of excess

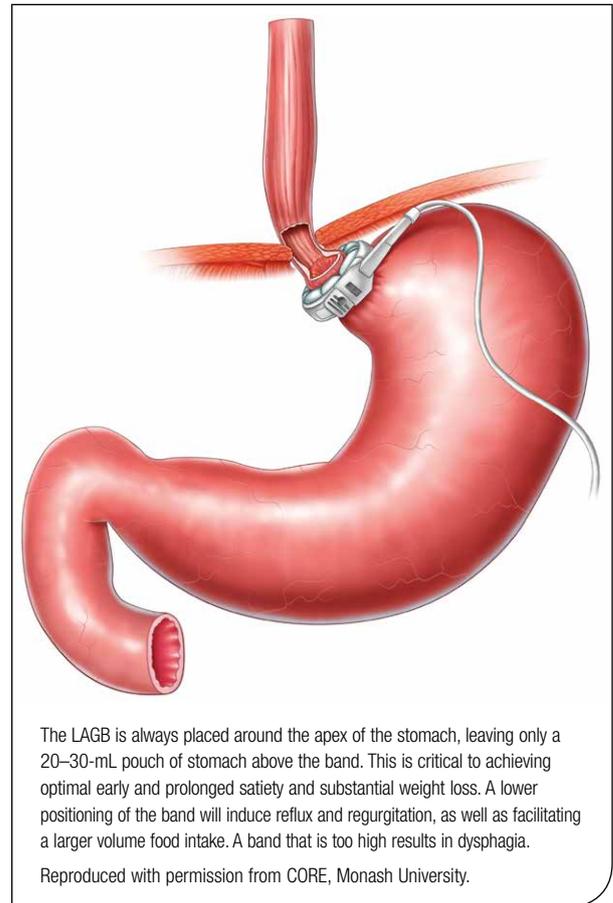


Figure 2. Laparoscopic adjustable gastric band correctly positioned

weight) with LAGB, which has been shown to be maintained for at least 15 years.<sup>7</sup> Improvements in diabetes outcomes, including remission of diabetes, have been reported following LAGB.<sup>8–10</sup>

**ANSWER 3**

NHMRC guidelines suggest that bariatric surgery may be considered for adults with BMI >40 kg/m<sup>2</sup>, or BMI >35 kg/m<sup>2</sup> and an obesity-related comorbidity. LAGB surgery by a specialist bariatric paediatric team may be considered in postpubertal adolescents with BMI >40 kg/m<sup>2</sup>, or BMI >35 kg/m<sup>2</sup> with obesity-related complications, where other interventions have been unsuccessful.<sup>1</sup> These criteria are similar to other international guidelines. Medicare defines the indication for bariatric surgery as clinically severe obesity.<sup>11</sup> Other factors that are often used to select suitable patients include:<sup>3</sup>

- age 18–65 years
- fitness to undergo surgery
- ability to give informed consent
- willing to participate in long-term follow-up
- selected patients, with BMI >30 kg/m<sup>2</sup> and an obesity-related comorbidity.

A recent randomised trial in 51 patients with BMI 25–30 kg/m<sup>2</sup> and T2DM evaluated the addition of LAGB to multidisciplinary diabetes care. The addition of LAGB improved glycaemic control with an acceptable adverse event profile. Diabetes remission was reported at follow-up 2 years later in 12 (52%) participants in the multidisciplinary care plus gastric band group and two (8%) participants in the multidisciplinary care only group.<sup>10</sup>

Contraindications to surgery include irreversible end-organ dysfunction, cirrhosis with portal hypertension, medical problems precluding general anaesthesia and centrally mediated obesity syndromes such as Prader-Willi syndrome or craniopharyngioma.

#### ANSWER 4

LAGB induces early and prolonged satiety,<sup>12</sup> which results in decreased overall energy intake and weight loss. Daily energy intake is reduced from approximately 10,545 kJ (2497 cal) to 4271 kJ (1020 cal) in the 12 months following LAGB.<sup>13</sup>

Compression of nerve endings in the proximal stomach results in suppression of hunger between meals and particularly in the mornings.<sup>14</sup>

An appropriately adjusted LAGB results in significant slowing of food transit into the stomach below the band.<sup>15</sup> Each bite must be appropriately sized, chewed well and a period of 45–60 seconds allowed for several oesophageal peristaltic contractions to transit the swallowed bolus in portions, through the band. *Figure 3* illustrates the resultant effect, which leads to a feeling of fullness and meal termination after consumption of a small volume of food.

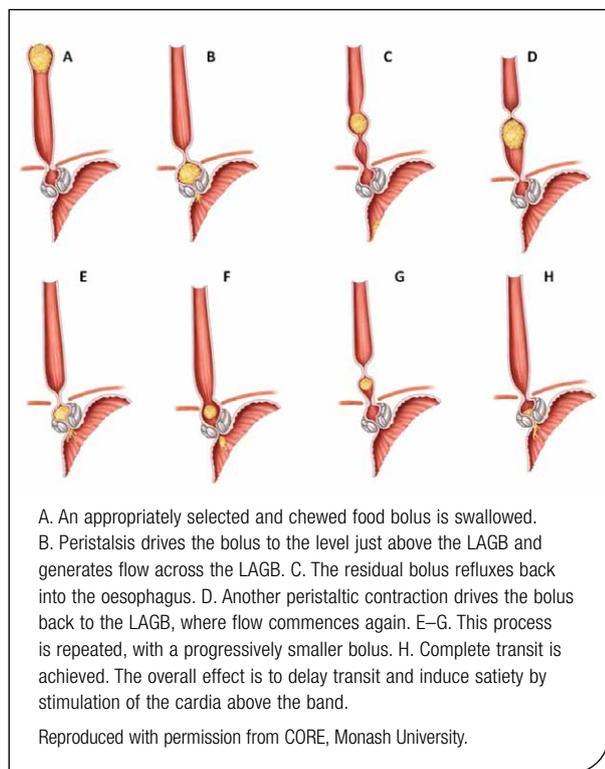


Figure 3. Effects of LAGBs on swallowing

During follow-up, adjustments can be made to the band, with saline added and removed. Patients are presented with the schema shown in *Figure 4*.<sup>2</sup> This divides a patient's eating and sensations into three zones:

- Yellow: indicates inadequate saline and too much hunger.
- Green (optimal zone): patients consume small meals and are satisfied, and weight loss is good.
- Red: there is too much saline and adverse symptoms, such as reflux and regurgitation, are experienced.

#### ANSWER 5

The GP is a key part of the bariatric follow-up team, while needing to continue to monitor other aspects of the patient's care.<sup>16</sup> For example the GP could:

- Remain central to overseeing management of all the patient's medical problem
- Adjust/review medical care and medication(s) as requirements change during weight loss, particularly if diabetic medications or anti-hypertensive agents are being used
- Monitor patients for adverse symptoms, complications and nutritional status relating to bariatric surgery
- Encourage patients to continue attendance at bariatric follow-up visits
- Reinforce healthy eating and exercise messages.

The GP should be aware of what is 'normal', following LAGB surgery. When the amount of fluid in the band is optimal the patient feels satisfied with 2–3 small meals per day of solid food with weight loss of about 0.5–1 kg per week<sup>3</sup> (or weight stability after substantial weight loss) and does not experience adverse symptoms.

A significant number of GP's have been trained to conduct LAGB follow up and perform adjustments. This can occur at bariatric surgical centres or within the general practice setting. Many regional centres have GPs able to perform LAGB follow-up. A small amount of training/mentoring is required to gain those skills.

#### ANSWER 6

A number of complications can arise following laparoscopic gastric band surgery, which may explain Fred's situation. The most likely problem is that Fred is in the red zone (*Figure 4*) and this can be remitted with removal of some saline and more frequent follow up. It is unlikely further investigations will be required. Weight loss can be worsened if the band is too tight as patients migrate to eating softer, high caloric foods. The other possibility is that Fred has developed a complication of pouch dilatation or prolapse (*Table 1*). This may be suspected if simple measures of optimally adjusting the band and better patient education do not improve symptoms. Fred should be advised to return to his bariatric surgeon for review. Common symptoms and complications following laparoscopic gastric band surgery are listed in *Table 1*.

LAGB is very low risk, however serious peri-operative events such as perforation/infection can occur. Surgical mortality is estimated at 1–5/10,000.<sup>7,17</sup>

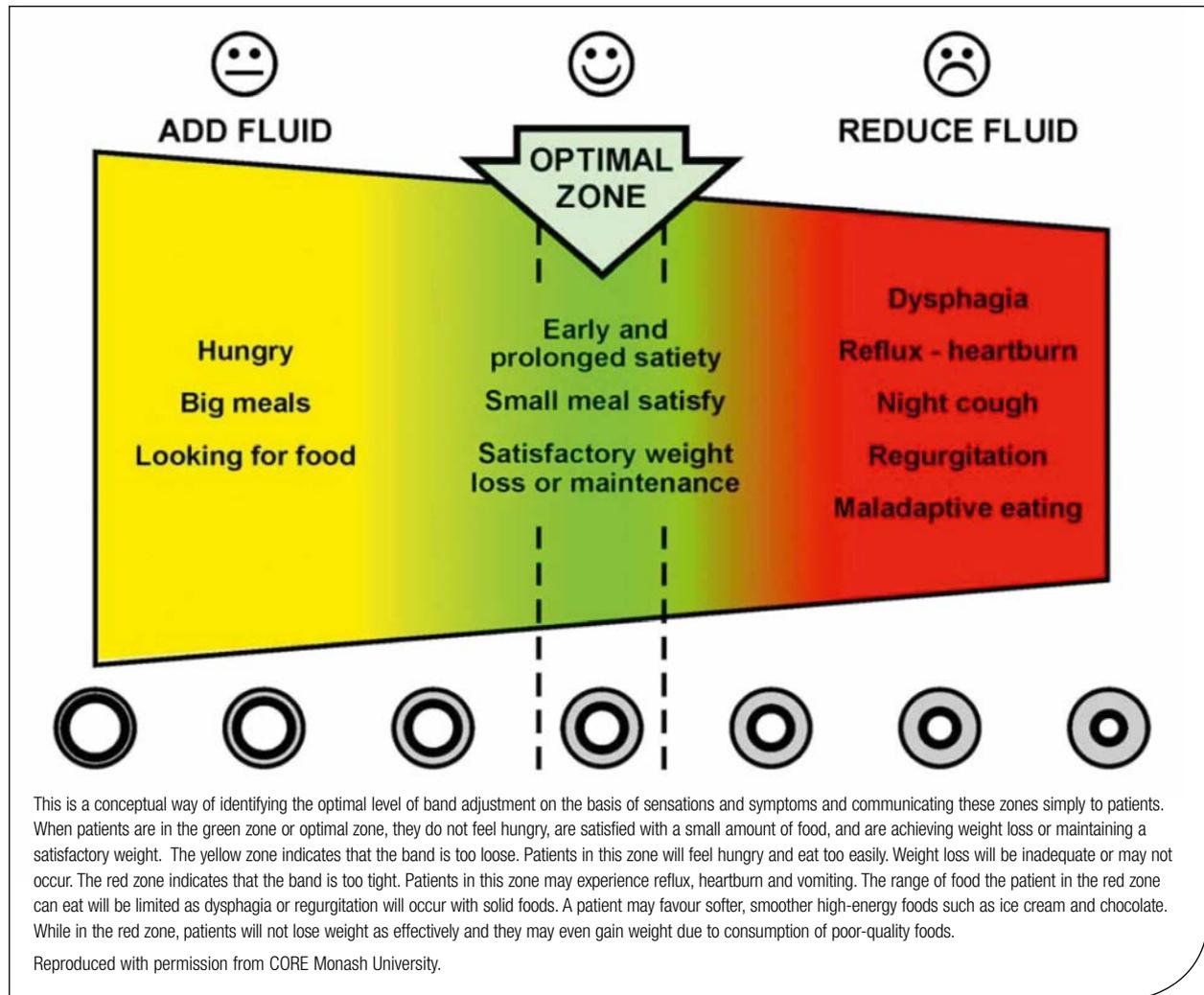


Figure 4. Clinical zones in patients with LAGBs<sup>2</sup>

A 15-year review of over 3000 patients identified a revisional surgery rate of 30%.<sup>7</sup> This included surgery on the port and tubing because of device wear and tear, and operations for symptomatic gastric enlargements above the band. A randomised controlled trial followed up to 10 years showed maintenance of weight loss (14 kg) in the LAGB group. The non-surgical group lost minimal weight (0.4 kg). Revisional surgery occurred in 30% of the LAGB patients and during the follow-up decade 12% had the LAGB removed.

Other series have shown very high failure and LAGB explant rates.<sup>18</sup> One possible explanation for the differences in reported outcomes is ensuring patients are diligently followed up in the long term.<sup>19</sup> Without that follow-up, LAGB cannot be expected to work. If LAGB patients elect to have the LAGB removed or have complications, then conversion to an alternative procedure (such as gastric bypass or sleeve gastrectomy) is generally safe and successful, although risks may be higher than with primary surgery.<sup>20,21</sup>

Longer-term prospective data or comparative randomised controlled trials are rare in bariatric surgery. The largest randomised controlled trial ( $n = 250$ ) comparing laparoscopic gastric bypass to LAGB found higher percentage excess weight loss at 4 years ( $68 \pm 19\%$  versus  $45 \pm 28\%$ ) in the bypass group but also a higher complication rate and one mortality, compared with none. The authors concluded that both procedures were effective: gastric bypass resulted in better weight loss but was associated with more perioperative and late complications and a higher 30-day readmission rate.<sup>22</sup>

One issue with LAGB is the incidence of dysphagia and patients cite the inability to consume certain types of foods and the necessity to substantially alter eating intake as the biggest problem following LAGB.<sup>15</sup> This is a reality of the procedure and a dysphagia score of around 20 out of 45 (with 0 being no dysphagia and 45 being the inability to consume liquid) being observed in patients with good weight loss and in a regular follow-up program.<sup>15</sup>

Nutritional deficiencies are rare with the LAGB and weight loss has been shown to be primarily of the fat mass.<sup>23,24</sup> Patients who have undergone procedures that permanently alter gastrointestinal anatomy or absorptive capacity, such as gastric bypass or sleeve gastrectomy are at higher risk of nutritional deficiencies.<sup>25</sup> These can include fat soluble vitamins such as vitamin A or D (including calcium and parathyroid hormone), folate and B<sub>12</sub>, iron and micronutrients such as zinc.<sup>25,26</sup>

**Table 1. Common problems and complications after laparoscopic adjustable gastric banding**

Complication/ problem	Features	Investigations/ management
Too much saline in the band	Red zone: Heartburn, dysphagia, regurgitation	Usually easily resolved with removal of saline and patient education
Too little saline in the band	Yellow zone: Weight gain, eating freely, inadequate satiety	More frequent follow up, addition of saline to the LAGB, patient education
Pouch dilatation – also known as slip or prolapse of the band	Regurgitation and reflux, or heartburn, particularly recumbent or nocturnal	Barium swallow
Port problem – leakage of saline	Weight gain, loss of satiety, usually associated with needlestick injury to tubing or hub of port	Check volume of saline in system, if a significant discrepancy then may need a port revision
Erosion of band into stomach	Loss of satiety, increased appetite, ability to eat freely  Possible spontaneous port infection	Gastroscopy

The obesity surgeons society of Australia and New Zealand ([www.ossanz.com.au](http://www.ossanz.com.au)) strongly advocates life long follow-up for all patients who have undergone bariatric surgery to ensure a good outcome is maintained, healthy lifestyle measures continue, complications are identified early and nutritional status is monitored (refer to *Resources for doctors*).

**REFERENCES**

- NHMRC. Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia. Melbourne 2013. Available at [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/n57\\_obesity\\_guidelines\\_131204\\_0.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57_obesity_guidelines_131204_0.pdf) [Accessed 23 July 2014].
- O'Brien PE. The LAP-BAND Solution a partnership for weight loss. Melbourne: Melbourne University Publishing; 2011.
- Diabetes Australia Position Paper on Bariatric surgery. Diabetes Australia 2011. Available at [www.diabetesaustralia.com.au/PageFiles/3/BariatricSurgery\\_PositionStatement\\_211211.pdf](http://www.diabetesaustralia.com.au/PageFiles/3/BariatricSurgery_PositionStatement_211211.pdf) [Accessed 21 July 2014].

- Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA* 2004;292:1724–37.
- Wentworth JM, Playfair J, Laurie C, et al. Multidisciplinary diabetes care with and without bariatric surgery in overweight people: a randomised controlled trial. *Lancet Diabetes Endocrinol* 2014;2:545–52.
- O'Brien PE, Dixon JB, Brown W. Obesity is a surgical disease: overview of obesity and bariatric surgery. *Aust N Z J Surg* 2004;74:200–04.
- O'Brien PE, MacDonald L, Anderson M, Brennan L, Brown WA. Long-term outcomes after bariatric surgery: fifteen-year follow-up of adjustable gastric banding and a systematic review of the bariatric surgical literature. *Ann Surg* 2013;257:87–94.
- Courcoulas AP, Goodpaster BH, Eagleton JK, et al. Surgical versus medical treatments for type 2 diabetes mellitus: a randomised controlled trial. *JAMA Surg* 2014;149:707–15.
- Edelman S, Ng-Mak DS, Fusco M, et al. Control of type 2 diabetes after 1 year of laparoscopic adjustable gastric banding in the helping evaluate reduction in obesity (HERO) study. *Diabetes Obes Metab* 2014 May 13. doi: 10.1111/dom.12313. [Epub ahead of print]
- Wentworth JM, Playfair J, Laurie C, et al. Multidisciplinary diabetes care with and without bariatric surgery in overweight people: a randomised controlled trial. *Lancet Diabetes Endocrinol* 2014;2:545–52.
- Australian Government Department of Health. MBS online. Available at [www.health.gov.au/internet/mbsonline/publishing.nsf/Content/Medicare-Benefits-Schedule-MBS-1](http://www.health.gov.au/internet/mbsonline/publishing.nsf/Content/Medicare-Benefits-Schedule-MBS-1) [Accessed 2 September 2014].
- Burton PR, Brown WA. The mechanism of weight loss with laparoscopic adjustable gastric banding: induction of satiety not restriction. *Int J Obes* 2011;35:S26–30.
- Colles SL, Dixon JB, O'Brien PE. Hunger control and regular physical activity facilitate weight loss after laparoscopic adjustable gastric banding. *Obes Surg* 2008;18:833–40.
- O'Brien PE. Bariatric surgery: mechanisms, indications and outcomes. *J Gastroenterol Hepatol* 2010;25:1358–65.
- Burton PR, Brown WA, Laurie C, Hebbard G, O'Brien PE. Mechanisms of bolus clearance in patients with laparoscopic adjustable gastric bands. *Obes Surg* 2010;20:1265–72.
- Brown W, Korin A, Burton P, O'Brien PE. Laparoscopic adjustable gastric banding. *Aust Fam Physician* 2009;38:972–76.
- Chapman AE, Kiroff G, Game P, et al. Laparoscopic adjustable gastric banding in the treatment of obesity: a systematic literature review. *Surgery* 2004;135:326–51.
- DeMaria EJ, Sugerman HJ, Meador JG, et al. High failure rate after laparoscopic adjustable silicone gastric banding for treatment of morbid obesity. *Ann Surg* 2001;233:809–18.
- O'Brien PE, Dixon JB, Brown W, et al. The laparoscopic adjustable gastric band (Lap-Band): a prospective study of medium-term effects on weight, health and quality of life. *Obes Surg* 2002;12:652–60.
- Fernando Santos B, Wallaert JB, Trus TL. Band removal and conversion to sleeve or bypass: are they equally safe? *Surg Endosc* 2014 [Epub ahead of print].
- Carandina S, Tabbara M, Bossi M, Helmy N, Polliand C, Barrat C. Two stages conversion of failed laparoscopic adjustable gastric banding to laparoscopic Roux-En-Y gastric bypass. A study of one hundred patients. *J Gastrointest Surg* 2014 [Epub ahead of print].
- Nguyen NT, Slone JA, Nguyen XM, Hartman JS, Hoyt DB. A prospective randomized trial of laparoscopic gastric bypass versus laparoscopic adjustable gastric banding for the treatment of morbid obesity: outcomes, quality of life, and costs. *Ann Surg* 2009;250:631–41.
- Strauss BJ, Marks SJ, Growcott JP, et al. Body composition changes following laparoscopic gastric banding for morbid obesity. *Acta Diabetologica* 2003;40(Suppl 1):S266–69.

24. Dixon JB, Strauss BJ, Laurie C, O'Brien PE. Changes in body composition with weight loss: obese subjects randomized to surgical and medical programs. *Obesity* 2007;15:1187–98.
25. Gehler S, Kern B, Peters T, Christoffel-Courtin C, Peterli R. Fewer nutrient deficiencies after laparoscopic sleeve gastrectomy (LSG) than after laparoscopic Roux-Y-gastric bypass (LRYGB)-a prospective study. *Obes Surg* 2010;20:447–53.
26. Bal BS, Finelli FC, Shope TR, Koch TR. Nutritional deficiencies after bariatric surgery. *Nature Rev Endocrinol* 2012;8:544–56.

### RESOURCES FOR DOCTORS

- Obesity Surgeons Society of Australia and New Zealand, [www.ossanz.com.au](http://www.ossanz.com.au)
- National Health and Medical Research Council clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia, 2013, [www.nhmrc.gov.au/guidelines/publications/n57](http://www.nhmrc.gov.au/guidelines/publications/n57)
- Monash University Centre for Obesity Research and Education (CORE), [www.core.monash.org](http://www.core.monash.org)
- O'Brien PE. *The LAP-BAND Solution a partnership for weight loss*. Melbourne: Melbourne University Publishing; 2011.

**CASE 6**

**ROBERT IS HAVING BLURRED VISION**

Robert, aged 65 years, has had type 2 diabetes mellitus (T2DM) for 15 years and is on insulin treatment. He visits you to discuss his eyes. For the past 4–6 months he has had increased blurring of his right eye and his glasses do not seem to help anymore. He is finding it harder to read the newspaper and driving has become more difficult. These are tasks he found easy to perform 6 months ago. He also has a history of hypertension and raised cholesterol for which he takes medications.

**QUESTION 1** 

What are the possible causes of Robert’s blurred vision?

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**QUESTION 2** 

What would be your initial management plan?

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**FURTHER INFORMATION**

Robert saw an optometrist 18 months ago and at the time his distance vision was corrected to 6/6 with glasses in both eyes. He went back to the optometrist after seeing you. The

optometrist was unable to refract his right eye beyond 6/18, although his left eye was still 6/6. No significant lens opacity was noted in either eye and no drusen or significant retinal pigment change was noted at either macula to suggest age-related macular degeneration. A copy of Robert’s fundus photos (Figure 1) and optical coherence tomography (OCT) scan (Figure 2) for the right eye were sent to you with the report and a recommendation for referral.



Figure 1. A colour fundus photograph of Robert’s right eye

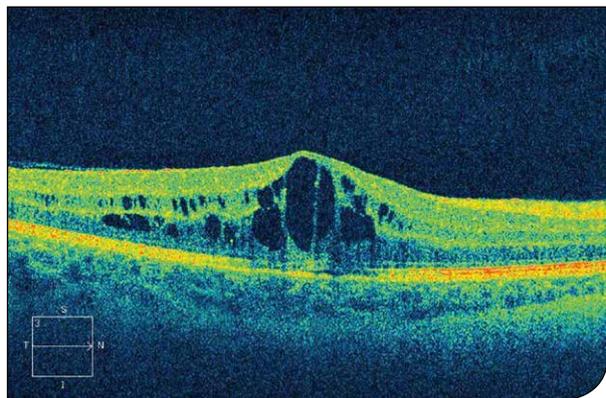


Figure 2. An OCT scan of Robert’s right central macula (scanned through the fovea)

**QUESTION 3** 

What do the images show?

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**QUESTION 4** 

What would you do next?

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**FURTHER INFORMATION**

Robert returns 6 months after treatment for his right diabetic macular oedema. He has had a number of injections of an agent that inhibits vascular endothelial growth factor (anti-VEGF) for his right eye. He now finds it easier to read the newspaper and feels that his vision has improved overall.

**QUESTION 5** 

What is your long-term management of Robert's eyes?

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**CASE 6 ANSWERS**

**ANSWER 1**

Common causes of vision loss for Robert would be refractive error, cataract, macular degeneration and diabetic retinopathy (a microvascular complication of diabetes). In Western countries, macular degeneration is currently the most important cause of blindness; however, uncorrected refractive error continues to be the leading cause of moderate and severe vision impairment.<sup>1</sup> Refractive error does not usually change significantly over 6 months, and age-related cataracts also progress slowly, so these are less likely to be responsible for Robert's vision changes. His blurred vision may be due to progression of the atrophic, or 'dry' form of age-related macular degeneration (choroidal neovascularisation or 'wet' age-related macular degeneration tends to cause sudden loss of vision and distortion). In any patient with diabetes, diabetic maculopathy is also a consideration.

**ANSWER 2**

In the first instance it would be useful to check Robert's vision. If his vision improves through a pinhole, this suggests his visual blur is at least partly due to refractive error.<sup>2</sup> Robert needs to have his refractive error checked and have a dilated ocular examination to determine the cause of his blurred vision. You may wish to perform the dilated examination in your rooms, or refer Robert to an optometrist or ophthalmologist for this initial review. It is recommended that this review occur within 1–2 weeks, as long-term visual outcomes for the treatment of wet age-related macular degeneration and diabetic retinopathy are correlated to the time from first symptoms to the time of initial treatment,<sup>3,4</sup> whereas long-term visual outcomes for the treatment of diabetic retinopathy are correlated to less severe diabetic retinopathy at presentation.<sup>5</sup> Current treatment options for both conditions aim to prevent further vision loss, but do not always restore vision already lost.

Checking Robert's blood pressure and blood glucose control would also be useful. Depending on the outcomes of these checks, it may be necessary to adjust his current medications to ensure that his blood pressure and blood glucose are within appropriate targets for him.

**ANSWER 3**

The first image is a fundus photograph of the right macula. It shows severe diabetic maculopathy with retinal haemorrhages and hard exudates, particularly in the superior macula, and these extend into the fovea. Severe diabetic maculopathy is defined as fovea-involving diabetic change.

The second image is an optical coherence tomography (OCT) scan. OCT images are increasingly being used to assess macular pathology and monitor progress with any treatment.<sup>6</sup> Robert's OCT scan shows cystoid macular oedema with multiple dark cystic spaces of fluid involving the fovea. Vision is usually significantly affected once the fovea is involved.

**ANSWER 4**

Robert has visually significant diabetic macular oedema (DMO) in his right eye. At this point he requires referral to an ophthalmologist for treatment. A 2012 systematic review using data from approximately 23,000 patients with diabetes estimated the following prevalence of diabetic eye problems:<sup>7</sup>

- any diabetic retinopathy: 34.6%
- diabetic macular oedema: 6.81%
- proliferative diabetic retinopathy: 6.96%
- any vision-threatening diabetic retinopathy: 10.2%.

For many years diabetic macular oedema was treated with argon laser photocoagulation in accordance with findings from the Early Treatment Diabetic Retinopathy Study.<sup>8</sup> This study showed that macular laser treatment for clinically significant diabetic macular oedema reduced the risk of losing three lines on a vision chart by 50% at 2 years, compared with no treatment.<sup>3</sup>

More recently intravitreal steroid and intravitreal anti-VEGF agents have been used. The duration of the effects of intravitreal triamcinolone acetonide (a steroid) is 4–6 months<sup>9</sup> and that of intravitreal anti-VEGF injection is approximately 1 month.<sup>10</sup>

In patients with DMO that has persisted or recurred after laser treatment, intravitreal triamcinolone acetonide has been shown to improve vision and reduce macular thickness. With repeated treatment, this benefit was seen for up to 2 years.<sup>4</sup> Adverse events associated with this treatment, however, include acceleration of cataract and raised intraocular pressure, which may lead to glaucoma<sup>11</sup> and, uncommonly, endophthalmitis.<sup>12</sup>

The Diabetic Retinopathy Clinical Research Network reported on a randomised trial evaluating intravitreal ranibizumab (an anti-VEGF agent) or 4 mg triamcinolone acetonide combined with macular laser, compared with macular laser alone, for treatment of DMO. The 2-year results for all patients showed an improvement in the mean change from baseline in the visual acuity letter score in the combined ranibizumab and laser groups, but was marginally worse in the combined triamcinolone and laser group, compared with laser treatment alone.<sup>13</sup> It was believed that part of the vision loss in the intravitreal steroid group was related to cataract formation. For patients who had already undergone cataract surgery, however, the combination of ranibizumab and laser had equivalent outcomes for vision when compared with steroid and laser. This finding has led many ophthalmologists to use anti-VEGF therapy as a first-line treatment for patients with DMO.<sup>14</sup> The anti-VEGF therapies have been associated with a doubling of the likelihood of vision gain and have one-third fewer cases of severe vision loss compared with previous laser treatment.<sup>13</sup>

**ANSWER 5**

The ophthalmic treatment of Robert's diabetic macular oedema addressed the diabetic eye changes he had at the time.

Continuing to monitor his diabetes and hypertension is important to prevent the development of further diabetic retinopathy, which may be associated with future vision loss. Optimising blood glucose levels

and blood pressure, and possibly blood lipid levels, is very important for reducing the risk of development or progression of diabetic retinopathy.<sup>15</sup>

The FIELD study, a randomised controlled study assessing the effects of fenofibrate, showed that patients with T2DM on fenofibrate 200 mg daily had a significantly lower requirement for first laser treatment, compared with placebo (3.4% fenofibrate group versus 4.9% placebo group).<sup>16</sup> This outcome seems to be independent of its effect on plasma lipid concentrations. Fenofibrate may be considered for any patient with diabetic retinopathy, and does not need to be limited to those cases with diabetic retinopathy that affects visual acuity. Note, fenofibrate is indicated for use in dyslipidaemia associated with T2DM, severe hypertriglyceridaemia and hypercholesterolaemia (second-line treatment for those unable to tolerate other lipid-lowering agents).<sup>17</sup> The Therapeutic Goods Administration (TGA) recently approved the use of fenofibrate in patients with diabetic retinopathy. The 200 mg dose used in the FIELD study is bioequivalent to three tablets of 48 mg or one tablet of 145 mg, which are available in Australia.<sup>18</sup>

**REFERENCES**

1. Bourne RR, Jonas JB, Flaxman SR et al. Prevalence and causes of vision loss in high-income countries and in Eastern and Central Europe: 1990–2010. *Br J Ophthalmol* 2014;98:629–38.
2. Elkington AR, Frank HJ, Greaney MJ. *Clinical Optics*, 3rd edition. Oxford: Wiley-Blackwell, 1999. p116–17.
3. Lim JH, Wickremasinghe SS, Xie J et al. Delay to treatment and visual outcomes in patients treated with anti-vascular endothelial growth factor for age-related macular degeneration. *Am J Ophthalmol* 2012;153:678–86.
4. Rauch R, Weingessel B, Maca SM, et al. Time to first treatment: the significance of early treatment of exudative age-related macular degeneration. *Retina* 2012;32:1260–64.
5. Bressler SB, Qin H, Beck RW, et al. Factors associated with changes in visual acuity and central subfield thickness at 1 year after treatment for diabetic macular oedema with ranibizumab. *Arch Ophthalmol* 2012;130:1153–61.
6. Sakata LM, Deleon-Ortega J, Sakata V, et al. Optical coherence tomography of the retina and optic nerve – a review. *Clin Experiment Ophthalmol* 2009;37:90–99.
7. Yau JW, Rogers SL, Kawasaki R et al. Global prevalence and major risk factors of diabetic retinopathy. *Diab Care* 2012;35:556–64.
8. Early Treatment Diabetic Retinopathy Study Research Group. Treatment techniques and clinical guidelines for photocoagulation of diabetic macular edema. Early treatment diabetic retinopathy study report number 2. *Ophthalmology* 1987;94:761–74.
9. Beer PM, Bakri SJ, Singh RJ, et al. Intraocular concentration and pharmacokinetics of triamcinolone acetonide after a single intravitreal injection. *Ophthalmology* 2003;110:681–86.
10. Stewart MW. Pharmacokinetics, pharmacodynamics and pre-clinical characteristics of ophthalmic drugs that bind VEGF. *Expert Rev Clin Pharmacol* 2014;7:167–80.
11. Gillies MC, Sutter FK, Simpson JM, et al. Intravitreal triamcinolone for refractory diabetic macular edema: two-year results of a double-masked, placebo-controlled, randomized clinical trial. *Ophthalmology* 2006;113:1533–38.
12. Rossi S, editor. Triamcinolone. In: *Australian Medicines Handbook* 2014. Adelaide: Australian Medicines Handbook Pty Ltd 2014.

13. Elman MJ, Bressler NM, Qin H, et al. Diabetic Retinopathy Clinical Research Network. Expanded 2-year follow-up of ranibizumab plus prompt or deferred laser or triamcinolone plus prompt laser for diabetic macular edema. *Ophthalmology* 2011;118:609–14.
14. Mitchell P, Wong TY. Diabetic Macular Edema Treatment Guideline Working Group. Management paradigms for diabetic macular edema. *Am J Ophthalmol* 2014;157:505–13.
15. Cheung N, Mitchell P, Wong TY. Diabetic retinopathy. *Lancet* 2010;375:124–36.
16. Keech AC, Mitchell P, Summanen PA, et al. Effect of fenofibrate on the need for laser treatment for diabetic retinopathy (FIELD study): a randomised controlled trial. *Lancet* 2007;370:1687–97.
17. Rossi S, editor. Fenofibrate. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd 2014.
18. Therapeutic Goods Administration. Australian Register of Therapeutic Goods: Fenofibrate product information. Available at [www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/PICMI?OpenForm&t=&q=fenofibrate](http://www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/PICMI?OpenForm&t=&q=fenofibrate) [Accessed 19 August 2014].

### RESOURCES FOR PATIENTS AND DOCTORS

- National Health and Medical Research Council. Guidelines for the management of diabetic retinopathy, [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/di15.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/di15.pdf)
- Mohamed Q, Gillies MC, Wong TY. Management of diabetic retinopathy: a systematic review, <http://jama.jamanetwork.com/article.aspx?articleid=208502>
- The Royal College of Ophthalmologists (UK). Diabetic retinopathy guidelines, [www.icoph.org/dynamic/attachments/taskforce\\_documents/2012-sci-267\\_diabetic\\_retinopathy\\_guidelines\\_december\\_2012.pdf](http://www.icoph.org/dynamic/attachments/taskforce_documents/2012-sci-267_diabetic_retinopathy_guidelines_december_2012.pdf)

**Diabetes and obesity (Activity ID: 9894)**

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 <http://gplearning.racgp.org.au>
- log into the *gplearning* website at <http://gplearning.racgp.org.au> and answer the following 10 multiple choice questions (MCQs) online
- 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 <http://gplearning.racgp.org.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.**

**CASE 1 – MABEL**

Mabel, aged 72 years, has had type 2 diabetes mellitus (T2DM) for the past 18 years, hypertension for 16 years and dyslipidaemia for 12 years. Vision in both of her eyes has deteriorated over the past 6 months, making reading and driving difficult.

**QUESTION 1**

Which of the following is the best way to manage this presentation?

- Reassure Mabel that the deterioration in her vision is normal for her age and advise her to see you again in 6 months.
- Refer Mabel to an ophthalmologist for assessment of her vision problems.
- Increase the dose of Mabel's antihypertensive medication.
- Increase the dose of Mabel's glucose-lowering medication.
- Advise Mabel to get stronger reading glasses.

**QUESTION 2**

A report from her optometrist says Mabel has central (fovea-involving) diabetic macular oedema (DMO). Which statement is correct regarding treatment for her DMO?

- The benefits of intravitreal triamcinolone acetonide injection last for approximately 1 month.

- Mabel should be referred for argon laser photocoagulation as first-line therapy.
- Optimising blood glucose levels, blood pressure and possibly blood lipid levels, is important in reducing the risk of development and/or progression of diabetic retinopathy.
- Optimising blood glucose levels, blood pressure and possibly blood lipid levels, is not important for reducing the risk of development and/or progression of diabetic retinopathy.
- Intravitreal anti-vascular endothelial growth factor (anti-VEGF) agents are last-line treatments for diabetic macular oedema.

**CASE 2 – CONNIE**

Connie is 79 years of age and is receiving end-of-life care. Her life expectancy is weeks to 1 month. Her condition has been deteriorating rapidly and she needs assistance with activities of daily living. She has difficulty eating, and nausea, significant gastrointestinal upset and problems swallowing. She has T2DM and moderate vision loss. She has also had several minor transient ischaemic attacks/strokes and endometrial cancer 10 years ago. Her medications include metformin, insulin, a statin, an angiotension converting enzyme inhibitor (ACEI) and aspirin.

**QUESTION 3**

Which one of the following statements is correct regarding Connie's blood glucose levels?

- The optimal blood glucose level for Connie is 6–15 mmol/L.
- The optimal blood glucose level for Connie is 6–20 mmol/L.
- The optimal blood glucose level for Connie is the same as that for all people with diabetes.
- Tight glycaemic control is beneficial in people with limited life expectancy.
- Poor glycaemic control does not have a negative impact on a patient's quality of life.

**CASE 3 – JOSIE**

Josie is 5 years of age and attends your clinic with her mother. Josie's mother tells you that she is very concerned about Josie's weight loss, occasional vomiting, increased thirst and polyuria over the last few weeks. When examining Josie you note abdominal pain.

**QUESTION 4**

Which of the following statements is the most correct?

- Her polydipsia is psychogenic.
- Her polyuria is due to a urinary tract infection.
- Her abdominal pain and occasional vomiting coupled with weight is due to gastroenteritis.
- The presenting symptoms are suggestive of type 1 diabetes mellitus (T1DM).
- The presenting symptoms are not suggestive of T1DM.

**QUESTION 5**

Using point-of-care testing at your clinic, you find that Josie's random blood glucose is 16 mmol/L and her blood ketones are 0.6 mmol/L. Which of the following statements is the most correct regarding the next steps you should take?

- A. A blood sample should be taken to confirm a diagnosis of T1DM.
- B. Josie should be sent for an oral glucose tolerance test (OGGT).
- C. Blood samples should be taken to confirm the random blood glucose and ketones test results.
- D. Josie should be referred for a specialist assessment over the next few weeks/month.
- E. Josie should be referred immediately (same day referral) for specialist assessment.

**QUESTION 6**

Josie was referred to the local emergency department. She was assessed and managed for ketoacidosis. Her T1DM was confirmed, insulin treatment initiated and diabetes education and support offered. Which of the statements below is the most correct regarding Josie's ongoing management?

- A. Josie and her family will require ongoing support and advice and about nutrition and healthy lifestyle practices.
- B. When Josie is older her insulin therapy could be changed to an oral anti-hypoglycaemic agent.
- C. Routine annual screening for retinopathy complications should commence immediately for Josie.
- D. Routine annual screening for nephropathy complications should commence immediately for Josie.
- E. Josie should have her lipid levels checked annually until puberty and from then every 5 years.

**CASE 4 – SIEW**

Siew is a Chinese woman aged 59 years and is a new to your practice. She tells you that she was diagnosed with T2DM 12 months ago and that she is due for her annual diabetes review. She has lost 5 kg since her diagnosis and would like to lose more weight. Her medications include metformin 2 g daily and ramipril 10 mg daily.

**QUESTION 7**

You determine that her body mass index (BMI) is 30.1 kg/m<sup>2</sup>. Which one of the following statements is correct?

- A. Siew is a good candidate for bariatric surgery.
- B. Use of a lower BMI threshold is recommended for Siew.
- C. Weight loss of 10 kg or more is required before any reduction in HbA1c levels may be observed.
- D. Siew should be able to lose weight with just a vigorous exercise program.
- E. Weight loss may improve her glycaemic control but will not improve her blood pressure.

**QUESTION 8**

A blood sample taken at the annual review showed an HBA1c of 7.7% (61 mmol/mol) and a CrCL of 73 mL/minute. What would you do now?

- A. You would do nothing.
- B. Increase Siew's dose of metformin.
- C. Increase Siew's metformin to total daily doses and if this does not improve her glycaemic control introduce a short-acting sulfonylurea.
- D. Switch Siew to another oral hypoglycaemic agent.
- E. In the short term, encourage further weight reduction and review Siew's HBA1c levels, with the view to adding a second oral hypoglycaemic agent if her HBA1c continues to be above target.

**CASE 5 – GINNY**

Ginny is 36 years of age and wants to discuss laparoscopic adjustable gastric band (LAGB) surgery after watching a documentary. Her BMI is 37.6 kg/m<sup>2</sup> and her weight is the highest she has experienced. Recently she was started on metformin for her T2DM, a statin for her lipids and a diuretic to control her blood pressure.

**QUESTION 9**

Which statement is the correct regarding LAGB surgery for Ginny?

- A. Ginny does not qualify for LAGB surgery.
- B. To qualify for LAGB surgery Ginny's BMI needs to be >40 kg/m<sup>2</sup>.
- C. Ginny's diabetes might improve or remit if she had LAGB surgery.
- D. There would be no change in Ginny's diabetes status as LAGB surgery does not improve diabetes outcomes.
- E. If Ginny were 17 years of age LAGB surgery would be contraindicated for her.

**QUESTION 10**

Ginny comes back for a routine Pap smear. She is scheduled for LAGB surgery and is keen to discuss the risk and benefits of surgery with you again. Which statement is correct?

- A. Weight loss of 2–3 kg per week is normal following LAGB surgery.
- B. Surgical mortality is estimated at 10–20/10,000.
- C. The risk of long-term complications is 3% over 15 years.
- D. Weight loss following LAGB surgery has been shown to be maintained for up to 15 years.
- E. There are no long-term complications following LAGB surgery.

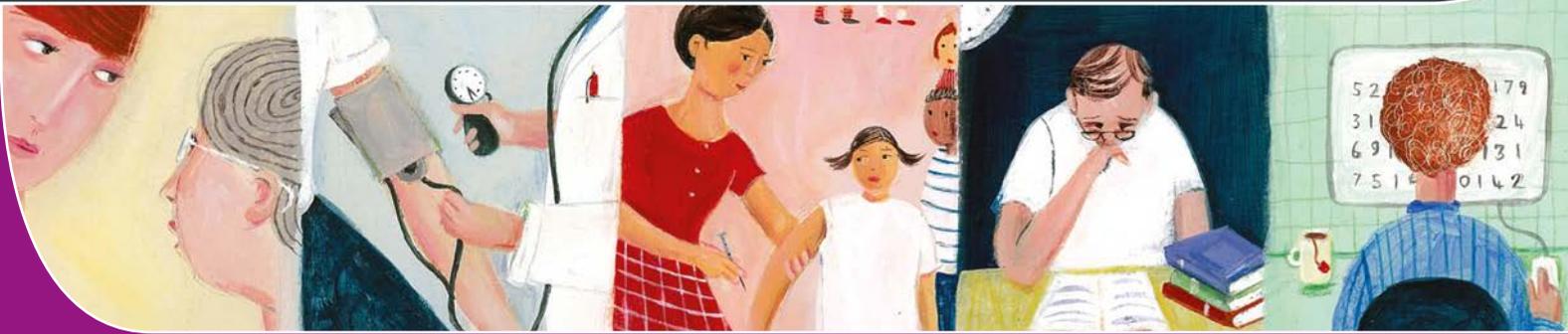


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Independent learning program for GPs

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Unit 511 November 2014

# GP health and wellbeing

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Whilst the text is directed to health professionals possessing appropriate qualifications and skills in ascertaining and discharging their professional (including legal) duties, it is not to be regarded as clinical advice and, in particular, is no substitute for a full examination and consideration of medical history in reaching a diagnosis and treatment based on accepted clinical practices.

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Independent learning program for GPs



## GP health and wellbeing

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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

### ABOUT THIS ACTIVITY

Doctors enjoy good general health<sup>1</sup> but are vulnerable to burnout<sup>2</sup> through their exposure to various stressors and pressures in their role as healthcare providers. This unit of *check* focuses on issues of general practitioner health and wellbeing, and provides education about the importance of seeking formal healthcare when necessary. It aims to foster a supportive, non-judgemental culture that instils confidence in their ability to access appropriate avenues of help.

### LEARNING OUTCOMES

At the end of this activity participants will be able to:

- describe situations that require mandatory reporting of a doctor
- explain how a complaint may affect a doctor's health and wellbeing
- outline best practice management of a needlestick injury sustained by a doctor
- discuss potential problems that have been identified when doctors self-treat
- discuss strategies that may help doctors in practice to accommodate the process of ageing.

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### REFERENCES

1. Australian Medical Association. Health and wellbeing of doctors and medical students – 2011. Canberra: AMA. 2011. Available at <https://ama.com.au/position-statement/health-and-wellbeing-doctors-and-medical-students-2011> [Accessed 15 May2014].
2. Cooke GPE, Doust, JA, Steele, MC. A survey of resilience, burnout and tolerance of uncertainty in Australian general practice registrars. BMC Med Ed 2013;13:2–6.

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

AHPRA	Australian Health Practitioner Regulation Agency	HCV	hepatitis C virus
BSL	blood sugar level	HIV	human immunodeficiency virus
GPCOG	GP assessment of cognition	MDO	medical defence organisation
HBV	hepatitis B virus	PEP	post-exposure prophylaxis

**CASE 1**

**DR ANN HAS A FEW DRINKS AT LUNCH**

Dr Ann is a GP aged 40 years. Her colleagues are aware she is going through an acrimonious divorce and she has mentioned she is not sleeping or eating well. She has been drinking more alcohol than usual.

One day she has a few drinks at lunch before her afternoon session. The practice manager receives a complaint from the patient attending the first appointment after lunch. The patient reports she smelt alcohol on Dr Ann, who was also dishevelled, vague and distracted throughout the consultation. The patient says that she had previously respected Dr Ann but the practice must do something or she will complain to the Medical Board of Australia (the Board) if this happens again. The practice manager, a trained nurse, immediately talks to Dr Fred, the senior doctor in the practice.

**QUESTION 1**  

What should Dr Fred do?

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**FURTHER INFORMATION**

Dr Fred interrupts his afternoon session to go to Dr Ann's office and finds his colleague smells of alcohol and looks the worse for wear. Dr Ann admits she had a few glasses of wine at lunch and says she had forgotten she was working that afternoon. Dr Ann then becomes tearful and tells Dr Fred she thinks she is

depressed and doesn't know what to do. Dr Fred advises Dr Ann to go home now. He asks her how she is feeling, and whether she will be ok at home, or if he should phone a friend for her. Dr Ann says she thinks she will be ok and her daughter will be home from university later that day. Dr Ann's appointments for that afternoon and the next day are cancelled, and her patients are advised that she is unwell.

The next day Fred meets with Ann to discuss the situation. He tells her he may need to inform the Board via the Australian Health Practitioner Regulation Agency (AHPRA) of what has happened.

**QUESTION 2**  

What is the role of the Board? How does it relate to AHPRA?

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**QUESTION 3**   

Under what circumstances must Dr Fred make a report to AHPRA? Is the practice manager required to make a report to AHPRA?

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**QUESTION 4**   

If Dr Fred had not observed Dr Ann himself, can he rely on someone else's observations?

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**QUESTION 5**   

What are the exceptions to the requirements for making a mandatory notification?

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**QUESTION 6**  

Given the exceptions to mandatory reporting requirements, with whom can Fred discuss this situation? Are there any other options for Dr Fred and Dr Ann?

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**FURTHER INFORMATION**

Dr Ann contacts her medical defence organisation (MDO) and with their assistance makes a self-notification to AHPRA, outlining her difficult circumstances, depression and alcohol abuse. Dr Ann decides to take a short break from work. She then consults her doctor, who confirms the diagnosis of depression and prescribes antidepressant treatment. Dr Ann decides to stop drinking alcohol.

**QUESTION 7**  

What is 'impairment' under the Health Practitioner Regulation National Law (National Law)?

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**QUESTION 8**  

What is the likely outcome of a notification of an impaired medical practitioner to AHPRA?

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**QUESTION 9**   

What are the possible consequences if neither Dr Fred nor Dr Ann makes a notification to AHPRA? Is there any protection for people making a notification?

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**QUESTION 10**  

If Dr Ann saw her doctor about her depression and commenced treatment for it, is her treating doctor obliged to make a mandatory report about Dr Ann's impairment?

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**CASE 1 ANSWERS**

**ANSWER 1**

This is a very difficult situation. It would be preferable for Dr Fred to:

- tell Dr Ann that a patient has made a complaint against her and provide details of the complaint
- give Dr Ann an opportunity to explain what happened at the consultation
- offer support for Dr Ann.

This course of action may be possible if they have a good relationship but under some circumstances it may be difficult.

**ANSWER 2**

The core role of the Board<sup>1</sup> and AHPRA<sup>2</sup> is to protect the public.

The Board is one of 14 national boards regulating registered health practitioners in Australia. As well as their core role to protect the public, national boards set the standards that practitioners must meet, and manage notifications (complaints) about the health, conduct or performance of health practitioners.

AHPRA works in partnership with the national boards to implement the National Registration and Accreditation Scheme under the Health Practitioner Regulation National Law (the National Law), which is in force in each state and territory.

**ANSWER 3**

According to AHPRA, 'All registered health practitioners have a professional and ethical obligation to protect and promote public health and safe healthcare. Under the National Law, health practitioners, employers and education providers have some mandatory reporting responsibilities.'<sup>1</sup>

The obligation is on any health practitioner or employer who forms a reasonable belief that another health practitioner has engaged in notifiable conduct to make a report to AHPRA as soon as practicable.<sup>2</sup>

Notifiable conduct is defined as:<sup>3-5</sup>

- practising while intoxicated by drugs or alcohol
- engaging in sexual misconduct
- placing the public at risk of substantial harm because of an impairment
- placing the public at risk because of a significant departure from accepted professional standards.

If Dr Fred considers that Dr Ann was intoxicated while at work, then that would be notifiable conduct and Fred has an obligation to inform AHPRA.

Mandatory reporting obligations apply to all registered health practitioners and not only to those in the same profession as the person making the report.<sup>3,5</sup> The National Law applies to various health professionals, including nurses (and midwives) and doctors.<sup>4</sup> Hence, if the practice nurse formed the view that Ann was intoxicated at work, then she would have an obligation to inform the Board. It is not necessary for both the practice nurse and Fred to make a mandatory notification, as an exception arises if someone else has already made a notification.

**ANSWER 4**

Making a notification to AHPRA about another health practitioner is a serious step to take. Fred must have formed a 'reasonable belief' that notifiable conduct occurred.<sup>4-6</sup> Speculation, rumours and gossip would not be enough to form a reasonable belief. A reasonable belief requires a stronger level of knowledge, such as direct knowledge or observation of the behaviour, or a report from a reliable source. If Fred had not observed Ann, he would need to consider whether his informant (in this case the practice manager) is reliable and, if so, that may be enough for him to form a belief that Ann has been seeing patients while intoxicated.

**ANSWER 5**

There are exceptions to the requirements to make a mandatory notification. Exceptions arise where the health practitioner who would be required to make the notification:<sup>5</sup>

- reasonably believes that someone else has already made a notification
- is employed or engaged by a professional indemnity insurer, and forms the belief because of a disclosure in the course of a legal proceeding or the provision of legal advice arising from the insurance policy
- forms the belief while providing advice about legal proceedings or the preparation of legal advice
- is exercising functions as a member of a quality assurance committee, council or other similar body approved or authorised under legislation which prohibits the disclosure of the information
- is a treating practitioner in Western Australia
- is a treating practitioner in Queensland, under some circumstances.

**ANSWER 6**

Fred can ring his MDO and discuss the situation with one of their staff. The discussion would be confidential and the doctor employed by the MDO will not be under any mandatory reporting obligation.

One of the exceptions is that if Fred believes that someone else has notified the relevant authorities about Ann's conduct, then he does not need to report her. Fred could give Ann the option to self-report and if she agrees, Fred would no longer have a mandatory obligation to report his colleague. He may want to document his discussion and this document should be stored securely.

### ANSWER 7

Impairment is defined in the National Law to mean a person has a physical or mental impairment, disability, condition or a disorder (including substance abuse or dependence) that detrimentally affects, or is likely to detrimentally affect, the person's capacity to practise as a doctor.<sup>4,7</sup>

### ANSWER 8

AHPRA and the Board take all notifications seriously. After undertaking a preliminary assessment of a notification, if the Board decides that a doctor may be impaired and further action is necessary, they may:<sup>8</sup>

- take immediate action, which may include suspending a doctor's registration, imposing conditions, accepting undertakings or accepting the surrender of registration
- require the doctor to undergo a health assessment
- refer the matter to a health panel.

The health assessment is conducted by an experienced and appropriately qualified, independent medical practitioner or psychologist. The Board pays for the assessment and the assessor writes a report for the Board.<sup>8</sup>

The doctor who was assessed is usually given a copy of the report, unless the Board believes it contains information that may be prejudicial to the doctor's health or wellbeing, in which case it is given to a doctor or psychologist nominated by the doctor. After receiving the report, the doctor who was assessed must discuss the report and ways of dealing with any adverse findings with a person nominated by the Board. The person nominated to discuss the report will be a registered medical practitioner.

If the Board believes that a practitioner's health is impaired, it can take one or more of the following courses of action:<sup>8</sup>

- caution the doctor
- accept an undertaking from them
- impose conditions on the practitioner's registration.

In Dr Ann's case, the Board may accept an undertaking from her. Such an undertaking would usually include Ann seeing her psychiatrist (if she is seeing one) and her GP regularly. Her treating doctor would provide the Board with reports. In some situations, the undertaking may be to undergo drug or alcohol testing.

### ANSWER 9

Any practitioner who fails to make a mandatory notification when required may be subject to an investigation and possible action by their medical board. However, no penalties are prescribed under the National Law for a practitioner who fails to make a mandatory notification. However,<sup>6</sup> In Dr Ann's case, if the patient makes a complaint to AHPRA about Ann, then AHPRA may investigate.

The National Law protects doctors who make a notification in good faith, which means well-intentioned or without malice.<sup>4,5</sup> Doctors who make mandatory notifications in good faith are protected from civil, criminal and administrative liability, including defamation. The National Law clarifies that making a notification is not a breach of professional etiquette or ethics, or a departure from accepted standards of professional conduct. There is no provision for confidential reporting and the doctor is likely to be aware of who made the report. Note that legally mandated notification requirements override privacy laws. However, doctors who make notifications that are frivolous, vexatious or not in good faith may be subject to conduct action.<sup>5</sup>

### ANSWER 10

To trigger a mandatory report regarding a doctor's impairment, the doctor must have placed the public at risk of substantial harm because of the impairment.<sup>5,7</sup> Impairment alone does not require a mandatory report. Therefore if Ann is able to practice safely then there is no need for a notification. If Ann is too unwell to work but she follows her doctor's advice, has some time off and returns to work when her treating doctor feels that she is well enough, then there is no need for a mandatory report.

### REFERENCES

1. Medical Board of Australia. Available at [www.medicalboard.gov.au](http://www.medicalboard.gov.au) [Accessed 11 August 2014].
2. Australian Health Practitioner Regulation Agency. Available at [www.ahpra.gov.au](http://www.ahpra.gov.au) [Accessed 11 August 2014].
3. Australian Health Practitioner Regulation Agency. Mandatory notifications. Available at [www.ahpra.gov.au/Notifications/Who-can-make-a-notification/Mandatory-notifications.aspx](http://www.ahpra.gov.au/Notifications/Who-can-make-a-notification/Mandatory-notifications.aspx) [Accessed 11 August 2014].
4. Bird S. Mandatory reporting of health practitioners: notifiable conduct. *Aust Fam Physician* 2010;39:593–94.
5. Medical Board of Australia. National Board guidelines for registered health practitioners. Guidelines for mandatory reporting. March 2014. Available at [www.medicalboard.gov.au/Codes-Guidelines-Policies/Guidelines-for-mandatory-notifications.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies/Guidelines-for-mandatory-notifications.aspx) [Accessed 11 August 2014].
6. Australian Health Practitioner Regulation Agency. Legal practice note: reasonable belief. LPN 11 (10 August 2012). Available at [www.ahpra.gov.au/Search.aspx?q=reasonable%20belief](http://www.ahpra.gov.au/Search.aspx?q=reasonable%20belief) [Accessed 11 August 2014].
7. Australian Health Practitioner Regulation Agency. Legal practice note: practitioners and students with impairment. LPN 12 (10 August 2012). Available at [www.ahpra.gov.au/Search.aspx?q=impairment](http://www.ahpra.gov.au/Search.aspx?q=impairment) [Accessed 11 August 2014].
8. Australian Health Practitioner Regulation Agency. The notification process. Available at [www.ahpra.gov.au/Notifications/The-notifications-process.aspx](http://www.ahpra.gov.au/Notifications/The-notifications-process.aspx) [Accessed 11 August 2014].

### RESOURCES FOR DOCTORS

- The Medical Board of Australia website has information sheets on notifications, management of impaired practitioners and students, and panel hearings, [www.medicalboard.gov.au](http://www.medicalboard.gov.au)
- Medical Board of Australia. Good Medical Practice: a Code of Conduct for Doctors in Australia, [www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx)
- National Board guidelines for registered health practitioners: guidelines for mandatory notifications, [www.medicalboard.gov.au/Codes-Guidelines-Policies/Guidelines-for-mandatory-notifications.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies/Guidelines-for-mandatory-notifications.aspx)

**CASE 2**

**DR MARY RECEIVES A COMPLAINT**

Dr Mary has received a letter of complaint from a long-term patient, Mrs Jones, who was diagnosed with breast cancer 1 month ago following a routine mammogram. The breast clinic informed Mrs Jones that the cancer had been detected 1 year ago when she had a mammogram that Dr Mary had ordered. Mrs Jones is angry because Dr Mary never informed her of the result. The cancer is now in an advanced stage, requiring surgery and chemotherapy, and has a poorer prognosis than if treatment had commenced earlier. In her letter, Mrs Jones says she will inform the Medical Board of Australia (the Board) and intends to sue Dr Mary for malpractice.

**QUESTION 1** 

What impact can such a complaint have on a doctor?

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**QUESTION 2** 

How common is it to receive a complaint?

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**QUESTION 3** 

What should Dr Mary do? Should she respond to the letter?

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**FURTHER INFORMATION**

Dr Mary and her practice manager look back through the records to find out what happened. They discovered they received the abnormal result and that Mrs Jones had made an appointment to see Dr Mary. The electronic records showed that Mrs Jones arrived at the practice for her appointment, but her medical records showed no clinical consultation took place. Dr Mary and the practice manager assumed Mrs Jones left before being seen. It was a particularly chaotic day and there were a number of emergencies. A follow-up appointment was not made.

**QUESTION 4**  

What should be included in the letter to Mrs Jones?

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**QUESTION 5** 

What is the natural history of recovery for the 'second victim'?

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**QUESTION 6** 

What strategies might help Dr Mary to cope?

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**FURTHER INFORMATION**

After the letter has been sent to Mrs Jones, Dr Mary and her practice manager organise a practice meeting with the staff to review how the practice handles abnormal results and follow-up appointments. They review all of their processes, consider what can go wrong, and put in place extra checks and a patient-recall system to circumvent future similar mishaps.

**CASE 2 ANSWERS**

**ANSWER 1**

Most doctors choose their profession because they want to improve the lives of others. Errors resulting in patient harm are distressing for the doctor concerned. Studies have reported on the emotional effect of adverse events on doctors. The term ‘second victim’ has been used to describe the healthcare professional, who may be traumatised by such events. The ‘first victim’ is always the patient.<sup>1</sup>

Commonly reported reactions among health professionals following adverse events are fear, guilt, shame, self-doubt, anger and disappointment.<sup>2</sup> Doctors with a current medico-legal matter have a higher prevalence of psychiatric morbidity, compared with those with no current matter. A study of 3171 physicians in internal medicine, paediatrics, family medicine and surgery in the USA and Canada reported that following errors, 61% of physicians described increased anxiety about future errors, 44% described loss of self-confidence, 42% experienced difficulty sleeping, 42% reported reduced job satisfaction and 13% reported harm to their reputation.<sup>3</sup>

Many doctors also experience periods of re-enactment of their errors, often with feelings of inadequacy, and of self-isolation and ‘what if’ questions.<sup>4</sup>

**ANSWER 2**

All medical practitioners can expect to receive at least one claim or complaint at some time in their professional career. In 2012–2013

the Australian Health Practitioner Regulation Agency (AHPRA) received notifications regarding 4.2% of medical practitioners.<sup>5</sup> The rate of claims against doctors in the private sector during that period was 3.4%.<sup>6</sup> It is hard to obtain data regarding direct patient complaints, and of course some of these complaints and claims may relate to the same doctors, but when considered over a 40-year career period, most doctors are going to receive at least one claim or complaint in their career. The threat of a medical negligence claim is commonly one of the most severe sources of stress in a doctor’s working life.

In the 2012–2013 financial year, AHPRA reported that approximately 1 in 25 (4.2%) of Australia’s 95,690 medical professionals received a notification of a complaint from the Board. The number of complaints is increasing and AHPRA reported a 14% increase in notifications across all professions over the 2012–2013 period.<sup>5</sup> Doctors received more complaints than other health professionals: doctors made up 16% of health practitioners in 2012–2013 but they received 54% of complaints.<sup>5</sup>

**ANSWER 3**

Dr Mary should immediately inform her medical defence organisation (MDO) that she has received this letter. Doctors should inform their MDO of all letters of complaint and seek the MDO’s assistance in responding to them. Responses to letters of complaint should be thorough, considered and compassionate.

**ANSWER 4**

With the help of the claims manager at her MDO, Dr Mary drafts a letter to Mrs Jones. In the letter, Mary expresses sympathy with the diagnosis of breast cancer. She explains that Mrs Jones’s follow-up appointment was marked in red for review of abnormal results and that her attendance at the practice was recorded. Unfortunately, it seems she did not wait to be seen and the practice did not have a system to flag that situation.

**ANSWER 5**

Most doctors who are the subject of a claim will have an emotional reaction. After the initial disbelief and denial, there may be anxiety and self-doubt. As the matter progresses, disbelief may be replaced with anger and resentment.<sup>1,2</sup> Of the doctors involved in claims for medical negligence, 16% may describe the onset or exacerbation of a previously diagnosed physical illness and 2% experience suicidal ideation.<sup>7</sup> Many doctors develop doubts about their clinical competence and lose confidence, so it may take them longer than usual to see patients and they find clinical practice more draining than previously.<sup>8</sup>

Although many doctors contemplate leaving medicine or changing the scope of their practice in response to a complaint, most are able to deal with these reactions and reach a resolution of symptoms within a reasonable time frame. For example, in a study of 21 health professionals, the majority described the impact of their error as long lasting, but the reported duration of impact ranged from a few months to 1 year or more.<sup>2</sup>

**ANSWER 6**

The following strategies might help Dr Mary to cope:

- **Emotional support**

Most doctors in this situation value being able to talk about the complaint and the adverse event.<sup>2</sup> A trusted colleague, friend or partner may be able to listen with a sympathetic ear. The MDO may also provide support. A claim or complaint can have physical and emotional effects so it is important that the doctor maintain contact with their own GP. The doctor's GP will be a valuable source of support concerning the doctor's wellbeing and provide a referral if further formal counselling or psychiatric assessment is needed.

- **Surviving the legal/investigative process**

The legal analysis of a medical complaint is not equivalent to a medical workup or the investigation of a patient. The processes are often unpredictable and it may take years to reach resolution. In this case, it is unclear whether this will be a medical board complaint or a claim or both. There can be a flurry of activity followed by months when nothing happens. These factors can cause feelings of powerlessness and frustration, and increase the difficulty in recovering from the adverse event and reaching closure.<sup>2</sup> The doctor's MDO should be a valuable source of information about the process, the likely course of the investigation and progress of the doctor's particular claim or complaint.

- **Making sense of the personal meaning of the claim or complaint**

After an adverse event, doctors may feel insecure in their professional roles. They may have a sense of shame and a belief they are 'bad' or incompetent. Many doctors discover that these feelings affect their work to some extent and some find they take extra care in performing their work to avoid problems.<sup>1,2</sup> It can be helpful to reflect on complaints and determine if the adverse event could have been prevented in some way. It is always helpful to reflect on whether changes could be made to improve practice processes.

**CONCLUSION**

Even the most experienced and competent medical practitioner can become involved in medical negligence claims and complaints. They can occur at any time in a doctor's career. Systems failure (as in this case) and issues with communication can often be important contributing factors. Most published studies in this area highlight that collegial support is very helpful in assisting doctors to navigate this period in their lives.<sup>2,4,9</sup>

**REFERENCES**

1. Wu AW. Medical Error: The second victim. The doctor who makes the mistake needs help too. *BMJ* 2000;320:726–27.
2. Ullstrom S, Andreen Sachs M, Hansson J, Ovretveit J, Brommels M. Suffering in silence: a qualitative study of second victims of adverse events. *BMJ Qual Saf* 2014;23:325–31.
3. Waterman AD, Garbutt J, Hazel E, et al. The emotional impact of medical errors on practicing physicians in the United States and Canada. *Jt Comm J Qual Patient Saf* 2007;33:467–76.
4. Scott SD, Hirschinger LE, Cox KR, McCoig M, Brandt J, Hall LW. The natural history of recovery for the healthcare provider "second victim" after adverse patient events. *Qual Saf Health Care* 2009;18:325–30.
5. Australian Health Practitioner Regulation Agency. 2012–13 Annual report: AHPRA and National Boards. Available at [www.ahpra.gov.au/Publications/Corporate-publications.aspx](http://www.ahpra.gov.au/Publications/Corporate-publications.aspx) [Accessed 12 August 2014].
6. Australian Institute of Health and Welfare. Australia's medical indemnity claims 2012-2013. Canberra: AIHW, 2014.
7. Charles SC, Wilbert JR, Kennedy EC. Physicians' self-reports of reactions to malpractice litigation. *Am J Psychiatr* 1984;141:563–65.
8. Jain A, Ogden J. General practitioners' experiences of patients' complaints: qualitative study. *BMJ* 1999;318:1596–99.
9. Hu YY, Fix ML, Hevelone ND, et al. Physicians' needs in coping with emotional stressors: the case for peer support. *Arch Surg* 2012;147:212–27.



and baseline blood tests, provide you with a 28-day course of HIV PEP, advise that you do not need HBV PEP, and arrange follow-up over the next 3 months. They tell you that until follow-up serology is complete you must take precautions, including practising safe sex, and should not donate blood, sperm or organs. Apart from experiencing a 'hangover' for the first few days of treatment you manage well and feel ready to resume work.

**QUESTION 6** 

Can you continue working as a GP while you wait for your final serology testing?

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**CASE 3 ANSWERS**

**ANSWER 1**

General practice staff are at risk of infection through exposure to:<sup>1</sup>

- aerosols or respiratory droplets
- contaminated surfaces or materials
- mucous membranes, blood (eg through needlestick injury) or body fluids.

Preventive activities depend on the risk involved and are outlined in the Royal Australian College of General Practitioners' guidelines *Infection prevention and control standards*.<sup>1</sup>

All blood and body fluids should be considered potentially infectious. For minor surgical procedures the following precautions are recommended:<sup>1</sup>

- Use standard aseptic technique.
- Use personal protective equipment, including single-use gloves and goggles and, where appropriate, impermeable gowns and facemasks.
- Use a no-touch technique, which aims to avoid direct contact between the health professional's hands and the patient during the procedure by using forceps when applying dressings or using clean single-use gloves if a no touch technique is not possible (eg probing a penetrating wound).
- Ensure safe handling and disposal of sharps.
- Ensure safe processing, sterilisation and tracking of reusable equipment.
- Have a written practice policy on managing sharps and exposure to blood and body fluids.

**ANSWER 2**

Approximately 26,000 people were reported to be living with the HIV in Australia at the end of 2012. HIV rates were reported to be slightly lower in Australia, compared with the United Kingdom in 2011, and several fold lower than rates in the United States in 2009.<sup>2</sup>

Approximately 207,000 people were reported to be living with chronic HBV in Australia in 2012 (estimated prevalence 0.9%),<sup>2</sup> the majority of whom migrated to Australia from countries of high prevalence.<sup>3</sup> Chronic hepatitis C virus (HCV) affects an estimated 230,000 Australians and is usually acquired through injecting drug use.<sup>2</sup>

People at most risk of HIV are men who have sex with men, and people from high prevalence countries and their partners. HBV and HCV transmission, however, continue to occur predominantly among those with a recent history of injecting drug use.<sup>2</sup>

**ANSWER 3**

The needle is contaminated with your blood and Andrew's blood. You need to stop the procedure, acknowledge what has happened and dispose of the needle and syringe.

You also need to decontaminate the exposed area immediately. This is best achieved by washing your hands with soap or standard handwash and water, and managing your wound. Use of caustic agents such as bleach for skin washing is not recommended as these may compromise the integrity of the skin.<sup>1</sup> Injection of antiseptics and disinfectants into wounds is also not recommended.<sup>4</sup> Exposure to mucous membranes is treated by rinsing the contaminated areas with water or saline.<sup>1</sup>

It may be appropriate to ask a colleague to assist with caring for your patient while you clean up and decontaminate, particularly if the patient has a bleeding wound, or if you will not be able to complete the procedure.

**ANSWER 4**

You will need to ask Andrew a number of personal questions. First ask any other people present to leave the room. Then explain the risks of infection through exposure to blood and other body fluids and that you need to ask Andrew about his exposure to or presence of bloodborne viruses such as HIV and hepatitis, acknowledging that it can be difficult to disclose this information.

Say you would like to run through a list of risk factors for bloodborne viruses,<sup>4-6</sup> and ask Andrew to identify any that apply to him, such as:

- having lived in, or having had a sexual partner from, a country with a high prevalence of HIV or viral hepatitis
- male-to-male sexual activity
- blood transfusions, dental procedures or surgery performed overseas
- blood transfusions in Australia prior to May 1990
- past or current injecting drug use
- piercings or tattoos performed outside of Australia or in a non-professional setting
- known previous exposure to a bloodborne viruses or previous incarceration.

Ask Andrew directly if he has ever been diagnosed with HIV or hepatitis. If not, ask if he will agree to be tested today. You could ask if he had been tested before or given a blood donation in Australia and, if so, when this took place.

Guidelines for initial risk assessment are included in the RACGP's *Infection prevention and control standards*.<sup>1</sup> It should be emphasised that where the risk of infection is high, preventive activities may need to be started within 3 days<sup>4,7</sup> and advice should be sought as soon as possible from either a sexual health physician or infectious diseases physician. Seeking advice from specialists should not be delayed by waiting for laboratory results as needlestick/sharps injury with fresh blood poses a significant risk.

### ANSWER 5

PEP stands for 'post-exposure prophylaxis' and is any preventive medical treatment started immediately after exposure to a pathogen. Its use is time-dependent.<sup>4,7</sup>

HIV PEP is a short course of HIV-antiretroviral therapy taken to reduce the risk of seroconversion. Where indicated, it should be started as soon as possible (ie do not delay use while establishing the source's HIV status), and within 72 hours of exposure.<sup>4</sup> PEP can be accessed from sexual health centres and hospital emergency departments. To find your nearest service you can call the PEP hotline for your state or territory on:

- NSW 1800 737 669
- VIC 1800 889 887
- SA 1800 022 226
- TAS 1800 005 900
- WA 1300 767 161
- QLD (healthline) 134 325 84.

Alternatively, check an online resource such as [www.getpep.info/where.html](http://www.getpep.info/where.html).

HBV PEP is anti-HBV immunoglobulin and/or immunisation, depending on immunisation status and serological testing. People with documented positive HBV surface antibody do not require HBV PEP.<sup>4,7</sup>

There is no HCV PEP, so management consists of monitoring for seroconversion, with rapid referral if this occurs.<sup>4,8</sup> New oral antiviral therapies for HCV have prompted interest in these agents as PEP and clinical trials are ongoing.

In conclusion, the risk of bloodborne viruses depends on the nature of the exposure and the risk that the source person was infected and able to pass on their infection. Exposures need to be assessed on a case-by-case basis to determine an appropriate management plan, including use of PEP.<sup>4</sup> The estimated risk of HIV transmission/exposure with a known HIV-positive source in the setting of a needlestick injury or other sharps exposure is 1 in 440, based on prospective studies.<sup>4</sup>

### ANSWER 6

National guidelines govern the activities of healthcare workers known to be infected with bloodborne viruses.<sup>9</sup> For healthcare workers who have potentially been exposed, modification of work practices (ie

avoidance of exposure-prone procedures) is recommended only in the case of percutaneous exposures with BOTH exposure to a large volume of blood AND exposure to blood containing high titre of HIV, HCV or HBV.<sup>1,10</sup> Note, exposure-prone procedures are defined as procedures where there is a risk of injury to the healthcare worker resulting in exposure of the patient's open tissues to the blood of the worker (eg contact with sharp instruments, needle tips or sharp tissues).<sup>9</sup> Individual medical insurers may have specific requirements in order to maintain insurance cover. In this case, you can continue working and should not need to modify your work practices, but should call your insurer to clarify any requirements they may have.

### REFERENCES

1. Royal Australian College of General Practice. *Infection prevention and control standards: for general practices and other office-based and community-based practices*, 5th edition. East Melbourne: RACGP, 2014. Available at [www.racgp.org.au/your-practice/standards/infectioncontrol](http://www.racgp.org.au/your-practice/standards/infectioncontrol) [Accessed 5 August 2014].
2. The Kirby Institute. *HIV, viral hepatitis and sexually transmissible infections in Australia: Annual Surveillance Report 2013*. Sydney: The Kirby Institute, the University of New South Wales, 2013. Available at [www.ashm.org.au/images/Media/ASR2013.pdf](http://www.ashm.org.au/images/Media/ASR2013.pdf) [Accessed 5 August 2014].
3. MacLachlan JH, Allerd N, Towell V, Cowie BC. The burden of chronic hepatitis B virus infection in Australia, 2011. *Aust N Z J Pub Health* 2013;37:416–22.
4. Australian Society for HIV Medicine. *National guidelines for post-exposure prophylaxis after non-occupational and occupational exposure to HIV*. Darlinghurst: Australasian Society for HIV Medicine, 2013. Available at [www.ashm.org.au/pep-guidelines/NPEPEPGuidelinesDec2013.pdf](http://www.ashm.org.au/pep-guidelines/NPEPEPGuidelinesDec2013.pdf) [Accessed 6 August 2014].
5. O'Sullivan BG, Gidding HF, Law M, Kaldor JM, Gilbert GL, Dore GJ. Estimates of chronic hepatitis B virus infection in Australia. *Aust N Z J Public Health*. 2004;28:212–16.
6. Siebert DJ, Breschkin AM, Bowden DS, Locarnini SA. Hepatitis C: diagnosis and monitoring. *Aust Prescr* 1999;22:91–94.
7. Department of Health. *Australian Immunisation Handbook*. 10th edition. Canberra: Commonwealth of Australia, 2013. Available at [www.health.gov.au/internet/immunise/publishing.nsf/Content/EE1905BC65D40BCFCA257B26007FC8CA/\\$File/handbook-Jan2014v2.pdf](http://www.health.gov.au/internet/immunise/publishing.nsf/Content/EE1905BC65D40BCFCA257B26007FC8CA/$File/handbook-Jan2014v2.pdf) [Accessed 27 August 2014].
8. Centers for Disease Control and Prevention (CDC). Updated US Public Health Service guidelines for the management of occupational exposures to HBV, HCV and HIV and recommendations for post-exposure prophylaxis. Atlanta: CDC, 2013. Available at [www.cdc.gov/mmwr/preview/mmwrhtml/rr5011a1.htm](http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5011a1.htm) [Accessed 5 August 2014].
9. Communicable Diseases Network Australia. *Australian national guidelines for the management of healthcare workers known to be infected with bloodborne viruses*. Canberra: Australian Government Department of Health and Ageing, 2012. Available at [www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdna-bloodborne.htm](http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdna-bloodborne.htm) [Accessed 5 August 2014].
10. New South Wales Government Ministry of Health. *Health Policy Directive: HIV, hepatitis B and hepatitis C – management of health care workers potentially exposed*. NSW Government Health PD2005\_311, 2005. Available at [www.health.nsw.gov.au/policies/pd/2005/pdf/PD2005\\_311.pdf](http://www.health.nsw.gov.au/policies/pd/2005/pdf/PD2005_311.pdf) [Accessed 5 August 2014].

### RESOURCES FOR PATIENTS AND DOCTORS

- The Australian Society for HIV Medicine provides information on their website for PEP, [www.ashm.org.au/default2.asp?active\\_page\\_id=494](http://www.ashm.org.au/default2.asp?active_page_id=494)
- Victorian Aids Council, [www.getpep.info](http://www.getpep.info)

**CASE 4**

**DR GEORGE IS NOT WELL**

Dr George, 48 years of age, is a busy GP who has worked in the same practice for many years. Lately he has not been feeling well. Since his wife left him 6 months ago he has not been sleeping or eating well, and is drinking more alcohol. He is unable to sleep unless he has drunk most of a bottle of wine and even then his sleep is disturbed. He awakes early and feels tired and lethargic during the day. He eats takeaway meals most evenings and has gained weight.

One day at work, Dr George does a fingerprick test and finds that his blood sugar level (BSL) is 12 mmol/L. Later that day in the staff room, he mentions his BSL to his friend and colleague Dr James and asks 'Do you think I should start myself on some metformin or have a formal blood test and check some other things? I've no idea what my cholesterol is. I have been feeling pretty ordinary lately and perhaps it's due to my blood sugar. What do you think?'

**QUESTION 1** 

How should Dr James respond? What are some of the problems associated with providing medical advice in this setting?

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**FURTHER INFORMATION**

Dr George does not look happy with Dr James's response. George mentions that he doesn't have a GP and doesn't think that he really has time for 'that sort of thing'.

**QUESTION 2** 

What are some of the problems with self-treatment?

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**QUESTION 3** 

Are there any legal barriers to self-prescribing?

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**QUESTION 4** 

What are some of the barriers to doctors seeking medical help?

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**FURTHER INFORMATION**

Dr James recommends a GP (Dr John) for Dr George, who is experienced in treating doctor-patients. He suggests that Dr George rings him now to make a long appointment to discuss his health. Dr George still looks uncomfortable, but he makes an appointment and presents to Dr John.

**QUESTION 5** 

What are some of the challenges for Dr John when treating a doctor-patient?

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**QUESTION 6** 

What issues should Dr John consider when treating doctor-patient colleagues? Are there any things he might want to clarify with Dr George at the first appointment?

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## CASE 4 ANSWERS

**ANSWER 1**

George seems to be seeking advice from James without a formal clinical consultation occurring. This is known as a 'corridor' or 'curbside' consultation. Advice provided in this setting is often inaccurate or incomplete and rarely best practice.<sup>1</sup>

James has received a very incomplete history from George. James may feel embarrassed to seek further information and may feel he is intruding if he questions his colleague further. To ensure appropriate medical care, a doctor requires access to information of a sensitive nature, such as drug and alcohol consumption, and mental health issues. Knowledge of such issues may place James in an awkward situation if George discloses information that may affect George's ability to practice.

James has not performed a physical examination and may feel awkward about performing such an examination on a friend/colleague. A survey of 430 clinicians reported that 86% had refused to write a prescription for a friend or a family member; 65% of those not prescribing stated that the need for a physical examination strongly influenced their decision not to prescribe.<sup>2</sup>

Difficulties may also arise with documenting an informal consultation. There could be concerns regarding confidentiality within the practice where Dr George is working. Corridor consultations often result in inadequate documentation, poor or fragmented care, and lack of follow-up.<sup>3</sup>

James has noticed that George has not been himself lately and is worried about him. James replies, 'Your health is important. It sounds like you have diabetes and you need a thorough assessment. You should make an appointment to see your GP and discuss this properly'.

**ANSWER 2**

The medical profession expects patients to seek appropriate medical help when they encounter problems with their health. Yet doctors do not behave this when it comes to their own health. An Australian study found that 90% of 358 surveyed doctors felt it was acceptable to self-treat acute illnesses, and 25% felt it was acceptable to self-treat chronic illnesses.<sup>4</sup> Self-treatment by doctors, which includes diagnosing and treating as well as prescribing for oneself, is deeply ingrained in medical culture and can be acquired as early as when aspiring physicians are medical students.<sup>5,6</sup>

Self-treatment poses a number of potential risks. Avoidance of a medical consultation means a physical examination has not taken place, so the diagnosis and necessary follow-up care may not take place. There is also a risk of misdiagnosis. Doctors may neglect their health until their symptoms become serious<sup>3</sup> and often present late with serious problems.<sup>7</sup> For example, an average delay of 6 years between onset and consulting about drug and alcohol problems has been reported.<sup>7</sup> The practice of self-medication may also be a risk

factor for later substance misuse.<sup>8</sup> When treating patients, doctors generally adhere to current guidelines and best practice. However, when treating themselves, doctors may not always accept appropriate treatments, particularly for medical problems with adverse social meanings such as mental illness.<sup>9</sup> Moreover, doctors who are treating themselves are less likely to sign themselves off work and may in fact be working when they should be on sick leave.<sup>10</sup> *Good Medical Practice: A Code of Conduct for Doctors in Australia* (the Code)<sup>11</sup> encourages doctors to have a GP and to seek independent, objective advice when medical care is needed and to be aware of the risks of self-diagnosis and self-treatment.

**ANSWER 3**

Self-prescribing is a complex area and there is significant variation in the relevant legislation between Australian states and territories. Doctors need to understand and comply with the laws regulating prescription medicines that apply in the area(s) where they practice. As a general rule, self-prescribing of Schedule 8 drugs (controlled drug) is not permitted except in a very limited set of emergency situations. No self-prescribing of Schedule 4 (prescription only medicine or prescription animal remedy) or Schedule 8 medications is permitted in Victoria under any circumstances and there are restrictions on the medications that can be self-prescribed in Western Australian and the Australian Capital Territory.<sup>12</sup> Lastly, all registered doctors in Australia are bound by the Code, which specifically cautions against prescribing for oneself, family, friends or 'those you work with'.<sup>11</sup>

**ANSWER 4**

Doctors often find it difficult to seek medical assistance for various reasons, which may be real or perceived. These reasons are often referred to as 'barriers' to seeking healthcare.<sup>13</sup> For example, doctors have difficulty adopting the sick role, may feel embarrassed about consulting a doctor and may not want to inconvenience colleagues. They may also feel guilty about consulting a doctor for a minor illness. There may be concerns about confidentiality or lack of time to see a doctor. The culture of medicine is one of working through illness; an image of invincibility is encouraged and vulnerability denied. Doctors with mental health issues may have concerns that seeking treatment may affect their registration and right to practice.<sup>14</sup>

Although the Code encourages doctors to have their own GP, a 2003 Australian study of doctors' health behaviour reported that only 55% of doctors had their own GP.<sup>4</sup> A similar study in 1995 reported that 42% of doctors had their own GP.<sup>15</sup>

**ANSWER 5**

The doctor-patient is entitled to the same high standard of treatment and respect as other patients; however, this group of patients has some unique challenges.<sup>16</sup> Possible pitfalls include treating the doctor-patient more as a colleague than a patient and having higher expectations for recovery and treatment compliance. Doctors treating doctor-patients should be as thorough in their assessment, examination, explanation and follow-up of results as they would be for other patients.<sup>16</sup>

Some doctor-patients may assume 'VIP status' and try to circumvent administrative and medical regimens. This can lead to confusion, poor medical care and poor outcomes.<sup>16</sup> There can also be problems with maintaining appropriate boundaries. The Code<sup>11</sup> advises that, whenever possible, doctors should avoid providing care to people with whom they have a close personal relationship, so caution should be exercised when treating doctor-patients who are also close friends and colleagues.

## ANSWER 6

The following issues should be considered and/or clarified:<sup>16</sup>

- **Confidentiality** – Fear of breach of confidentiality can be a significant barrier to doctors seeking care and this concern could be weighing on Dr George's mind. Dr John should discuss patient confidentiality, assuring George that he will ensure confidentiality as much as possible. It is important to note that doctors' rights to confidentiality are not absolute and in some circumstances the medical board might need to be notified following a consultation with a doctor-patient.
- **History and examination** – Doctors treating doctor-patients should not avoid asking personal questions or taking a history of drug and alcohol use. The physical examination should be as thorough as for other patients and should be performed in the same clinical setting as for any other patient.
- **Prescribing, ordering and follow-up of test results** – Given the risks of self-prescribing, John assures George that he will provide all necessary prescriptions, and will order and review all of George's pathology tests. John advises George to make an appointment to see him to discuss the results of the pathology tests and at any other time if he has concerns, and not to feel in any way that he is 'wasting his time'.
- **Discuss the diagnostic and/or treatment plan in detail** – Do not assume the doctor-patient has a wealth of knowledge about their medical problem, especially if it is outside their practice area. Similarly, discuss medications in detail without assuming knowledge of doses, adverse events and related information.
- **Billing** – Some doctors may choose to bulk bill or waive their fees for doctor-patients, but this is a matter of etiquette rather than ethics. Some doctor-patients may feel more comfortable paying.

## CONCLUSION

Dr John undertakes a thorough history and examination of Dr George, and orders appropriate further testing. John counsels George about his lifestyle in general and his escalating alcohol consumption in particular. George takes a short period of leave and continues to see John regularly. George manages to reduce his alcohol consumption and change his diet, which leads to a gradual weight loss and normalisation of his BSL. When he sees John again, George comments that he is a better doctor himself as a result of going through this experience.

## REFERENCES

1. Burden M, Sarccone E, Keniston A, et al. Prospective comparison of curbside versus formal consultations. *J Hosp Med* 2013;8:31–35.
2. Walter JK, Lang CW, Ross LF. When physicians forego the doctor-patient relationship, should they elect to self-prescribe or curbside? An empirical and ethical analysis. *J Med Ethics* 2010;36:19–23.
3. Richer S. Should family physicians treat themselves or not?: No. *Can Fam Physician* 2009;55:781–82.
4. Davidson SK, Schattner PL. Doctors' health-seeking behaviour: A questionnaire survey. *Med J Aust* 2003;179:302–05.
5. Montgomery AJ, Bradley C, Rochfort A, Panagopoulou E. A review of self-medication in physicians and medical students. *Occup Med (Lond)* 2011;61:490–97.
6. Shadbolt NE. Attitudes to healthcare and self-care among junior medical officers: a preliminary report. *Med J Aust* 2002;177:S19–20.
7. Brandon S, Oxley J. Getting help for sick doctors. *BMJ* 1997;314:S2–7092.
8. Bennett J, O'Donovan D. Substance misuse by doctors, nurses and other healthcare workers. *Curr Opin Psychiatry* 2001;14:195–99.
9. Gardner M, Ogden J. Do GPs practice what they preach? A questionnaire study of GPs' treatments for themselves and their patients. *Pat Educ and Couns* 2005;56:112–15.
10. Williams ES, Manwell LB, Konrad TR, Linzer M. The relationship of organizational culture, stress, satisfaction, and burnout with physician-reported error and suboptimal patient care: results from the MEMO study. *Health Care Manage Rev* 2007;32:203–12.
11. Medical Board of Australia. Good medical practice: a code of conduct for doctors in Australia. March 2014. Available at [www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx) [Accessed 12 August 2014].
12. Australian Medical Association. Can I prescribe. 2013. Available at <https://ama.com.au/ausmed/can-i-prescribe> [Accessed 12 August 2014].
13. Australian Medical Association. Health and wellbeing of doctors and medical students. Canberra: AMA. 2011. Available at <https://ama.com.au/position-statement/health-and-wellbeing-doctors-and-medical-students-2011> [Accessed 12 August 2014].
14. *beyondblue*. National Mental Health survey of Doctors and Medical Students. Available at [www.beyondblue.org.au/docs/default-source/default-document-library/bl1132-report---nmhdms-full-report\\_web](http://www.beyondblue.org.au/docs/default-source/default-document-library/bl1132-report---nmhdms-full-report_web) [Accessed 8 October 2014].
15. Pullen D, Leonie CE, Lyle DM, Cam DE, Doughty MV. Medical care of doctors. *Med J Aust* 1995;162:481–84.
16. Schneck SA. 'Doctoring' doctors and their families. *JAMA* 1998;280:2039–42.

**CASE 5**

**PAUL IS CONCERNED**

Paul, a GP aged 67 years, presents for a check-up. You have not seen him before and he does not have a regular GP. He is a full-time procedural GP working in a small rural practice of three doctors who service the general practice clinic, a 12-bed hospital and emergency department. Paul's clinical load includes general practice, antenatal care and care of low-risk inpatients. Surgery and deliveries are no longer undertaken at the hospital. He shares in a one-in-three after-hours on call roster. He has some weekends off but rarely takes longer holidays. His wife works part-time as the practice manager.

Paul reports he is generally fit and well but often feels tired and is forgetful. He has concerns about his professional performance and is beginning to wonder how much longer he can maintain his current workload. A colleague who shares these concerns suggested he should have a full medical review. He has no localising symptoms and has undertaken a thorough batch of blood tests for himself, which he presents to you. The tests are all normal except for mildly elevated fasting lipids. He also participates in the bowel screening program and recent results were negative.

**QUESTION 1** 

What are the possible causes of lethargy and forgetfulness in this case?

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**QUESTION 2** 

What are some of the barriers that prevent doctors seeking healthcare?

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**FURTHER INFORMATION**

A full and accurate history is taken and does not show any concerning features. Physical examination is normal. There is no indication of current cardiovascular disease and his calculated absolute cardiovascular disease risk is 15% (moderate).<sup>1</sup> Use of the GP assessment of cognition (GPCOG) tool,<sup>2</sup> a screening tool for cognitive impairment designed for use in the primary care setting, indicates low risk of significant cognitive impairment. There is no history or symptoms of alcohol misuse, substance abuse or mood disorder. Liver function tests are normal. Paul's Kessler Psychological Distress Scale (K10)<sup>3</sup> score was 14 (low). Paul states he wishes to continue working but acknowledges it may be time to start planning a gradual reduction in his workload.

**QUESTION 3** 

What are the effects of normal ageing that Paul should consider and plan for in relation to his professional duties?

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**QUESTION 4**  

How can doctors alter their work practices to accommodate the developmental process of ageing?

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**QUESTION 5**   

Is there a mandatory retirement age or other conditions on practice for doctors and other professionals?

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**QUESTION 6** 

Is there a role for formal cognitive testing in this case?

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**QUESTION 7**   

As the treating doctor, are you obliged to make a mandatory report to the Australian Health Practitioner Regulation Agency (AHPRA) about Paul's concerns about his professional practice?

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**CASE 5 ANSWERS**

**ANSWER 1**

Possible causes of lethargy and forgetfulness include:

- normal ageing
- depression, anxiety, substance abuse
- mild cognitive impairment or early dementia
- organic illness and chronic diseases.

Note, doctors have a lower risk of lifestyle-related illnesses such as heart disease and smoking-related conditions<sup>4,5</sup> but are at a greater risk of psychological problems including mental illness and stress related problems, as well as substance abuse,<sup>6,7</sup> compared with the general population. A *beyondblue* survey reported higher rates of psychological distress and attempted suicide in doctors, compared with the general population and other health professionals. Very high levels of psychological distress were significantly higher in doctors, compared with the general population and other professionals (3.4% versus 2.6% versus 0.7%).<sup>8</sup> Certain subgroups of doctors may also be at increased risk of poor health because of their professional circumstances (eg doctors who work excessive hours and/or are unable to take sufficient leave, and doctors working in rural/remote areas with inadequate resources and professional support).<sup>9</sup>

**ANSWER 2**

There are many barriers,<sup>9,10</sup> both real and perceived, that prevent some doctors and medical students from seeking medical advice. These include:

- concerns about lack of confidentiality
- embarrassment
- perceptions of weakness
- the stigma of ill health within the medical community
- perceived impact on career development
- perceived impact on colleagues and patients
- the expectation that doctors will work when unwell
- the implications of mandatory notification
- access to professional treatment (time, experienced personnel, geographic location).

**ANSWER 3**

Ageing doctors are at risk of chronic and degenerative diseases and malignancies, as well as a number of sensory and neurocognitive changes associated with normal ageing, including:<sup>11</sup>

- impaired hearing and sight
- reduced manual dexterity
- decline in processing speed and memory
- reduced problem solving and fluid intelligence
- reduced ability to multitask.

**ANSWER 4**

Examples of how doctors can alter their work practices to accommodate the ageing process include:<sup>11,12</sup>

- reducing hours on call
- reducing shift work
- a transition to part-time work
- ceasing or decreasing procedural work
- reducing exposure to situations requiring rapid decision-making and response (time-critical emergency situations)
- pursuing roles in chronic disease management
- increasing consulting time allocated per patient
- using memory aids such as prescribing software and practice guidelines
- seeking second opinions and advice from colleagues in difficult cases
- pursuing non-clinical duties such as teaching, medico-legal work and research.

**ANSWER 5**

There is no current mandated retirement age or requirement for mandatory performance assessment for older doctors in Australia.<sup>12</sup> All registered medical practitioners, regardless of age, need to 'recognise and work within the limits of their competence' as

specified in the Medical Board of Australia's *Code of Conduct*.<sup>13</sup> The Royal Australian College of Surgeons has a position statement for ageing surgeons in active practice, which recommends annual health and vision checks and performance reviews; however, this has not been mandated.<sup>14</sup>

Other professionals with high levels of responsibility, such as pilots, judges and directors of publically listed companies, have mandatory retirement ages. For example, commercial pilots must retire at 65 years and are required to have 6-monthly physical and mental examinations from age of 40 years. Judges and directors of publically listed companies must retire at age 72 years.<sup>12</sup>

### ANSWER 6

There is no agreed method or standard of cognitive testing that reliably predicts clinical performance.<sup>12</sup> There may be some benefit in baseline testing and ongoing surveillance in selected individuals, particularly those continuing to work in higher-risk practices well into older age, but there is no firm evidence to support such testing.

### ANSWER 7

As there is no evidence that Paul has any impairment or is placing the public at risk, there is no need to report him to AHPRA. On the contrary, he has good insight and has taken responsible steps to ensure his fitness to practice.

Notifiable conduct is defined in the Medical Board of Australia Guidelines for Mandatory Reporting. Section 140 of the Health Practitioner Regulation National Law defines 'notifiable conduct' as when a practitioner has:<sup>15</sup>

- practised while intoxicated by drugs or alcohol
- engaged in sexual misconduct
- placed the public at risk of substantial harm because of an impairment
- placed the public at risk because of a significant departure from accepted professional standards.

### CONCLUSION

Paul returns 12 months later for his planned annual health check. He reports that he has ceased his weeknight on-call duties and that his practice is actively recruiting a new doctor and a GP registrar. Once a new doctor is appointed he plans to cease weekend calls and to reduce his consulting hours. He has already increased his appointment times from 15 to 20 minutes and reports that this has reduced his work stress and increased his enjoyment. He plans to take on some registrar supervision duties and has undertaken some 'train the trainer' education, as well as updating his clinical knowledge. The practice may also accommodate some medical students. He is excited about his changing role in the practice and the prospect of sharing his knowledge with the next generation of doctors.

Paul and his wife have sought financial advice and have begun planning for a gradual transition to comfortable retirement in the next 5 years.

### REFERENCES

1. National Vascular Disease Prevention Alliance. Australian absolute cardiovascular disease risk calculator. Available at [www.cvdcheck.org.au](http://www.cvdcheck.org.au) [Accessed 4 August 2014].
2. GPCOG. The general practitioner assessment of cognition. Available at [www.gpcog.com.au](http://www.gpcog.com.au) [Accessed 4 August 2014].
3. Black Dog Institute. Kessler Psychological Distress Scale. Available at [www.blackdoginstitute.org.au/docs/5.k10withinstructions.pdf](http://www.blackdoginstitute.org.au/docs/5.k10withinstructions.pdf) [Accessed 4 August 2014].
4. Carpenter L, Swerdlow A, Fear N. Mortality of doctors in different specialties: findings from a cohort of 20,000 NHS hospital consultants. *Occup Environ Med* 1997;54:388–95.
5. Clode D. The conspiracy of silence: Emotional health among medical practitioners. East Melbourne: The Royal Australian College of General Practitioners, 2004.
6. Willcock SM, Daly MG, Tennant CC, Allard BJ. Burnout and psychiatric morbidity in new medical graduates. *Med J Aust* 2004;181:357–60.
7. Schattner P, Davidson S, Serry N. Doctors' health and wellbeing: taking up the challenge in Australia. *Med J Aust* 2004;181:348–49.
8. *beyondblue*. National mental health survey of doctors and medical students. Available at [www.beyondblue.org.au/docs/default-document-library/bl1132-report---nmhdms-full-report\\_web.pdf?sfvrsn=2](http://www.beyondblue.org.au/docs/default-document-library/bl1132-report---nmhdms-full-report_web.pdf?sfvrsn=2) [Accessed 4 August 2014].
9. Australian Medical Association. Health and wellbeing of doctors and medical students. Canberra: AMA, 2011. Available at <https://ama.com.au/position-statement/health-and-wellbeing-doctors-and-medical-students-2011> [Accessed 4 August 2014].
10. Hillis JM, Perry WRG, Carroll EY, Hibble BA, Davies MJ, Yousef J. Painting the picture: Australasian medical student views on wellbeing teaching and support services. *Med J Aust* 2010;192:188–90.
11. Skowronski GA, Peisah C. The greying intensivist: ageing and medical practice – everyone's problems. *Med J Aust* 2012;196:505–07.
12. Addler RG, Constantinou C. Knowing or not knowing – when to stop: cognitive decline in ageing doctors. *Med J Aust* 2008;189:622–24.
13. Medical Board of Australia. Good medical practice: a code of conduct for doctors in Australia. March 2014. Available at [www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx) [Accessed 4 August 2014].
14. Royal Australasian College of Surgeons. Position paper: senior surgeons in active practice. Ref. No. FES-FEL-048. Available at [www.surgeons.org/media/20264872/2013-10-29\\_pos\\_fes-fel-048\\_senior\\_surgeons\\_in\\_active\\_practice.pdf](http://www.surgeons.org/media/20264872/2013-10-29_pos_fes-fel-048_senior_surgeons_in_active_practice.pdf) [Accessed 4 August 2014].
15. Medical Board of Australia. Guidelines for mandatory reporting. Available at [www.medicalboard.gov.au/Codes-Guidelines-Policies/Guidelines-for-mandatory-notifications.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies/Guidelines-for-mandatory-notifications.aspx) [Accessed 4 August 2014].

### RESOURCES FOR DOCTORS

- Skowronski GA, Peisah C. The greying intensivist: aging and medical practice – everyone's problems. *Med J Aust* 2012;196:505–07.
- Addler RG, Constantinou C. Knowing or not knowing – when to stop: cognitive decline in ageing doctors. *Med J Aust* 2008;189:622–24.
- McNamara. Older doctors may face mandatory review. *MJA Insight*. May 7 2012, [www.mja.com.au/insight/2012/17/older-doctors-may-face-mandatory-review](http://www.mja.com.au/insight/2012/17/older-doctors-may-face-mandatory-review)
- Royal Australian College of Surgeons. Position paper: senior surgeons in active practice, [www.surgeons.org/media/20264872/2013-10-29\\_pos\\_fes-fel-048\\_senior\\_surgeons\\_in\\_active\\_practice.pdf](http://www.surgeons.org/media/20264872/2013-10-29_pos_fes-fel-048_senior_surgeons_in_active_practice.pdf)
- Peisah C, Gautam M, Goldstein M. Medical masters: a pilot study of adaptive ageing in physicians. *Aust J Aging* 2009;28:134–38.
- Medical Board of Australia. Code of Conduct, [www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx](http://www.medicalboard.gov.au/Codes-Guidelines-Policies.aspx)

**GP health and wellbeing (Activity ID: 8567)**

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 <http://gplearning.racgp.org.au>
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The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office.

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**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.**

**DR JAMES**

Dr James is a GP aged 68 years who works in a remote country practice. He shares the after-hours on-call roster with you, performs antenatal care and attends to low-risk inpatients at the closest hospital, which is 120 km from your practice. Dr James has been increasingly vague and anxious in the past year, and his overall condition has worsened since his wife's recent sudden death.

**QUESTION 1**

Which statement is correct with regards to the incidence of health problems in clinicians?

- Doctors experience the same levels of lifestyle-related diseases as the general population.
- Doctors experience fewer mental health problems than the general population.
- Doctors are at lower risk of substance abuse than the general population.
- Doctors have lower rates of attempted suicide than the general population.
- Certain subgroups of doctors are at increased risk of health problems because of their professional circumstances.

**QUESTION 2**

Taking your advice, James sees a GP in the city, who reports that he is fit to continue practising and advises James to reduce his clinical load more manageable. Which statement is correct with regards to ageing doctors in practice?

- Concern about lack of confidentiality is not a barrier for ageing doctors seeking healthcare.
- Pursuing roles in chronic disease management is not recommended as a way of accommodating the process of ageing.
- A transitioning to part-time work is one way that doctors can alter their work practices to accommodate ageing.
- Ageing GPs in active practice must have annual health and vision checks.
- The mandatory retirement age for doctors is the same as for the general population.

**SAM**

Sam is a homeless man aged 22 years who presents with a hand injury requiring suturing. While you are stitching the wound, Sam sneezes violently and you stab your finger with the needle.

**QUESTION 3**

What should you do next?

- Wash your wound with soap or standard handwash and water.
- Finish stitching Sam's wound.
- Wash your wound with bleach.
- Inject antiseptic into your wound.
- Call the post-exposure prophylaxis (PEP) hotline immediately to access PEP.

**QUESTION 4**

Sam reluctantly discloses he is an occasional injecting drug user and has no knowledge of his viral status. Which statement correctly describes the implications of Sam's disclosures for you?

- You definitely require hepatitis B virus (HBV) PEP.
- You should have hepatitis C virus (HCV) PEP.
- Your decisions regarding use of PEP can be delayed until Sam's viral status is determined.
- Should you require human immunodeficiency virus (HIV) PEP, timing is not an issue as its use is not time-dependent.
- Your risk of HIV transmission with a known HIV-positive source in the setting of a needle stick injury is estimated to be 1 in 440.

**DR JULIA**

Dr Julia received an anonymous letter from a patient claiming to have been sexually involved with Dr James, her practice partner.

**QUESTION 5**

Which statement regarding notifiable conduct and mandatory reporting in general practice is correct?

- A. Practicing while intoxicated by alcohol does not constitute notifiable conduct.
- B. Engaging in sexual misconduct is defined as notifiable conduct.
- C. A doctor must directly observe a notifiable behaviour to make a notification.
- D. There are no exceptions to the requirements for mandatory reporting.
- E. Mandatory reporting guidelines do not apply to practice nurses.

**QUESTION 6**

Dr Julia recalls that Dr James had a reputation for being a ladies' man at university and while training. What is the most advisable course of action for Dr Julia?

- A. Report Dr James to AHPRA.
- B. Report Dr James to the Medical Board of Australia.
- C. Do nothing and store the letter in a confidential, secure place.
- D. Destroy the letter.
- E. Discuss the letter with the practice nurse.

**DR JONAS**

Dr Jonas received a letter of complaint from a patient, Mr Atkins, who has inoperable lung cancer and claims that Dr Jonas failed to diagnose this condition. Mr Atkins states that he has started proceedings to sue Dr Jonas.

**QUESTION 7**

Which action should be the immediate priority for Dr Jonas?

- A. Telephone the patient.
- B. Write to the patient.
- C. Talk to a colleague.
- D. Contact his medical defence organisation (MDO).
- E. Dispose of the letter.

**QUESTION 8**

Which of the following statements is correct with regards to adverse events and medical negligence complaints?

- A. Fear, guilt, shame, self-doubt, anger and disappointment are common reactions that health professionals experience following an adverse event.
- B. Australian doctors receive the least complaints of all health professionals.
- C. Of doctors involved in a claim for medical negligence, 20% experience suicidal ideation.
- D. Dr Jonas is the first victim in this case.
- E. Mr Atkins is the second victim.

**DR SMITH**

Dr Smith is a GP aged 48 years. His wife and only child died in a motor vehicle accident 3 months ago. Since returning to work, he has been noticeably subdued and melancholic. Dr Brown, a colleague and personal friend asks him how he is. Dr Smith reports that he is unable to sleep without medication. He thinks he is depressed and might start himself on antidepressants.

**QUESTION 9**

Which statement is correct with regards to doctor self-treatment?

- A. No problems have been associated with clinician self-treatment.
- B. Self-medication may be a risk factor for future substance abuse problems.
- C. *Good Medical Practice: A Code of Conduct for Doctors in Australia* (the Code) encourages doctors to self-treat.
- D. There are no legal barriers to doctors self-prescribing in Australia.
- E. Australian doctors can self-prescribe schedule 8 drugs.

**QUESTION 10**

Dr Smith asks Dr John if he could see him as a patient or, alternatively, just give him a prescription for sleeping tablets and an antidepressant. Which of the following statements is the most correct regarding treatment of doctor-patients and/or friends?

- A. There are no cautions against treating friends.
- B. There are no pitfalls or potential problems with treating a doctor-patient.
- C. The Code advises that doctors should avoid providing care to doctor-patients who are close friends and colleagues.
- D. Doctors treating doctor-patients should avoid asking personal questions.
- E. Doctors treating doctor-patients should always waive their fees.



# check

Independent learning program for GPs

# check

Independent learning program for GPs



Unit 512 December 2014

## Preventive health

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Independent learning program for GPs



## Preventive health

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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

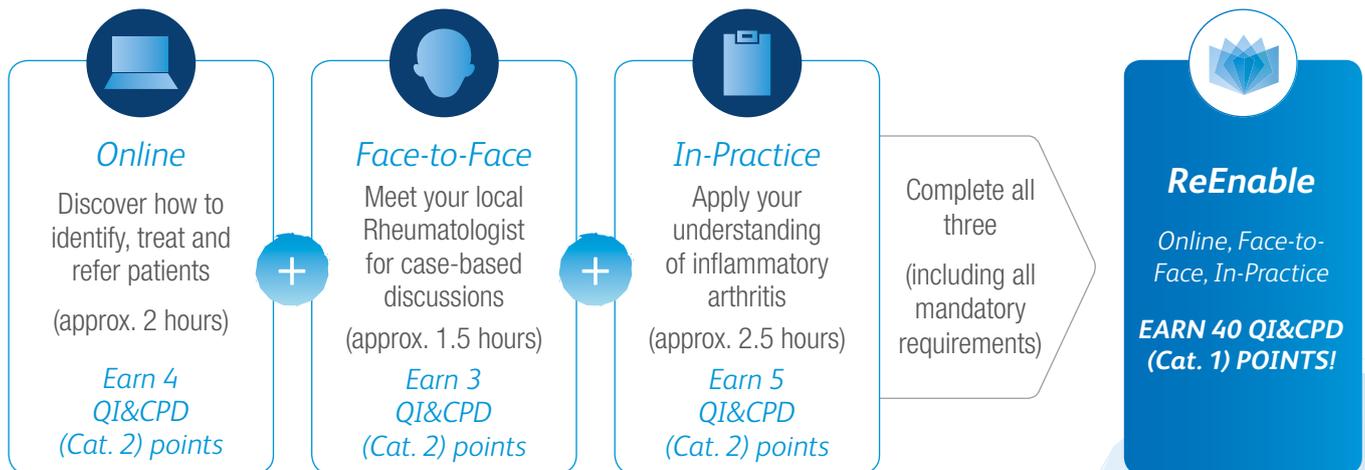


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## ABOUT THIS ACTIVITY

'Prevention is better than cure' is a concept embedded in medical culture and preventive healthcare reflects measures that can be taken for disease prevention. These measures include the prevention of illness, the early detection of specific disease and the promotion and maintenance of good health.<sup>1</sup> In Australia, chronic disease is the leading cause of death and disability.<sup>2</sup> However, modification of risk factors through lifestyle change and pharmacological intervention can reduce the incidence of chronic disease and reduce premature death and disability.<sup>2</sup> The RACGP's *Guidelines for preventive activities in general practice* (Red book)<sup>1</sup> provides a framework for preventive activities in general practice. This edition of *check* considers case studies that explore preventive health in general practice.

## LEARNING OUTCOMES

At the end of this activity participants will be able to:

- summarise evidence-based guidelines for primary prevention including calculation of a person's absolute cardiovascular disease risk
- develop a systematic approach to managing patients after myocardial infarction
- describe the approach to undertaking a vulval examination as part of a regular Pap smear
- outline preventive health activities suitable for people working in rural settings
- explain current immunisation recommendations for Aboriginal and Torres Strait Islander peoples.

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**REFERENCES**

1. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice, 8th edn. Melbourne: RACGP, 2012. Available at [www.curriculum.racgp.org.au](http://www.curriculum.racgp.org.au) [Accessed 29 April 2014].
2. Australian Institute of Health and Welfare. Risk factors contributing to chronic disease. Canberra: AIHW, 2012. Available at [www.aihw.gov.au/publication-detail/?id=10737421466](http://www.aihw.gov.au/publication-detail/?id=10737421466) [Accessed 2 June 2014].

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

5As	assess, advice, agree, assist and arrange	FBE	full blood evaluation	NHMRC	National Health and Medical Research Council
ACEI	angiotensin converting enzyme inhibitor	HbA1c	glycated haemoglobin	NRT	nicotine replacement therapy
AF	atrial fibrillation	HDL-C	high-density lipoprotein cholesterol	NSAID	non-steroidal anti-inflammatory drug
ARB	angiotensin receptor blocker	HEADSS	Home, Education/Employment, Activities, Drugs, Sexuality and Suicide	PBS	Pharmaceutical Benefits Scheme
BMI	body mass index	HIV	human immunodeficiency virus	PENCAT	Pen Computer Systems Audit Tool
BP	blood pressure	HMR	home medicines review	PHQ	patient health questionnaire
CHD	coronary heart disease	HPV	human papillomavirus	RAST	radioallergosorbent test
CIN	cervical intraepithelial neoplasia	HRT	hormone replacement therapy	SCC	squamous cell carcinoma
COPD	chronic obstructive pulmonary disease	IgE	immunoglobulin E	SNAP	smoking, nutrition, alcohol and physical activity
COX-2	cyclooxygenase-2	LDL-C	low-density lipoprotein cholesterol	STI	sexually transmissible infection
CVD	cardiovascular disease	LFT	liver function test	TG	triglycerides
DTPa	diphtheria, tetanus, pertussis vaccine	MCS	microscopy, culture and swab	TSH	thyroid stimulating hormone
ECG	electrocardiogram	MI	myocardial infarction	UACR	urinary albumin:creatinine ratio
eGFR	estimated glomerular filtration rate	NHF	National Heart Foundation	UAE	urea and electrolytes
				VIN	vaginal intraepithelial neoplasia

**CASE 1**

**RONALD HAS A COUGH**

Ronald, aged 26 years, is an Aboriginal man who coaches a junior football team. Ronald presents with a 3-week cough. He previously had rhinorrhoea, which settled after a few days. He has a strong family history of asthma and had asthma as a child. He considers himself a healthy man and takes no regular medications but is worried that his asthma may have come back.

**QUESTION 1**  

How could you manage this consultation to provide preventive care for Ronald?

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**QUESTION 2**  

What preventive health screening/assessments would you consider for Ronald?

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**FURTHER INFORMATION**

Ronald is a smoker, having started smoking tobacco in his early teens, and is interested in quitting. His wife is pregnant and this is part of his motivation to quit smoking. Apart from his health concerns, he tells you he had money problems recently and it would save him money if he could stop smoking.

On examination, Ronald has a normal body mass index (BMI) and waist circumference, blood pressure, random blood sugar level and heart rate. His respiratory and cardiovascular examination is unremarkable. You perform spirometry, which shows mild, reversible airways obstruction, and you advise him his cough might be due, at least in part, to asthma. There is no evidence of chronic obstructive pulmonary disease (COPD) on spirometry.

**QUESTION 3** 

Is there a role for spirometry in screening of asymptomatic individuals for early detection of asthma and COPD?

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**FURTHER INFORMATION**

In view of Ronald's interest in stopping smoking, you discuss his options in depth, advising smoking cessation counselling and discuss medication options.

**QUESTION 4**  

What type of smoking cessation counselling is likely to be most valuable for Ronald?

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**FURTHER INFORMATION**

You note that Ronald is up-to-date on tetanus immunisation but has not had any other adult immunisations.



- rheumatic heart disease
- hearing
- sexual health
- mental health
- respiratory health, cardiovascular health, chronic kidney disease, diabetes
- immunisation status
- dental health.

More information about screening/assessments can be obtained from the *National guide to preventive health assessment for Aboriginal and Torres Strait Islander people*.<sup>2</sup>

Questioning and history taking for Ronald could be tailored to obtaining information about the preventive areas of relevance to him, in addition to obtaining more information about his presenting concerns. The SNAP (smoking, nutrition, alcohol and physical activity) risk factors and the 5As framework (Ask, Assess, Advise, Assist, Arrange) can be used to engage people in lifestyle discussions and the 5As framework can be used to facilitate the detection, assessment and management of SNAP risk factors.<sup>1,2</sup>

### ANSWER 3

In Ronald's case, spirometry was indicated given his symptoms.<sup>6</sup> However, there is no evidence to recommend routine spirometry for screening of asymptomatic individuals for either asthma<sup>2</sup> or COPD.<sup>1,2</sup> Spirometry should only be undertaken in those with symptoms suggestive of asthma or COPD.<sup>6,7</sup> However, early detection of asthma and COPD is recommended. Strategies for early detection of asthma include clinical vigilance and detailed history taking, considering conditions that mimic asthma.<sup>2</sup> Screening for symptoms of COPD should be undertaken opportunistically in all smokers and in all ex-smokers over the age of 35 years.<sup>2,7</sup>

### ANSWER 4

There is strong evidence for the effectiveness of brief advice, brief interventions, cessation counselling, proactive Quitlines and pharmacotherapy in increasing quit rates in general populations.<sup>8</sup> There are few studies assessing the effectiveness of smoking cessation interventions for Aboriginal and Torres Strait Islander peoples; however, there is no evidence to suggest interventions found to be effective in other populations would be any less effective for Aboriginal and Torres Strait Islander peoples. Flexible and culturally targeted modes of delivering smoking cessation interventions<sup>1,2</sup> are likely to improve their effectiveness for Aboriginal and Torres Strait Islander peoples. For example, resources created for Aboriginal and Torres Strait Islander peoples and brief interventions delivered by Aboriginal health workers may be useful.<sup>9,10</sup> Treatment could be delivered by a range of health professionals, including Aboriginal health workers, practice nurses, GPs and smoking cessation counsellors to increase tobacco cessation rates.<sup>10,11</sup> The RACGP's *Supporting smoking cessation: a guide for health professionals*<sup>11</sup> contains a number of specific points of relevance to Aboriginal and

Torres Strait Islander peoples, including the following:

- People who identify as Aboriginal or Torres Strait Islander qualify for Pharmaceutical Benefits Scheme (PBS) authority listing that provides up to two courses per year of nicotine patches, each of a maximum of 12 weeks. Under this listing, participation in a support and counselling program is recommended but not mandatory.
- Access to nicotine patches, bupropion and varenicline for Aboriginal and Torres Strait Islander peoples can be facilitated through the Closing the Gap PBS co-payment measure.

A number of guidelines provide advice on smoking cessation, often using the 5As model.<sup>1,2,12</sup> The basic principles of setting a quit date, emphasising the importance of abstinence and providing multi-session support (preferably four or more sessions) are important evidence-based strategies that assist people to stop smoking.<sup>1,2</sup> Even minimal advice assists people to quit smoking.<sup>11</sup> There is also a positive correlation between successful smoking cessation and counselling activities such that the longer the duration of the person-to-person intervention, the more effective it is.<sup>13,14</sup> Smoking cessation advice should be sensitive to the patient's preferences, needs and circumstances and should be delivered in a culturally appropriate manner.<sup>1</sup> There is no evidence that any particular behaviour change method is more effective than another in promoting change.

Studies suggest GPs underuse effective smoking cessation treatment approaches such as Quitline, pharmacotherapy and motivational interviewing.<sup>1</sup> Referral to Quitline should be strongly considered for all smokers.<sup>1,2,12</sup> Quitline has been shown to be effective in many populations worldwide and is likely to be beneficial for Aboriginal and Torres Strait Islander peoples. Quitline includes the option for online registration of patients, which can be done during a GP consultation, so they can receive follow-up phone calls.

### ANSWER 5

Some groups of Aboriginal and Torres Strait Islander peoples may be at increased risk of vaccine-preventable diseases.<sup>15</sup> Influenza, pneumococcal and hepatitis B vaccines are recommended for Ronald, as discussed below.<sup>7,15</sup>

An annual influenza vaccine should be considered for Ronald. All Aboriginal and Torres Strait Islander peoples over the age of 15 years should be offered the current influenza vaccine annually, preferably in March to April before the Australian influenza season. This recommendation has been incorporated into Australian guidelines in view of the substantially increased risk of hospitalisation and death from influenza and pneumonia for Aboriginal and Torres Strait Islander peoples.<sup>15</sup> An influenza pandemic, the first since 1968, occurred in 2009 with the emergence of a new H1N1 influenza strain. Aboriginal and Torres Strait Islander peoples were disproportionately affected by the 2009 H1N1 influenza outbreak,<sup>15</sup> being four times more likely than non-Indigenous Australians to be admitted to hospital.<sup>16</sup>

Although Ronald does not have a chronic health condition, as a smoker he should be advised to have the pneumococcal vaccine (23vPPV) for the prevention of pneumococcal disease.

This vaccination is recommended for Aboriginal and Torres Strait Islander peoples aged 15–49 years who are smokers or have an underlying high-risk condition (eg chronic cardiac, renal or lung disease, diabetes, alcohol-related problems, immunosuppression).<sup>2,15</sup> Aboriginal and Torres Strait Islander children and adults have a significantly higher incidence of all pneumococcal disease than non-Indigenous Australians.<sup>15</sup> The risk of invasive pneumococcal disease in young Aboriginal and Torres Strait Islander adults is 11-fold higher than in non-Indigenous Australians.<sup>17</sup>

Pneumonia is the most common communicable disease contributor to premature death in Aboriginal and Torres Strait Islander adults. Hospitalisation for pneumonia is four times more common in Aboriginal and Torres Strait Islander peoples than in non-Indigenous Australians and up to eight times higher in younger Aboriginal and Torres Strait Islander adults compared with non-Indigenous young adults.<sup>15,18</sup>

Current immunisation guidelines recommend all Aboriginal and Torres Strait Islander adults should be offered testing for previous hepatitis B infection and, if non-immune, they should be offered vaccination.<sup>15</sup> These guidelines also recommend pertussis vaccination for those at risk of transmitting it to vulnerable persons (eg neonates). Given that Ronald's wife is pregnant, he should be offered the pertussis vaccination to prevent transmission to the baby.

#### ANSWER 6

There are several validated tools to screen for gambling that may be useful in community education and screening programs for those at high risk;<sup>19</sup> however, these have not yet been validated in Aboriginal and Torres Strait peoples.<sup>2</sup> Simple questions such as 'Do you gamble?' and 'Have you ever had an issue with your gambling?'<sup>2</sup> may be as effective as more detailed tools and more appropriate for primary care screening.<sup>20</sup>

The National Aboriginal Community Controlled Health Organisation (NACCHO) and the Royal Australian College of General Practitioners (RACGP) guidelines, *National guide to a preventive health assessment for Aboriginal and Torres Strait Islander people*, recommends that opportunistic screening for gambling behaviour and problems should be undertaken for all people over the age of 12 years. Screening may be undertaken in response to concerns arising within consultations, as with Ronald, or may be included as part of an annual health assessment.<sup>2</sup>

Gambling is a significant issue for many Aboriginal and Torres Strait Islander peoples, and can have serious consequences for individuals, families and communities. Aboriginal and Torres Strait Islander peoples are more likely to be regular and problem gamblers than non-Indigenous Australians, and to start gambling at a younger age. Surveys of Aboriginal and Torres Strait Islander communities have found that problems associated with gambling include financial hardship, social and emotional difficulties, substance misuse, and contact with the criminal justice system. Shame and stigma may prevent Aboriginal and Torres Strait Islander peoples from accessing help for gambling-related problems.<sup>21</sup> Specific groups at risk of problem gambling include young people or adults with stress related medical problems, mental health issues, substance misuse.<sup>2,20,21</sup>

#### ANSWER 7

Environmental and lifestyle factors interact with genetic factors, such as an allergic tendency, to increase the risk of developing asthma.

There is no evidence that maternal dietary changes or allergen avoidance in pregnancy decreases the risk of asthma in the child. For example, avoidance of house dust mite or pet allergens has not been shown to be effective in preventing asthma prenatally.<sup>22</sup>

There is also no evidence that taking probiotic dietary supplements, vitamin A, D or E supplements, or fish oil in pregnancy is effective in preventing asthma.<sup>22</sup> However, family lifestyle changes are likely to improve the health of the whole family and, for example, quitting smoking and ensuring that the child is not exposed to environmental tobacco smoke are important preventive measures.<sup>2</sup>

#### REFERENCES

1. Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne: RACGP, 2012. Available at [www.racgp.org.au/your-practice/guidelines/redbook](http://www.racgp.org.au/your-practice/guidelines/redbook) [Accessed 1 September 2014].
2. National Aboriginal Community Controlled Health Organisation, Royal Australian College of General Practitioners. National guide to a preventive health assessment for Aboriginal and Torres Strait Islander people. 2nd edn. East Melbourne: RACGP, 2012. Available at [www.naccho.org.au/promote-health/national-guide-to-a-preventive-health-assessment/](http://www.naccho.org.au/promote-health/national-guide-to-a-preventive-health-assessment/) [Accessed 1 September 2014].
3. Australian Institute of Health and Welfare. Chronic diseases and associated risk factors in Australia. Canberra: AIHW, 2006.
4. Brown A, Walsh W, Lea T, Tonkin A. What becomes of the broken hearted? Coronary heart disease as a paradigm of cardiovascular disease and poor health among Indigenous Australians. *Heart, Lung Circ* 2005;14:158–62.
5. Australian Bureau of Statistics. National Aboriginal and Torres Strait Islander Social Survey. Canberra: Commonwealth of Australia, 2008.
6. National Asthma Council Australia. Australian Asthma Handbook, Version 1.0. Melbourne: Asthma Council Australia, 2014. Available at [www.asthmahandbook.org.au](http://www.asthmahandbook.org.au) [Accessed 1 September 2014].
7. Lung Foundation Australia, The Thoracic Society of Australia and New Zealand. The COPDX Plan: Australian and New Zealand guidelines for the management of chronic obstructive pulmonary disease. Milton: Lung Foundation Australia, 2014. Available at [www.copdx.org.au](http://www.copdx.org.au) [Accessed 1 September 2014].
8. Stead LF, Bergson G, Lancaster T. Physician advice for smoking cessation. *Cochrane Database Syst Rev* 2008;16:CD000165.
9. Centre for Excellence in Indigenous Tobacco Control. Available at [www.ceitc.org.au/welcome-ceitc](http://www.ceitc.org.au/welcome-ceitc) [Accessed 27 October 2014].
10. Queensland Health. Smokecheck – Indigenous smoking program. Available at [www.health.qld.gov.au/atod/prevention/smokecheck.asp](http://www.health.qld.gov.au/atod/prevention/smokecheck.asp) [Accessed 27 October 2014].
11. The Royal Australian College of General Practitioners. Supporting smoking cessation: a guide for health professionals. Melbourne: RACGP, 2011 [Updated 2014]. Available at [www.racgp.org.au/your-practice/guidelines/smoking-cessation/](http://www.racgp.org.au/your-practice/guidelines/smoking-cessation/) [Accessed 2 September 2014].
12. Royal Australian College of General Practitioners. Putting prevention into practice: Guidelines for the implementation for prevention in the general practice setting. 2nd edn. East Melbourne: RACGP, 2006. Available at [www.racgp.org.au/download/documents/Guidelines/Greenbook/racpggreenbook2nd.pdf](http://www.racgp.org.au/download/documents/Guidelines/Greenbook/racpggreenbook2nd.pdf) [Accessed 1 September 2014].
13. Stead LF, Buitrago D, Preciado N, Sanchez J, Hartmann-Boyce J, Lancaster T. Physician advice for smoking cessation. *Cochrane Database Syst Rev* 2013; 5:CD000165. doi: 10.1002/14651858.CD000165.pub4.

14. Fiore MC, Jaen CR. Tobacco Use and Dependence Guideline Panel. Treating tobacco use and dependence. Rockville, MD: US Department of Health and Human Services, 2008. Available at [www.ncbi.nlm.nih.gov/books/NBK12193/](http://www.ncbi.nlm.nih.gov/books/NBK12193/) [Accessed 27 October 2014].
15. Australian Technical Advisory Group on Immunisation. The Australian Immunisation Handbook. 10th ed. Canberra: Australian Government Department of Health, 2013. Updated January 2014. Available at [www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home](http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home) [Accessed 1 September 2014].
16. Australian Institute of Health and Welfare. Australia's health. Canberra: AIHW, 2009.
17. Menzies R, Tumour C, Chiu C, McIntyre P. (2008) Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia 2003 to 2006. *Commun Dis Intell Q Rep* 2008;32(Suppl):S2-67.
18. Australian Institute of Health and Welfare. Asthma, chronic obstructive pulmonary disease and other respiratory diseases in Australia. Canberra: AIHW, 2010.
19. Problem Gambling Research and Treatment Centre. Guideline for screening, assessment and treatment in problem gambling. Melbourne: Monash University, 2011. Available at [www.med.monash.edu.au/assets/docs/sphc/pgrtc/guideline/problem-gambling-guidelines-web.pdf](http://www.med.monash.edu.au/assets/docs/sphc/pgrtc/guideline/problem-gambling-guidelines-web.pdf) [Accessed 27 October 2014].
20. Thomas SA, Piterman L, Jackson AC. What do GPs need to know about problem gambling and what should they do about it? *Med J Aust* 2008;189: 135–36.
21. Aboriginal Health and Medical Research Council. Pressing problems; gambling issues and responses for NSW Aboriginal communities. Sydney: AH&MRC. Available at [www.olgr.nsw.gov.au/rr\\_pp.asp](http://www.olgr.nsw.gov.au/rr_pp.asp) [Accessed 2 September 2014].
22. National Asthma Council Australia. Asthma prevention for children at risk of developing asthma. In: Australian Asthma Handbook. Melbourne: Asthma Council Australia, 2014. Available at [www.asthmahandbook.org.au/prevention/primary/children](http://www.asthmahandbook.org.au/prevention/primary/children) [Accessed 27 October 2014].

**CASE 2**

**PETE'S CHECK-UP**

Pete reluctantly visits you at the urging of his wife, who is concerned about his osteoarthritis and diminishing dexterity. You ask Pete how the arthritis is affecting him and he replies 'it does not stop me fishing'. He also states that he will soon retire from his landscaping business to concentrate on the things he loves – his family and fishing. Pete is 65 years of age and has smoked up to 10 cigarettes a day for most of his adult life. Recently, he reduced his smoking to three cigarettes a day but finds it hard to give up smoking completely.

**QUESTION 1** 

What should you assess in Pete?

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**FURTHER INFORMATION**

Pete attends a follow-up visit 1 week later. At this visit his systolic blood pressure (BP) is 135 mmHg (target <140 mmHg). His blood test results are shown in *Table 1*. Pete's waist circumference and body mass index (BMI) were in the normal range at 92 cm and 24 kg/m<sup>2</sup> respectively.

Test	Result	Target
Total cholesterol	6.0 mmol/L	<4.0 mmol/L
High density lipoprotein	1.5 mmol/L	≥1.0 mmol/L
Fasting blood sugar	6.3 mmol/L	<7.0 mmol/L

**QUESTION 2** 

Given Pete's unremarkable results, how can you assess his risk of a future cardiovascular event?

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**QUESTION 3** 

Can Pete's absolute cardiovascular disease (CVD) risk be better assessed?

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**QUESTION 4** 

If intensive lifestyle modification advice did not alter Pete's absolute CVD risk after 3 months, what therapeutic steps could you take to ensure Pete's risk of a stroke or heart attack is reduced?

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**QUESTION 5** 

What is the best way of ensuring that high CVD risk patients in your practice are identified and managed appropriately?

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**QUESTION 6** 

How could you implement assessment of absolute CVD risk in your general practice?

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## CASE 2 ANSWERS

## ANSWER 1

As Pete infrequently visits a GP, this is a good opportunity to perform a general check-up and update Pete's health record, for example, his smoking status, BP, BMI and waist circumference. It would also be useful to discuss his diet, other lifestyle/ psychosocial issues and any other concerns that he may have regarding his health in addition to his arthritis. Given Pete's outdoor occupation, a skin cancer screen should be considered. In addition, a clinic blood pressure reading and a pathology request for fasting glucose and lipid profile are the beginnings of establishing a CVD risk profile.<sup>1</sup> Note, screening for diabetes with HbA1c is now acceptable and does not require a fasting blood test. In relation to his arthritis, physical approaches to arthritis management and pharmacological interventions for pain management could be discussed and arranged; however, care must be taken in prescribing anything in addition to paracetamol, as many of the immunosuppressive drugs including non-steroidal anti-inflammatory drugs (NSAIDs) and cyclooxygenase-2 (COX-2) inhibitors have an adverse effect on CVD risk. There are no evidence-based cardiovascular guidelines specifically for patients on immunosuppressive drugs, but efforts to achieve risk factor reduction should be more rigorous than for the general population. In particular, further encouragement and external assistance to cease smoking altogether should be offered.

The Royal Australian College of General Practitioners' *Guidelines for preventive activities in general practice*<sup>2</sup> provides a useful framework for undertaking preventive activities in general practice settings, tailored to individual patient needs for people such as Pete.

## ANSWER 2

Pete's modifiable risk factors of systolic BP, blood lipids and blood glucose, considered individually, are only moderately raised. With the exception of smoking, Pete presents as being reasonably healthy. However, if his risk factors were to be considered as a coherent risk profile, the cumulative effect of BP, lipid profile, smoking status and age, Pete's clinical picture will become clearer with regards to his risk of a future cardiovascular event. Assessment of a person's CVD risk using multiple risk factors is more accurate than consideration of their individual risk factors, given that the cumulative effects of multiple risk factors in a person can have additive or synergistic effects.<sup>4-6</sup>

The *Guidelines for the management of absolute cardiovascular disease risk*<sup>1</sup> were approved by the National Health and Medical Research Council (NHMRC) in 2012 and should be used to assess adults who have no existing CVD and are 45 years of age and over, or 35 years and over for Aboriginal and Torres Strait islander peoples.

## ANSWER 3

Absolute CVD risk refers to the likelihood of a person experiencing a cardiovascular event within the next 5 years. In a treatment context, absolute risk offers a means of predicting the impact of Pete's risk factors on his overall cardiovascular wellbeing. The *Guidelines for the management of absolute cardiovascular disease risk* recommends at least three points of care for Pete:<sup>1</sup>

1. Risk assessment using the *Australian absolute cardiovascular disease risk calculator*<sup>3</sup> and collection of data on:
  - kidney function using urine albumin:creatinine ratio (UACR) for albuminuria and a blood test for serum creatinine to estimate glomerular filtration rate (eGFR)
  - diabetes status
  - age
  - sex
  - smoking status
  - systolic BP
  - total cholesterol levels
  - high-density lipoprotein cholesterol (HDL-C) levels
  - left ventricular hypertrophy (if known).

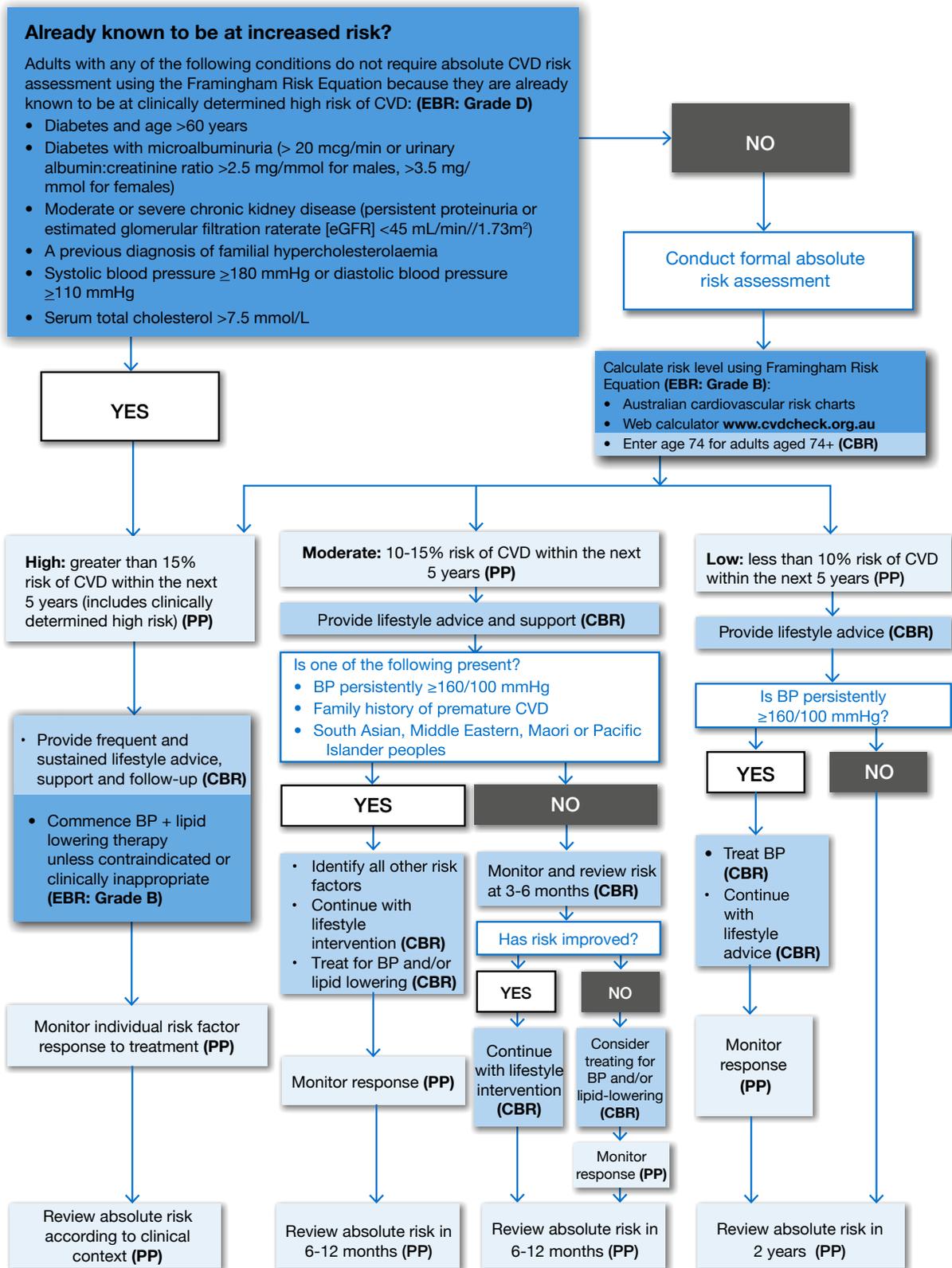
Using the *Australian absolute cardiovascular disease risk calculator* ([www.cvdcheck.org.au](http://www.cvdcheck.org.au))<sup>3</sup>, and Pete's clinical data a CVD risk score can be calculated for Pete. He has an absolute risk score of 18%. This score is considered high as it means that Pete has approximately a one in five chance of experiencing a stroke or heart attack over the next 5 years.<sup>2</sup>

Additional risk factors that may influence treatment decisions that should be considered include:<sup>1,2</sup>

- waist circumference and BMI
  - family history of premature cardiovascular disease
  - ethnicity
  - psychosocial factors including depression, social isolation and socio-economic status
  - atrial fibrillation (AF).
2. Provision of lifestyle modification advice on alcohol consumption, physical activity, diet and smoking cessation. The calculator can be used to demonstrate to Pete the benefits of giving up smoking. He can be shown that by giving up smoking completely, his CVD risk would decrease from 18% to 10% over 12 months (ie from 1 in 5 to 1 in 10 chance of having a cardiovascular event in the next 5 years). Furthermore, multiple lifestyle modifications could reduce his risk even further.
  3. Reviewing Pete's risk after 3 months of undertaking lifestyle modifications.

*Figures 1 and 2* outline aspects of the management suggested above in an algorithm.

**RISK ASSESSMENT AND MANAGEMENT ALGORITHM: ADULTS AGED 45 YEARS AND OVER WITHOUT KNOWN HISTORY OF CVD**

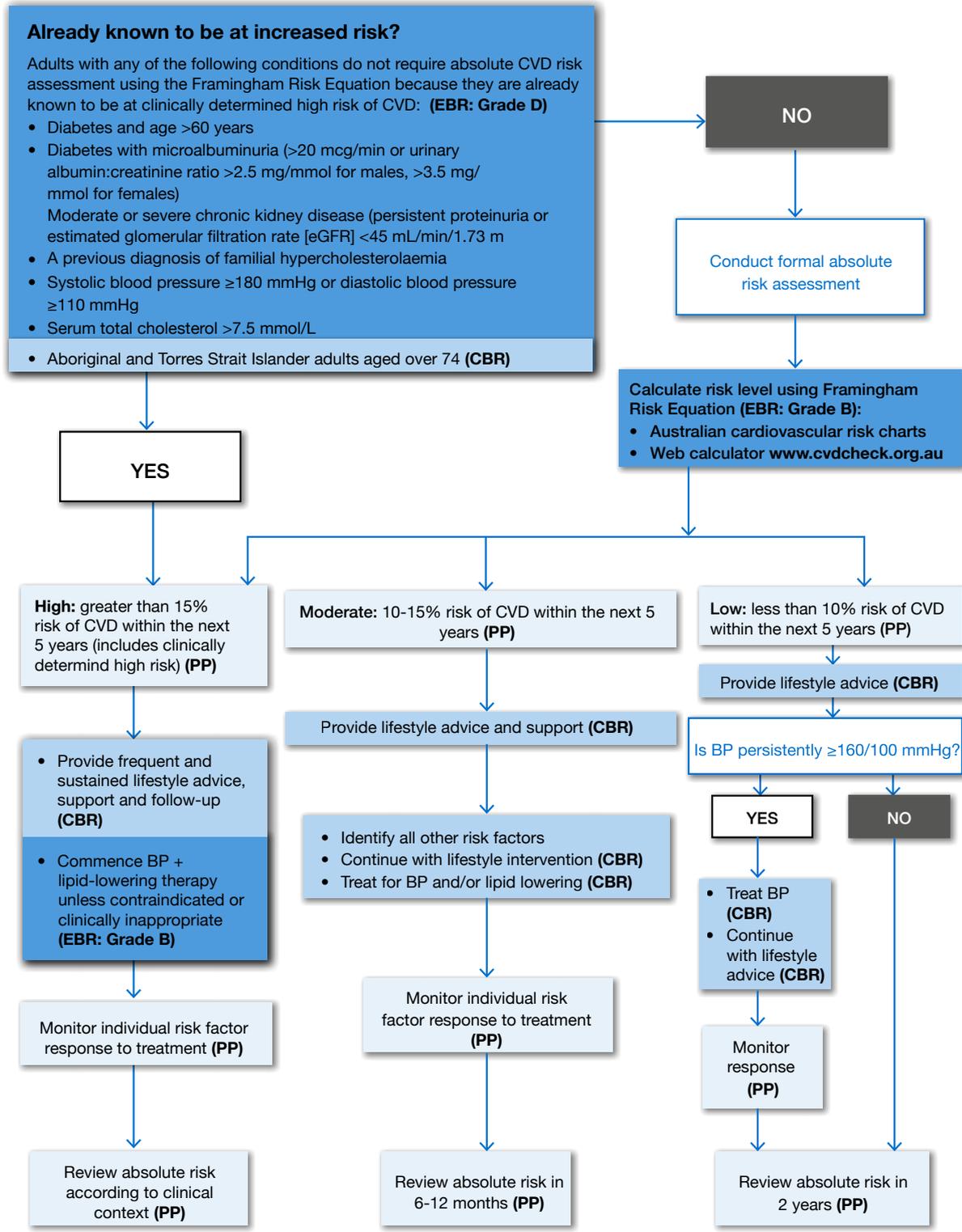


EBR: Evidence-based recommendation (Graded A-D) CBR: Consensus-based recommendation PP: Practice point

Figure 1. Absolute CVD risk algorithm for adults ≥45 years of age

Reproduced with permission from the National Vascular Disease Prevention Alliance. Guidelines for the management of Absolute cardiovascular disease risk. ©2012 National Stroke Foundation

**RISK ASSESSMENT AND MANAGEMENT ALGORITHM: ABORIGINAL AND TORRES STRAIT ISLANDER ADULTS AGED 35 YEARS AND OVER WITHOUT KNOWN HISTORY OF CVD**



EBR: Evidence-based recommendation (Graded A-D), CBR: Consensus-based recommendation, PP: Practice point

Figure 2. Absolute CVD risk algorithm for Aboriginal and Torres Strait Islanders ≥35 years of age

Reproduced with permission from the National Vascular Disease Prevention Alliance. Guidelines for the management of Absolute cardiovascular disease risk. ©2012 National Stroke Foundation

**ANSWER 4**

If lifestyle modifications had not altered Pete's pathology, anthropometric measurements or his overall CVD risk, it would have been important to consider appropriate therapeutic options. The following could be considered in such circumstances:

- Determine whether a lack of improvement is due to non-adherence to lifestyle changes and consider if further support could be provided by referral to additional health professionals.
- Commence BP and/or lipid lowering therapy in accordance with current guidelines to reduce CVD risk. Patients at moderate risk may be put on one or other therapy and reviewed. Patients at high risk should be put on both BP and lipid-lowering therapy unless contraindicated.<sup>1</sup> The aim is to reduce CVD risk regardless of the level of BP (as long as the medication does not cause hypotension) or lipids. If it is deemed clinically appropriate to commence medication, a statin is recommended as first-line therapy for lipid lowering and/or one of the following for reducing BP:<sup>1</sup>
  - angiotensin-converting-enzyme inhibitor (ACEI)
  - angiotensin II receptor blocker (ARB)
  - calcium channel blocker
  - low-dose thiazide
  - thiazide-like diuretic
- Provide structured advice, support, referral and/or pharmacotherapy for smoking cessation, if not previously done.<sup>1,2</sup>
- If risk reduction remains unsatisfactory, reassess for underlying causes that may be contributing to the person's overall CVD risk.
- Where indicated, dual antihypertensive therapy, using a second agent from a different pharmacological class, could be considered for patients whose BP is not sufficiently reduced with a single agent. Addition of a third antihypertensive agent could be considered if dual therapy does not sufficiently reduce the person's BP. Similarly, where statins do not adequately reduce cholesterol levels, addition of one or more lipid lowering agents may be required. Referral may also be warranted if treatment resistance is suspected.<sup>1</sup>

Note, low-dose aspirin is no longer routinely recommended for people clinically determined or calculated to be at a high absolute risk of a cardiovascular event (>15%) in the next 5 years, with the exception of Aboriginal and Torres Strait Islander peoples.<sup>1</sup>

**ANSWER 5**

The best way to ensure that patients at high risk of CVD are identified is to use a systematic approach to assessing absolute CVD risk within your practice. This involves the collection of a patient's risk factor information (eg blood pressure, smoking status, presence of diabetes, lipid levels) over a series of visits. Before calculating an actual absolute CVD risk score for a person, two fundamental questions need to be considered:

1. Do the guidelines apply to this patient?

The *Guidelines for the management of absolute cardiovascular*

*disease risk*<sup>1</sup> are applicable to adults who have no existing cardiovascular disease and are 45 years of age and over, or 35 and over for Aboriginal and Torres Strait Islander peoples.

2. Is this patient immediately at high risk?

No absolute CVD risk score is required for people who meet any of the immediate high-risk criteria, as they are already known to be clinically at high risk of CVD. High-risk criteria include:

- diabetes and age >60
- diabetes with microalbuminuria (>20 µg/min or UACR >2.5 mg/mmol for males, >3.5 mg/mmol for females)
- moderate-to-severe chronic kidney disease (persistent proteinuria or eGFR <45 mL/min/1.73 m<sup>2</sup>)
- previous diagnosis of familial hypercholesterolaemia
- systolic or diastolic BP ≥180 mmHg or ≥110 mmHg, respectively
- serum (fasting) total cholesterol >7.5 mmol/L
- Aboriginal and Torres Strait Islander peoples aged over 74 years.<sup>1</sup>

People with any of the above risk factors should be managed in accordance with the high-risk recommendations of the guidelines.<sup>1,2</sup> *Figures 1 and 2* provide algorithms for assessment and management in accordance with currently approved guidelines.<sup>1</sup>

A patient who has previously experienced a CVD event should be treated in accordance with secondary prevention guidelines. These patients are at high risk of further events and require aggressive therapy.<sup>7</sup>

**ANSWER 6**

Practice level implementation of absolute CVD risk assessment can be supported by streamlining practice systems and optimising practice teams and processes, including:

- using registers to identify patients
- collecting missing data then undertaking assessment
- review and treat where recommended.

Each member of the practice team, including the receptionist, practice manager, practice nurse and GP, can contribute to implementing an absolute CVD risk program by:

- opportunistically identifying eligible patients: practice staff can review patient's records and flag them for an absolute CVD risk assessment
- excluding those patients not eligible (eg those under the age of 45 years and with existing cardiovascular disease): this will involve data cleaning at the practice level
- beginning with a subset of patients, for example, those with at least one known risk factor and then systematically collecting other risk factor information over a series of visits
- collecting missing data: this can be done by practice nurses, practice managers or GPs with assistance from receptionists, who can identify missing data required for an absolute CVD risk assessment and notify the GP of the tests required prior to the patient entering the consulting room

- undertaking risk assessments: any trained member of the practice team can conduct the assessment with or without the patient being present. Once pathology reports are received assessments can be done in 'batches', with nominated staff allocating dedicated time to enter patient details onto the calculator and to systematically record results. Alternatively, the patient could be invited to attend and the calculation can be performed with them. The approved absolute CVD risk assessment algorithm provided in the approved guidelines and/or website ([www.cvdcheck.org.au](http://www.cvdcheck.org.au)) should be used.

An audit tool, such as the Pen Computer Systems Audit Tool (PENCAT), can be used to identify patients at high risk of CVD and to target them for recall and/or education/follow-up.

Irrespective of the severity or intensity of a risk factor, each additional risk factor intensifies the overall risk of a cardiovascular event.<sup>8</sup> Therefore, conducting an absolute risk assessment and managing according to absolute risk has been taken up by many countries around the world. In general, the processes for implementing absolute CVD risk are comparable to the approach used by GPs to fulfill public health obligations, such as cervical cancer screening and immunisation.

### RESOURCES FOR DOCTORS

- National Heart Foundation, [www.heartfoundation.org.au/information-for-professionals/Clinical-Information/Pages/absolute-risk.aspx](http://www.heartfoundation.org.au/information-for-professionals/Clinical-Information/Pages/absolute-risk.aspx)
- Australian absolute cardiovascular disease risk calculator, [www.cvdcheck.org.au](http://www.cvdcheck.org.au)

### REFERENCES

1. National Vascular Disease Prevention Alliance. Guidelines for the management of absolute cardiovascular disease risk. 2012. Available at [www.nhmrc.gov.au/guidelines/publications/ext10](http://www.nhmrc.gov.au/guidelines/publications/ext10) [Accessed 17 September 2014].
2. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice. 8th edn. East Melbourne: RACGP, 2012. Available at [www.racgp.org.au/your-practice/guidelines/redbook](http://www.racgp.org.au/your-practice/guidelines/redbook) [Accessed 17 September 2014].
3. National Vascular Disease Prevention Alliance. Australian absolute cardiovascular disease risk calculator. Available at [www.cvdcheck.org.au](http://www.cvdcheck.org.au) [Accessed 25 September 2014].
4. Anderson KM, Odell PM, Wilson PW, Kannel WB. Cardiovascular disease risk profiles. *Am Heart J* 1991;121:293–98.
5. Jackson R, Lawes CM, Bennett DA, Milne RJ, Rodgers A. Treatment with drugs to lower blood pressure and blood cholesterol based on an individual's absolute cardiovascular risk. *Lancet* 2005;365:434–41.
6. Kannel WB. Some lessons in cardiovascular epidemiology from Framingham. *Am J Cardiol* 1976;37:269–82.
7. National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand. Reducing risk in heart disease: an expert guide to clinical practice for secondary prevention of coronary heart disease. Canberra: NHF, 2012. Available at [www.heartfoundation.org.au/SiteCollectionDocuments/Reducing-risk-in-heart-disease.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/Reducing-risk-in-heart-disease.pdf) [Accessed 25 September 2014].
8. Sundstrom J, Arima H, Woodward M, et al. Blood pressure-lowering treatment based on cardiovascular risk: a meta-analysis of individual patient data. *Lancet* 2014;384:591–98.

**CASE 3**

**ANGUS WORKS ON A FARM**

Angus is 17 years of age and works on his family's farm. Angus presents for review following an acute exacerbation 2 weeks ago of his longstanding asthma, which was first diagnosed at 5 years of age. He responded well to treatment and continues to use his regular preventive inhaler. He is concerned about future exacerbations, which always seem to occur during busy harvesting times when he can least afford to take time off work. These exacerbations are often associated with allergic rhinitis and conjunctivitis.

He smokes 20 cigarettes per day, drinks alcohol on 2–4 days each week and sometimes exceeds safe adult drinking guidelines. Angus recently got his 'P' plates. He and his family are well known to the local general practice.

Angus is well, apart from previous presentations for asthma, acute sinusitis and musculoskeletal injuries.

**QUESTION 1** 

What non-pharmacological measures can Angus take to reduce his risk of asthma exacerbations?

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**QUESTION 2** 

What is the role of allergy testing and desensitisation therapy in this case?

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**QUESTION 3** 

What risk factors does Angus have for chronic lung disease? How can they be managed?

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**FURTHER INFORMATION**

Angus left school at 15 years of age to help manage the family farm after his father's heart attack at the age of 47 years. Angus is now taking on much of the physical side of running the farm, while his father takes care of the business. The farm consists of mixed livestock and cropping and there are significant financial pressures.

**QUESTION 4** 

How would you assess Angus's psychosocial health risks?

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**QUESTION 5** 

Should Angus have cardiovascular and metabolic screening?

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and other occupational allergens could be triggers for Angus's asthma. Household triggers could include house dust mites.

Use of skin-prick testing would be ideal but may be limited for rural patients. Blood tests for allergen-specific immunoglobulin E (IgE), formerly known as the radioallergosorbent test (RAST), can be a useful alternative.<sup>5</sup> Testing may suggest desensitisation<sup>5</sup> as a management option and this could be carried out at the local general practice following consultation with an allergist.

### ANSWER 3

Angus has a number of risk factors for chronic lung disease. These include smoking, which is best managed as discussed earlier by encouraging and supporting smoking cessation. Exposure to allergens and other particles and gases is a risk for asthma and chronic respiratory disorders, including COPD, extrinsic allergic alveolitis and pneumoconiosis.<sup>6,7</sup>

It is important to educate Angus about his increased risk of work-related disease, particularly his risk for chronic respiratory illnesses associated with agriculture and exposure to dusts, bacteria, fungal spores and chemicals.<sup>8–11</sup> He should be aware of mechanisms to reduce these exposures in the occupational setting. Appropriate resources could also be made available to him, for example, discussing the availability of farmer resources and training on the Australian Centre for Agricultural Health and Safety website ([www.aghealth.org.au](http://www.aghealth.org.au)).

### ANSWER 4

About 50% of adult mental health disorders have an onset by age 14 years. Preventive guidelines, therefore, recommend screening of adolescents aged 12–18 years for major depressive disorder if systems are in place to facilitate an accurate diagnosis and provide appropriate psychotherapy and follow-up. A chronic medical condition, such as asthma, is a risk factor for depression.<sup>2</sup>

The Home, Education/Employment, Activities, Drugs, Sexuality and Suicide (HEADSS) framework offers a means of getting to know Angus and his strengths and risks in this regard. The HEADSS framework also offers a chance to provide brief messages and encourage risk reduction strategies, for example, in relation to alcohol and other drug use, driving and accidents, nutrition and sexual practices.<sup>2,12</sup>

Given that rural patients may find access to further assessment and/or care limited, if a significant psychological disorder is suspected, rural GPs may arrange alternatives such as online or telehealth services. Their Medicare Local may be of assistance in collating or providing local solutions to access problems. Note, however, that Medicare Locals are being phased out in 2015. GPs, and their patients, may find the website [www.mindhealthconnect.org.au](http://www.mindhealthconnect.org.au) a useful online repository of Australian mental healthcare resources. Access to means of suicide, especially firearms, is of importance in a psychosocial assessment.

The GP should use the 5As approach (assess, advice, agree, assist and arrange) with regard to Angus's smoking.<sup>3,13</sup> For detailed advice on smoking cessation, see the RACGP's *Supporting smoking cessation: a guide for health professionals*.<sup>3</sup>

### ANSWER 5

Angus should have cardiovascular and metabolic screening as he has several risk factors for these diseases, including smoking, family history and living in a rural area.

Angus's physical fitness also needs review, as fitness cannot be taken for granted in an age of labour-saving devices.

The RACGP's *Guidelines for preventive activities in general practice* recommend:<sup>2</sup>

- opportunistic assessment of nutrition and physical activity
- opportunistic assessment of height, weight and calculated body mass index (BMI)
- smoking assessment and early intervention from 10 years of age
- 2-yearly measurement of blood pressure from 18 years of age.

The guidelines do not recommend population screening for cholesterol and lipids, diabetes (except in Aboriginal and Torres Strait Islander peoples)<sup>14</sup> or assessment of absolute cardiovascular disease (CVD) risk in young adults;<sup>15</sup> however, these may be indicated on clinical grounds depending on other findings. In adults not known to have CVD or not clinically determined to be at high risk, absolute CVD risk assessments should be conducted every 2 years, starting from age 45, up to 74 years of age. For Aboriginal and Torres Strait Islander peoples, CVD risk assessments should commence at 35 years of age.<sup>2</sup>

### ANSWER 6

Angus left school early and may have missed his routine school-based immunisations, including the combined diphtheria, tetanus, pertussis vaccine (DTPa), cervical cancer and varicella vaccines.

An annual influenza vaccination is recommended for all people wishing to reduce their risk of influenza and for those with chronic illnesses such as severe asthma. There is insufficient data to make clear recommendations about the benefits of vaccination for people with mild–moderate asthma; however, it is known that influenza infection can cause severe exacerbations of wheezing. Note, vaccination may not necessarily reduce the risk or severity of asthma flare-ups during the influenza season.<sup>1,16</sup>

Q fever vaccination is recommended given the occupational risks (pre-vaccination skin testing is required).<sup>15</sup> It is worth noting that post-Q fever fatigue syndrome has been reported to occur in 10–15% of people who previously had acute Q fever.<sup>17–19</sup>

### ANSWER 7

The Australian Centre for Agricultural Health and Safety ([www.aghealth.org.au](http://www.aghealth.org.au)) has produced many useful resources, including *The Farm Health & Safety Toolkit for Rural General Practices* (the Toolkit; refer to *Resources for doctors*), to assist in injury and disease prevention for farmers. The website and Toolkit are highly recommended to GPs caring for farmers. The Toolkit provides a contextualised framework for addressing the health risks farmers face with regard to life-threatening injury on farms and roads, suicide, early death from CVD, skin and other cancers, exposure to organic dust, zoonoses and arboviruses, and hearing loss.

**RESOURCES FOR PATIENTS**

- The Australian Centre for Agricultural Health and Safety, [www.aghealth.org.au](http://www.aghealth.org.au)

**RESOURCES FOR DOCTORS**

- Australasian Society of Clinical Immunology and Allergy (ASCIa). Allergy and asthma, [www.allergy.org.au](http://www.allergy.org.au)
- The Australian Centre for Agricultural Health and Safety, [www.aghealth.org.au](http://www.aghealth.org.au)
- Farm Health & Safety Toolkit for Rural General Practices, [www.aghealth.org.au/tinymce\\_fm/uploaded/Health%20Workers/\\_gp\\_toolkit\\_booklet\\_lores.pdf](http://www.aghealth.org.au/tinymce_fm/uploaded/Health%20Workers/_gp_toolkit_booklet_lores.pdf)

**REFERENCES**

1. National Asthma Council Australia. The Australian Asthma Handbook – quick reference guide version 1.0. Melbourne: National Asthma Council Australia, 2014. Available at [www.astmahandbook.org.au](http://www.astmahandbook.org.au) [Accessed 1 September 2014].
2. The Royal Australian College of General Practitioners. Guidelines for preventative activities in general practice. 8th edn. Melbourne: RACGP, 2012 Available at [www.racgp.org.au/your-practice/guidelines/redbook](http://www.racgp.org.au/your-practice/guidelines/redbook) [Accessed 1 September 2014].
3. The Royal Australian College of General Practitioners. Supporting smoking cessation: a guide for health professionals. Melbourne: RACGP, 2011 [Updated 2014]. Available at [www.racgp.org.au/your-practice/guidelines/smoking-cessation/](http://www.racgp.org.au/your-practice/guidelines/smoking-cessation/) [Accessed 2 September 2014].
4. Rossi S, editor. Asthma. In: Australian Medicines Handbook. Adelaide: Australian Medicines Handbook Pty Ltd; 2013
5. Australasian Society of Clinical Immunology and Allergy. Skin prick testing for diagnosis of allergic diseases: a manual for practitioners. Balgowlah: ASCIA, 2013. Available at [www.allergy.org.au/health-professionals/papers/skin-prick-testing](http://www.allergy.org.au/health-professionals/papers/skin-prick-testing) [Accessed 2 September 2014].
6. United Kingdom Government Health and Safety Executive. Agriculture health and safety. Available at [www.hse.gov.uk/lung-disease/agriculture.htm](http://www.hse.gov.uk/lung-disease/agriculture.htm) [Accessed 8 September 2014].
7. Hoy RF. Respiratory problems: occupational and environment allergy exposures. *Aust Fam Physician* 2012;41:856–60.
8. Montano D. Chemical and biological work-related risks across occupations in Europe: a review. *J Occup Med Toxicol* 2014;9:28.
9. Ye M, Beach J, Martin JW, Senthilselvan A. Occupational pesticide exposures and respiratory health. *Int J Environ Res Public Health* 2013;10:6442–71.
10. Tual S, Clin B, Leveque-Morlais N, Raheison C, baldi I, Lebailly P. Agricultural exposures and chronic bronchitis: findings from the AGRICAN (AGRIculture and CANcer) cohort. *Ann Epidemiol* 2013;23:539–45.
11. Lee WJ, Cha ES, Moon EK. Disease prevalence and mortality among agriculture workers in Korea. *J Korean Med Sci* 2010;25:S112–18.
12. The Royal Australasian College of Physicians. Routine adolescent psychosocial health assessment – position statement. Approved by RACP Board July 2008. Available at [www.racp.edu.au/index.cfm?objectid=39396AC9-E30B-7941-0FD53740FF78DBC8](http://www.racp.edu.au/index.cfm?objectid=39396AC9-E30B-7941-0FD53740FF78DBC8) [Accessed 25 August 2014].
13. The Royal Australian College of General Practitioners. Putting prevention into practice: guidelines for the implementation of prevention in the general practice setting. 2nd edn. Melbourne: RACGP, 2006. Available at [www.racgp.org.au/download/documents/Guidelines/Greenbook/racpgreenbook2nd.pdf](http://www.racgp.org.au/download/documents/Guidelines/Greenbook/racpgreenbook2nd.pdf) [Accessed 1 September 2014].
14. National Aboriginal Community Controlled Health Organisation. National guide to a preventive health assessment for Aboriginal and Torres Strait Islander people. 2nd edn. Melbourne: The RACGP, 2012. Available at [www.naccho.org.au/promote-health/national-guide-to-a-preventive-health-assessment/](http://www.naccho.org.au/promote-health/national-guide-to-a-preventive-health-assessment/) [Accessed 1 September 2014].
15. National Vascular Disease Prevention Alliance. Guidelines for the management of absolute cardiovascular disease risk. 2012. Available at [http://strokefoundation.com.au/site/media/AbsoluteCVD\\_GL\\_webready.pdf](http://strokefoundation.com.au/site/media/AbsoluteCVD_GL_webready.pdf) [Accessed 3 September 2014].
16. Australian Technical Advisory Group on Immunisation. The Australian Immunisation Handbook. 10th edn. Canberra: Australian Government Department of Health, 2013. Available at [www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home](http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home) [Accessed 1 September 2014].
17. Marmion BP, Shannon M, Maddocks I, Storm P, Penttila I. Protracted debility and fatigue after acute Q fever [letter]. *Lancet* 1996;347:977–78.
18. Penttila IA, Harris RJ, Storm P, et al. Cytokine dysregulation in the post-Q fever fatigue syndrome. *QJM* 1998;91:549–60.
19. Ayres JG, Flint N, Smith EG, et al. Post-infection fatigue syndrome following Q fever. *QJM* 1998;91:105–23.

**CASE 4**

**BARBARA REQUESTS A ROUTINE PAP SMEAR**

Barbara, a businesswoman aged 48 years, has booked a double appointment for a routine Pap smear. She is often busy with her work and finds it difficult to make time to attend routine medical appointments. She looks slightly embarrassed as she admits that she is not sure when her last Pap smear was done.

**QUESTION 1** 

What information should you obtain at a visit for a routine Pap smear?

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**FURTHER INFORMATION**

Barbara used to have regular Pap smears but in the past 10 years she has been having them less regularly because of increased pressures at work. She thinks her last Pap smear was 3 years ago and has she has never had an abnormal Pap smear result. She is currently single, and has had six casual male partners over the past 12 months. Prior to this, she had been married for 20 years.

When asked about lifestyle, Barbara says she is a smoker and has smoked about 10 cigarettes per day for the past 25 years. She has a balanced diet (ie regular meals and snacks in moderation) and walks to and from work each day (20 minutes each way). Her body mass index (BMI) is 21 kg/m<sup>2</sup>, which is within the normal range.

When asked about genital and continence symptoms, Barbara reveals she has had problems with thrush. For the past year, she has had vulval itch and soreness, which have failed to settle despite multiple courses of clotrimazole cream and fluconazole tablets. She has not noticed any changes to her vaginal discharge.

**QUESTION 2** 

What are possible causes of Barbara’s vulval itch?

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**FURTHER INFORMATION**

After allowing Barbara some privacy and time to undress appropriately for her Pap smear, and to cover up with a sheet, you join her at the examination couch. The Pap smear is easily performed. You examine the external genitalia as well as the vaginal walls and cervix in good light, with Barbara positioned to ensure adequate visualisation of these areas. The vaginal walls and cervix appear unremarkable and there is no abnormal discharge seen. You check for the presence or absence of rash, erythema, pallor, other colour change, lichenification, erosions and any change to the normal architecture including atrophic changes or the presence of adhesions. Examination of the vulva identifies an obvious abnormality of the inferior labia bilaterally (*Figures 1, 2*). Barbara confirms these are areas of maximum itch. There are no palpable inguinal lymph nodes and the mons appears normal.



Figure 1. External genitalia demonstrating an area of abnormality affecting the inferior labia majora



Figure 2. Detail of the affected area demonstrating abnormality of both labia majora and both labia minora inferiorly, including the navicular fossa

**QUESTION 3**

What is the most likely diagnosis now? What serious cause must be excluded?

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**QUESTION 4**

What investigations should be ordered at this stage?

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**FURTHER INFORMATION**

Barbara agrees to have a punch biopsy performed. The result is reported by telephone the following day as 'florid vulval intraepithelial neoplasia 3 (VIN3) with features consistent with HPV infection'. She is contacted to come in to discuss the results and is referred to her nearest gynaecology-oncology service.

A wide excision biopsy is performed by her gynaecology-oncologist and the biopsy is later reported as squamous cell carcinoma (SCC). Pelvic ultrasound is unremarkable.

Barbara has an uncle who had multiple SCCs on his face and was told these were caused by smoking. She asks if smoking caused her SCC.

**QUESTION 5**

How would you respond to Barbara's query about smoking?

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**QUESTION 6**

What ongoing care will Barbara need?

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## CASE 4 ANSWERS

## ANSWER 1

Current guidelines recommend that Pap smears should be done every 2 years for any woman who has ever had sex (and still has an intact cervix), from the age of 18 years or 2 years post-coitarche (whichever is later) up to the age of 70 years.<sup>1</sup> Note, these guidelines are currently under review.<sup>2,3</sup> Pap smear visits provide the opportunity to review past Pap smear history, any current urogenital symptoms and sexual history, and to undertake any other preventive activities appropriate for the woman's age range.<sup>1</sup>

Depending on the time available and clinical judgment regarding the patient's individual risk factors and presenting concerns, questions could be asked about Pap smear history, current symptoms and sexual history. Examples of questions include:

- **Pap smear history:**
  - Has she had a Pap smear before?
  - When was the last Pap smear?
  - Has she had any previous abnormal Pap smear results? If so, when and did she require any treatment?
  - Has she had any human papillomavirus (HPV) vaccinations?
- **Current symptoms:**
  - Does she have any concerns about her genital area?
  - Are there any lumps, bumps or rashes?
  - Is she suffering from itch?
  - Has her vaginal discharge changed recently?
  - Is she having any pain with urination or with sex?
  - Is she experiencing bleeding after sex?
  - Have her periods changed recently?
  - Does she have any urinary or faecal continence issues?
- **Sexual history:**
  - Is she in a regular relationship?
  - Does she have casual sexual partners?
  - Does she have male/female partners or both?
  - What types of sexual activity does she engage in?
  - When was her last unprotected sex?
  - Has she ever been screened for sexually transmissible infections (STIs)? If so, when and did she require any treatment?
  - What contraception is she using?

Examples of a more detailed sexual history are reviewed in *check* unit 496 July 2013 – Sexuality and Sexual Health and in the National Sexual Health Guidelines.<sup>4</sup>

## ANSWER 2

Vulval itch is a common symptom in women and is easily assumed to be vulvovaginal candidiasis. Nevertheless, itch can have a wide range of

causes.<sup>5</sup> It is important to note that chronic vulvovaginal candidiasis is unusual in postmenopausal women who are otherwise healthy and not taking hormone replacement therapy (HRT).<sup>6</sup>

Common and important dermatological causes of vulval itch include:<sup>5,7,8</sup>

- vulval dermatitis (atopic or contact)
- lichen simplex chronicus
- lichen sclerosus, lichen planus
- plasma cell vulvitis
- psoriasis
- vulvovaginal candidiasis
- *Streptococcus agalactiae* vulvitis
- dermatophytosis
- VIN
- SCC
- STIs
- atrophic vaginitis.

## ANSWER 3

The changes seen on examination are consistent with HPV infection, showing areas of discrete wart growth and areas of less well-defined changes consistent with dysplasia VIN/SCC. SCC needs to be excluded.

HPV infection is very common and a history of genital warts is reported by 5–10% of women in northern Europe, UK, USA and Australia.<sup>9</sup> However, vulval cancers are relatively rare – fewer than 300 cases are reported annually in Australia. These cancers account for 0.4% of cancer deaths in women, with a 5-year relative survival rate of approximately 72%.<sup>10</sup>

## ANSWER 4

A histological diagnosis is essential at this stage. Depending on the services available locally, Barbara might have a punch biopsy taken in the GP rooms, she might be referred to a dermatologist, plastic surgeon or general surgeon for biopsy, or might be referred directly to a specialist gynaecological-oncology team for further assessment and management.

Histological grade can vary substantially between regions of a given lesion<sup>11</sup> and VIN/SCC may be multifocal.<sup>12</sup> Therefore, where punch biopsy is utilised, the entire anogenital region should be examined carefully for skin changes and the biopsy taken from the most abnormal area. If appropriate, multiple biopsies should be taken.<sup>11</sup>

There are a wide range of treatment options for VIN/SCC, including local ablation, topical medical management and surgical options;<sup>7,13,14</sup> however, surgical management is the gold standard for the management of both VIN and SCC.<sup>13,14</sup> Wide local excision by a specialist service may therefore be both an initial investigation and appropriate management.

Given Barbara's history of recent partner change, STI screening is appropriate (and should include chlamydia, human Immunodeficiency virus (HIV), syphilis and hepatitis B screening),<sup>1</sup> as is vulval/vaginal microscopy, culture and swab (MCS) to exclude infectious of itch or superinfection at the affected site.<sup>7</sup>

**ANSWER 5**

Smoking has been found to increase the risk of cutaneous SCC by approximately 50%<sup>15</sup> and the risk of smoking associated SCC is reported to be threefold greater in women, compared with men.<sup>16</sup> Identified risks for vulval SCC include tobacco use, HPV exposure, alcohol intake, immunosuppression, chronic dermatoses, radiotherapy and chemotherapy.<sup>17–19</sup> Women who smoke are also at increased risk of VIN recurrence.<sup>20</sup>

**ANSWER 6**

Studies have found that women with SCC benefit from multidisciplinary care, ideally involving a centralised specialist cancer service.<sup>19,21</sup> The anogenital region, including the draining lymph nodes, will require ongoing monitoring for recurrence, and the importance of ongoing routine Pap smears must be emphasised as there is an association between VIN/SCC and cervical intraepithelial neoplasia (CIN).<sup>22</sup>

Vulvovaginal conditions and their management can have a significant negative impact on sexual identity and function, both psychologically and physically.<sup>23,24</sup> Barbara will need ongoing emotional and psychological support and may benefit from referral to a sexual health counsellor.

**RESOURCES FOR PATIENTS**

- The Cancer Council NSW. Understanding vulva and vagina cancers, a guide for women with cancer, their families and friends 2011, [www.cancercouncil.com.au/wp-content/uploads/2011/10/Understanding-Vulva-and-Vagina-Cancers\\_LR-website.pdf](http://www.cancercouncil.com.au/wp-content/uploads/2011/10/Understanding-Vulva-and-Vagina-Cancers_LR-website.pdf)
- Martin KA et al (Eds.) UpToDate. Patient information: vulvar itching (the basics). Topic 83970 Version 2. 2014, [www.uptodate.com/contents/vulvar-itching-the-basics?source=search\\_result&search=vulvar+itch&selectedTitle=2~29](http://www.uptodate.com/contents/vulvar-itching-the-basics?source=search_result&search=vulvar+itch&selectedTitle=2~29)

**REFERENCES**

1. The Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice 8<sup>th</sup> edn. Melbourne: RACGP, 2012. Available at [www.racgp.org.au/your-practice/guidelines/redbook/](http://www.racgp.org.au/your-practice/guidelines/redbook/) [Accessed 30 September 2014].
2. Australian Government Medical Services Advisory Committee. Application no 1276 – Final decision. Analytic protocol to guide the assessment of the National Cervical Screening Program. 2014. Available at [www.msac.gov.au/internet/msac/publishing.nsf/Content/FD36D6990FFAA639CA25799200058940/\\$File/1276-NCSP-FinalDAP.pdf](http://www.msac.gov.au/internet/msac/publishing.nsf/Content/FD36D6990FFAA639CA25799200058940/$File/1276-NCSP-FinalDAP.pdf) [Accessed 28 October 2014].
3. Australian Government Medical Services Advisory Committee. Application no 1276 – Renewal of the National Cervical Screening Program. 2012. Available at [www.msac.gov.au/internet/msac/publishing.nsf/Content/FD36D6990FFAA639CA25799200058940/\\$File/1276%20-%20Final%20MSAC%20PSD%20-%20NCSP%20Renewal.pdf](http://www.msac.gov.au/internet/msac/publishing.nsf/Content/FD36D6990FFAA639CA25799200058940/$File/1276%20-%20Final%20MSAC%20PSD%20-%20NCSP%20Renewal.pdf) [Accessed 28 October 2014].
4. Australasian Sexual Health Alliance. Australian STI management guidelines for use in primary care. Available at [www.sti.guidelines.org.au](http://www.sti.guidelines.org.au) [Accessed 28 October 2014].
5. Stander S, Weisshaar E, Mettang T et al. Clinical classification of itch: a position paper of the International Forum for the Study of Itch. *Acta Derm Venereol* 2007;87:291–94.
6. Antibiotic Expert Group. Genital and sexually transmitted infections. Vulvovaginal candidiasis. In eTG Complete [Internet] Melbourne. Therapeutic Guidelines. Ltd 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 15 September 2014].

7. British Association for Sexual Health and HIV. 2014 UK National guideline on the management of vulval conditions. Available at [www.bashh.org/documents/UK%20national%20guideline%20for%20the%20management%20of%20vulval%20conditions%202014.pdf](http://www.bashh.org/documents/UK%20national%20guideline%20for%20the%20management%20of%20vulval%20conditions%202014.pdf) [Accessed 30 September 2014].
8. Drummond C. Common vulval dermatoses. *Aust Fam Physician* 2011;40:490–96.
9. Dochez C, Bogers JJ, Verhelst R, Rees H. HPV vaccines to prevent cervical cancer and genital warts: an update. *Vaccine* 2013;32: 1595–1601.
10. Australian Institute of Health and Welfare and Cancer Australia 2012. Gynaecological cancers in Australia: an overview. Cancer series no. 70. Cat. no. CAN 66. Canberra: AIHW, 2012.
11. Polterauer S, Catharina Dressler A, Grimm C et al. Accuracy of preoperative vulva biopsy and the outcome of surgery in vulvar intraepithelial neoplasia 2 and 3. *Int J Gynecol Pathol* 2009;28:559–62.
12. van Beurden M, ten Kate FJ, Smits HL et al. Multifocal vulvar intraepithelial neoplasia grade III and multicentric lower genital tract neoplasia is associated with transcriptionally active human papillomavirus. *Cancer* 1995;75:2879–84.
13. Royal College of Obstetricians and Gynaecologists. Vulval skin disorders (management). Green-top Guideline 58. London: RCOG, 2011. Available at [www.rcog.org.uk/globalassets/documents/guidelines/gtg58vulval22022011.pdf](http://www.rcog.org.uk/globalassets/documents/guidelines/gtg58vulval22022011.pdf) [Accessed 30 September 2014].
14. Royal College of Obstetricians and Gynaecologist, British Gynaecological Cancer Society. Guidelines for the diagnosis and management of vulval carcinoma. London: RCOG, 2014. Available at [www.rcog.org.uk/globalassets/documents/guidelines/vulvalcancer guideline.pdf](http://www.rcog.org.uk/globalassets/documents/guidelines/vulvalcancer guideline.pdf) [Accessed 27 October 2014].
15. Leonardi-Bee J, Ellison T. Smoking and the risk of nonmelanoma skin cancer: Systematic review and meta-analysis. *Arch Dermatol* 2012;148:939–46.
16. Rollison DE, Lannacone MR, Messina JL et al. Case-control study of smoking and non-melanoma skin cancer. *Cancer Causes Control* 2012;23:245–54.
17. Ansink A. Vulvar squamous cell carcinoma. *Semin Dermatol* 1996;15:51–59.
18. Madsen BS, Jensen HL, van den Brule AJ, Wohlfahrt J, Frisch M. Risk factors for invasive squamous cell carcinoma of the vulva and vagina – population-based case-control study in Denmark. *Int J Cancer* 2008;122:2827–34.
19. Kutlubay Z, Engin B, Zara T, Tuzun Y. Anogenital malignancies and premalignancies: facts and controversies. *Clin Dermatol* 2013;31:362–73.
20. Leonard B, Kridelka F, Delbecque K et al. A clinical and pathological overview of vulvar condyloma acuminatum, intraepithelial neoplasia, and squamous cell carcinoma. *BioMed Res Int* 2014; doi:10.1155/2014/480573
21. Yap JK, Baker LJ, Balega JZ, Chan KK and Luesley DM. 2011. Impact of improving outcome guidance in gynaecological cancer on squamous cell carcinoma of the vulva in the West Midlands, UK. *J Obstet Gynaecol* 31:754–58.
22. de Bie RP, van de Nieuwenhof HP, Bekkers RL, et al. Patients with usual vulvar intraepithelial neoplasia-related vulvar cancer have an increased risk of cervical abnormalities. *Br J Cancer* 2009;101:27–31.
23. Andreasson B, Moth I, Jensen SB, Bock JE. Sexual function and somatopsychic reactions in vulvectomy-operated women and their partners. *Acta Obstetrica* 1986;65:7–10.
24. Dominiak-Felden G, Cohet C, Atrux-Tallau S, Gilet H, Tristram A, Fiander A. 2003. Impact of human papillomavirus-related genital diseases on quality of life and psychosocial wellbeing: results of an observational health-related quality of life study in the UK. *BMC Public Health* 2003;13:1065–76.

**CASE 5**

**JENNY HAD A HEART ATTACK**

Jenny, 62 years of age, is a retired seamstress who lives with her husband. She has been coming to your practice for many years. She has four children and six grandchildren. You have treated her previously for angina, hypertension and osteoporosis. She has a history of non-adherence to her angina medication because of dizziness. She presents today following discharge from hospital after experiencing a heart attack last week.

**QUESTION 1** 

What would be important for you to consider in your assessment of Jenny following her myocardial infarction (MI)?

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**FURTHER INFORMATION**

During her admission to hospital, Jenny had a drug-eluting stent implanted. Her cardiologist discharged her with a few changes to her medication, which you can see on the discharge summary Jenny has given you.

New medication list:

- Perindopril 10 mg one tablet daily (*altered dose*)
- Atenolol 50 mg one tablet daily (*unchanged*)
- Atorvastatin 40 mg one tablet daily (*altered dose*)
- Aspirin 100 mg one tablet daily (*new*)
- Clopidogrel 75 mg one tablet daily (*new*)
- Calcium carbonate 600 mg, two tablets daily (*unchanged*)
- Alendronate 70 mg/cholecalciferol 70 µg weekly (*unchanged*)

The pharmacist also provided Jenny with a new glyceryl trinitrate spray and counselled her on its use, along with a heart attack action plan.

It is unclear whether Jenny has been referred for cardiac rehabilitation. Her discharge letter indicates she will be seen again in the cardiology clinic but does not indicate when and she has not received an appointment as yet.

**QUESTION 2** 

In developing a management plan for Jenny, what would you consider?

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**FURTHER INFORMATION**

History and examination reveal that Jenny has never smoked but her husband does, she exercises irregularly and has 3–4 glasses of wine at the weekends.

Physical examination findings are:

- height: 177 cm
- weight: 94 kg
- body mass index (BMI): 31 kg/m<sup>2</sup> (healthy range: 18.5–24.9 kg/m<sup>2</sup>)
- blood pressure (BP): 130/80 mmHg (goal <130/80 mmHg)
- waist circumference: 91 cm (goal <80 cm for women)
- cardiac exam: normal
- peripheral pulses: normal
- no organomegaly.

Laboratory/investigation results obtained before discharge from hospital are:

- urea and electrolytes (UAEs), full blood evaluation (FBE), liver function tests (LFTs), thyroid stimulating hormone (TSH): all within normal range
- triglycerides (TG): 1.6 mmol/L (<2.0 mmol/L)
- high-density lipoprotein cholesterol (HDL-C): 1.1 mmol/L (>1.0 mmol/L)
- low-density lipoprotein cholesterol (LDL-C): 2.2 mmol/L (<1.8 mmol/L)
- HbA1C: 42 mmol/mol (6.0%)
- urinalysis: without protein
- resting electrocardiogram (ECG): old anterior infarction with left axis deviation, otherwise normal.

**QUESTION 3** 

How would you interpret these results? What action should be taken?

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**QUESTION 4** 

What medications are commonly prescribed for people requiring secondary prevention of coronary heart disease (CHD)?

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**QUESTION 5** 

How would you approach or structure a discussion to provide lifestyle advice for Jenny?

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**QUESTION 6** 

What advice regarding lifestyle and psychosocial risk factors would you give to patients such as Jenny following an MI?

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**QUESTION 7** 

How would attending a cardiac rehabilitation program help Jenny?

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**FURTHER INFORMATION**

Jenny complains of not sleeping well and having too many medicines. She is not sure why she needs all these medicines when she has a stent. She asks 'haven't I been fixed?'.

**QUESTION 8** 

How would you manage Jenny's sleep concerns?

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**QUESTION 9** 

How would you respond to Jenny's concerns regarding having 'too many' medications?

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## CASE 5 ANSWERS

**ANSWER 1**

The initial assessment of Jenny after her MI provides you with an opportunity to re-assess her condition, current symptomology and her risk factors. This information will help to determine her risk of future cardiovascular events and inform your management plan. The key components of the initial assessment should include:<sup>1,2</sup>

- Review her discharge summary, including the diagnosis, procedures performed during hospitalisation, risk factors for follow-up, new or altered medicines, referral to cardiac rehabilitation or other specialist or allied health services.
- Identify cardiovascular disease (CVD) risk factors, with an emphasis on modifiable risk factors.
- Assess individual physical, social, cultural and psychosocial factors that would influence their ability to engage in behaviour change strategies.

This information can be gathered through a combination of a review of her health record, the hospital discharge summary and the consultation with Jenny (and her family member, if in attendance). The outcomes of this assessment will assist you to personalise Jenny's management according to her prognosis, comorbidities, medication tolerance, lifestyle and living circumstances, and wishes.<sup>1,2</sup>

**ANSWER 2**

The following could be considered in a management plan for Jenny:<sup>1,2</sup>

- Review current treatment/medications to address cardiac risk factors.
- Organise follow-up tests and ongoing review.
- Consider referral for cardiac rehabilitation or secondary prevention if not already referred.
- Consider a team care arrangement to help Jenny access Medicare-rebated allied health services, especially if she does not attend cardiac rehabilitation.
- Consider a home medicines review (HMR), given Jenny's recent hospital admission, changes to her medications and that she takes more than five medications per day. Additionally, a medication review could be considered when poor medication adherence is suspected.
- Provide written information outlining the warning signs of heart attack and an action plan for patients to follow in the event they have warning signs of a heart attack.
- Provide education/reinforcement as to what to do if further symptoms arise.
- Provide written information and self-management resources, such as *My heart for life* and *My heart my life*, available from the National Heart Foundation (NHF).

**ANSWER 3**

Most of these results are normal. The main results that require action are the cholesterol readings. Ideally, Jenny's HDL-C should be higher, while her LDL-C should be lower. Given Jenny is already taking a statin, this should be continued to reduce her LDL-C. Jenny should be encouraged to increase her physical activity to raise her HDL-C.<sup>3</sup>

**ANSWER 4**

A number of medications may be prescribed for patients who require secondary prevention. These include:<sup>2</sup>

**Antiplatelet agents**

The usual antiplatelet regimen consists of aspirin (at least 75 mg up to 150 mg/day) indefinitely and clopidogrel (75 mg/day). Drug-eluting stents have been associated with late restenosis and, therefore, an increasing number of cardiologists recommend combination therapy (aspirin plus clopidogrel) for at least 1 year and, in some instances, indefinite combination therapy for patients with drug-eluting stents.<sup>4</sup>

**Anticoagulants**

The NHF 2012 *Guidelines for reducing risk in heart disease*<sup>2</sup> recommend use of warfarin in patients at high risk of thromboembolism due to atrial fibrillation (AF) post-MI. The NHF does not presently provide guidance on the use of newer anticoagulants for AF prevention in this context. Other guidelines such as the *Australian Medicines Handbook 2014* recommend consideration of warfarin for secondary prevention in all people with AF, with newer agents such as apixaban, dabigatran or rivaroxaban suggested as alternatives to warfarin in patients without a prosthetic heart valve or significant valvular disease.<sup>5</sup> *Therapeutic Guidelines 2012* recommend use of warfarin or dabigatran for patients with AF requiring long-term anticoagulation.<sup>6</sup>

**Statins**

Statins should be prescribed on a risk basis, not on lipid levels. Jenny has been commenced on a statin because of her risk of a secondary event. Statins are recommended for all patients with CHD, unless contraindicated.

**Angiotensin converting enzyme inhibitors (ACEIs)/angiotensin II receptor blockers (ARBs)**

These are recommended for all patients unless contraindicated, especially those at high risk of recurrent events, and should be started early post-MI. Consider an ARB for patients who develop unacceptable side effects with ACEIs such as persistent, distressing coughing.

**Beta-blockers**

Beta-blockers are recommended for all patients post-MI, unless contraindicated, especially in high-risk patients. High-risk patients are defined as those with significant myocardial necrosis, left ventricular systolic dysfunction, persistent evidence of ischaemia or ventricular arrhythmia.

The benefit of long-term treatment with beta blockers post-MI is well-established. Given Jenny has achieved her target BP (<130/80 mmHg),

she may not require both beta blocker and ACEI therapy, unless she shows signs of heart failure, left ventricular (LV) dysfunction or develops diabetes.<sup>7–11</sup>

### Short-acting nitrates

These are recommended for all patients with CHD, unless contraindicated. As mentioned earlier, all patients should be provided with a written action plan to manage chest pain.

Patients should have the risks and possible side effects, and benefits of their medications explained to them and should be provided with written information for drugs such as warfarin that have a narrow therapeutic index.

### ANSWER 5

The 5As is a model that can be used to provide Jenny with structured and suitable lifestyle advice. The 5As include:<sup>12,13</sup>

#### Assess

Begin with an assessment of Jenny's knowledge, beliefs and behaviours and your review of her clinical data. This first step provides useful feedback to Jenny on her health status. Include questions about lifestyle factors such as smoking, physical activity, diet, medicine adherence, psychosocial health (as above) and health literacy.

#### Advice

The next step is to tell Jenny about her health risks and the benefits of change. Offer brief advice with clear messages of encouragement to change.

#### Agree

This step involves working with Jenny to set specific behavioural goals on the basis of her interests, confidence and priorities. Use empathic listening to encourage Jenny to share her beliefs and to develop an understanding about her disease and to negotiate appropriate management plans.

#### Assist

Explore Jenny's commitment to her goals and discuss any barriers and concerns she may have. This could include a referral for Jenny to attend a suitable cardiac rehabilitation program if she has not done so already, or to attend another long-term prevention program such as 'NHF Walking' or 'Heart Moves'.

#### Arrange

The final step involves arranging a follow-up plan. The GP management team needs to be aware of Jenny's action plan and to reinforce her goals. Arrange for Jenny to have regular follow up visits with a GP or practice nurse.

This method allows for short consultations structured around detection, assessment and management of her risk factors. It will engage Jenny by encouraging her to discuss her experiences and issues, thus focusing the clinical encounter around Jenny and her concerns. This approach allows for Jenny to make decisions regarding her health, while giving consideration to her condition, lifestyle, psychosocial factors and individual capabilities and barriers. As a health professional, this model also allows for the

assessment of Jenny's readiness to change so that a suitable treatment plan is agreed.

### ANSWER 6

Patients should be provided with advice to manage any lifestyle and psychosocial risk factors that apply for them. For example:<sup>2</sup>

#### Smoking

Advise complete cessation of smoking (for smokers) and avoidance of the husband's second-hand smoke. Consider referral to Quitline (13 7848), a specialised smoking cessation program, use of nicotine replacement therapy (NRT) in selected patients and pharmacotherapy.

#### Return to an active lifestyle

People with heart disease gain great benefits from regular exercise and it can assist them with return to a normal life after a heart attack. Exercise can also improve other risk factors such as lowering BP and cholesterol and maintaining a healthy weight.

Encourage Jenny to continue to follow the physical activity advice from her rehabilitation program if she has attended one. If she hasn't, inform Jenny that she can resume normal physical activities as long as you consider it safe.

#### Physical activity

Patients should be advised to progress, over time, to at least 30 minutes of moderate-intensity physical activity on most, if not all, days of the week (150 minutes per week minimum). Regular, moderate-intensity physical activity such as walking is ideal. You could refer Jenny to a local and free NHF Walking Group through the NHF website.

#### Nutrition/healthy eating

Healthy eating and drinking is an important part of Jenny's recovery. It can reduce her risk of future events by reducing her BP and cholesterol and helping her achieve a healthy weight. Encourage the establishment/maintenance of healthy eating patterns and recommend that Jenny limit her salt intake to  $\leq 4$  g/day (1550 mg sodium). The following healthy eating goals could be discussed and the information provided to Jenny in a written format:

- Reduce intake of saturated and trans fats, which are found in fatty meats, full-cream dairy products, butter, coconut and palm oils, most fried foods and commercially baked products.
- Eat vegetables, whole grains, fruit, nuts and seeds everyday.
- Eat 2–3 serves of oily fish per week.
- Use healthier fats and oils such as olive, canola, sunflower, soybean, sesame and peanut oils.
- Avoid or limit fried or baked foods including chips, biscuits, cakes and other baked cereal products.
- Limit sugary, fatty and salty takeaway meals and snacks.
- Avoid adding salt to foods and choose foods stating 'no added salt', 'low salt' or salt reduced' where possible.
- Drink mainly water.

You could refer Jenny to the NHF website for heart healthy recipe ideas.

**Alcohol**

Low-risk alcohol consumption should be encouraged in all people with CHD. All patients should be advised to drink  $\leq 2$  standard drinks per day. Women with high BP or who are taking antihypertensive medicine should drink  $\leq 1$  standard drink per day.

**Healthy weight**

Jenny is currently overweight. Carrying excess body weight can have a serious negative impact on Jenny's health and can increase her risk of another heart event, developing diabetes and a range of other diseases. It is not about dieting or running marathons – it is about making small, easy changes so that they become the norm for life. Slow progress is more likely to deliver long-term results.

Advice for constructing a weight management plan can be found in the National Health and Medical Research Council (NHMRC) summary guide for the management of overweight and obesity in primary care 2013, which uses the 5As approach.<sup>14</sup> Jenny will need coaching regarding her weight, aiming for weight loss to achieve a waist measurement  $\leq 80$  cm (females), with BMI = 18.5–24.9 kg/m<sup>2</sup>. Consider referral to professionals such as an accredited practicing dietician or an exercise physiologist.

**Depression**

Assess all patients for comorbid depression. Initiate psychosocial and medical management if appropriate.

**Social support**

Assess all patients for their level of social support and provide follow-up for people considered at risk by referral to cardiac rehabilitation and/or a social worker or psychologist.

**ANSWER 7**

The aim of cardiac rehabilitation programs is to assist people with heart disease return to a full, active and satisfying life as quickly as possible. They can also help prevent further heart events, such as a second heart attack. For Jenny, a program can:<sup>2</sup>

- help her learn about her condition
- provide an exercise program tailored to her needs and condition
- guide her to change her lifestyle to improve heart health
- educate her in taking her prescribed medicines and assist with medication adherence
- answer any questions or practical concerns she may have about living with heart disease
- support her social and emotional wellbeing during this time of change.

In addition to these core goals, attending a program can also:<sup>2</sup>

- increase her independence and confidence
- reduce depression and anxiety
- connect her with other people in similar situations
- increase her ability to be physically active.

**ANSWER 8**

Jenny's sleeping problems could be an indication of depression or anxiety. The prevalence of depression is high in patients with CHD. Rates of major depressive disorder of around 15% have been reported in patients following MI or coronary artery bypass grafting. The benefits of treating depression include improved quality of life and adherence to therapy, and potentially improved CHD prognosis.<sup>15</sup>

The NHF recommends that all patients with CHD be routinely screened for depression by their GP or a health professional at first presentation, using the Patient Health Questionnaire 2 (PHQ2) shown below (*Table 1*). If Jenny answers yes to either question in the PHQ2, it is recommended that GPs follow up with the Patient Health Questionnaire 9 (PHQ9),<sup>16</sup> which can be used to quantify depression severity, assess change over time and response to treatment. This should be repeated at the next follow-up appointment.<sup>16</sup>

Preparation of a GP mental health treatment plan could be considered to help Jenny access Medicare-rebated psychology services.

**Table 1. Patient Health Questionnaire (PHQ2)<sup>16</sup>**

During the past month, have you often been bothered by feeling down, depressed or hopeless? Yes/No

During the past month, have you often been bothered by little interest or pleasure in doing things? Yes/No

**ANSWER 9**

It's important to talk to Jenny about the importance and benefits of adherence to her medications, particularly given her history of non-adherence to her angina medication and her possible depression following her MI. There are a number of options that might assist her in managing her medication adherence:

- Referring Jenny for a HMR may be useful. This will involve an accredited pharmacist visiting Jenny in her home environment and discussing the medicines she takes and the troubles she has with adherence.<sup>17</sup>
- Use of combination medicines could be an option to reduce the number of individual tablets Jenny is taking.<sup>18</sup> First, a combination clopidogrel/aspirin product could be considered, given that Jenny is likely to continue this therapy long term. Also, Jenny currently takes a combination of calcium carbonate and alendronate/cholecalciferol for osteoporosis. This would further reduce the number of tablets she takes. Although there are a number of combination products available for BP and cholesterol, it may be advisable for Jenny to continue taking these medicines individually until her dose(s) are stabilised. This may take up to 1 month following the most recent dose change.<sup>19</sup>
- Price isn't always a barrier to adherence but should be considered. If Jenny finds that the cost of the co-payments of her new medication regime are adding up, she may benefit from use of generic medicines, along with provision of information to understand which medicines are the same, so she does not confuse different brands as different medicines.

- A dose administration aid, either self-filled or prepared by her local pharmacy, may also improve Jenny's adherence.

### RESOURCES FOR PATIENTS

- My Heart My Life is a patient resource produced by the Heart Foundation containing information and action plans important to recovery – including healthy eating, physical activity, smoking cessation, medicines, services and support, heart disease facts and the warning signs of a heart attack. It is now available as a mobile app or can be ordered through the Heart Foundation's Health Information Service (1300 36 27 87) or online at [heartfoundationshop.com/main\\_menu/](http://heartfoundationshop.com/main_menu/)
- The Heart Foundation, [www.heartfoundation.org.au](http://www.heartfoundation.org.au), and the Health Information Service, provide free, personalised information and support on heart health, nutrition and healthy lifestyle. The service is run by qualified health professionals and an appointment is not needed. Call the Health Information Service on 1300 36 27 87 (cost of a local call).

### RESOURCES FOR DOCTORS

- The Heart Foundation has produced a toolkit for health professionals called *Improving Adherence in cardiovascular care*. This resource is also available as a series of learning modules for GPs through ThinkGP and has been accredited with RACGP/ACCRM for continuing professional development. Access the toolkit at [http://www.heartfoundation.org.au/SiteCollectionDocuments/FAT-WEB\\_20130420.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/FAT-WEB_20130420.pdf) or go to ThinkGP for more information and access to the learning modules.
- A General Practice Management Plan (GPMP) template is available to download from the Heart Foundation website. Visit <http://www.heartfoundation.org.au/information-for-professionals/Clinical-Information/Pages/gp-management-plan-chd.aspx>
- HEART Online is an online resource developed to support clinicians in delivering evidence-based care in cardiovascular disease prevention and rehabilitation, and heart failure management. Visit <http://www.heartonline.org.au/Pages/default.aspx>
- The Australian Dietary Guidelines were endorsed by the NHMRC before their release in 2013 and have been developed to provide recommendations for all Australians. They are available to download in full or part <https://www.nhmrc.gov.au/guidelines/publications/n55>
- In addition to My Heart My Life, listed below, the Australian Government has developed a physical activity resource for older Australians – Choose Health: Be Active which can aid GPs in making recommendations for their patients aged 65 years and over. [http://www.health.gov.au/internet/main/publishing.nsf/Content/3244D38BBEBED284CA257BF001FA1A7/\\$File/choosehealth-brochure.pdf](http://www.health.gov.au/internet/main/publishing.nsf/Content/3244D38BBEBED284CA257BF001FA1A7/$File/choosehealth-brochure.pdf)

### REFERENCES

1. HEART Online. HEART Online risk and symptom management. Available at: [www.heartonline.org.au/CDPR/risk-and-symptom-management/Pages/default.aspx](http://www.heartonline.org.au/CDPR/risk-and-symptom-management/Pages/default.aspx) [Accessed 15 August 2014].
2. National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand. Reducing risk in heart disease: An expert guide to clinical practice for secondary prevention of coronary heart disease. Canberra: NHF, 2012. Available at [www.heartfoundation.org.au/SiteCollectionDocuments/Reducing-risk-in-heart-disease.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/Reducing-risk-in-heart-disease.pdf) [Accessed 20 October 2014].
3. Kodama S, Tanaka S, Saito K, et al. Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol. *Arch Intern Med* 2007;167:999–1008.
4. McCann, A. Antiplatelet therapy after coronary occlusion. *Aust Presc* 2007;30:92–96.
5. Rossi S, editor. Ischaemic stroke and transition ischaemic attack. Secondary prevention. Australian Medicines Handbook 2014. In: Australian Medicines Handbook 2014. Adelaide: Australian Medicines Handbook Pty Ltd; 2014.
6. Cardiovascular Expert Group. Atrial fibrillation. Anticoagulation in the long term. (revised February 2012). In: eTG Complete [Internet] Melbourne. Therapeutics Guidelines. Ltd. 2014. Available at [www.tg.org.au](http://www.tg.org.au) [Accessed 22 September 2014].
7. A randomized trial of propranolol in patients with acute myocardial infarction. I. Mortality results. *JAMA* 1982;247:1707–14.
8. A randomized trial of propranolol in patients with acute myocardial infarction. II. Morbidity results. *JAMA* 1983;250:2814–19.
9. Freemantle N, Cleland J, Young P, Mason J, Harrison J. Beta Blockade after myocardial infarction: systematic review and meta regression analysis. *BMJ* 1999;318:1730–37.
10. Gheorghiadu M, Goldstein S. Beta-blockers in the post-myocardial infarction patient. *Circulation* 2002;106:394–98.
11. Steg PG, James SK, Atar D, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J* 2012;33:2569–19.
12. Royal Australian College of General Practice. Guidelines for preventive activities in general practice. 8th edn. East Melbourne: RACGP, 2013. Available at [www.racgp.org.au/your-practice/guidelines/redbook](http://www.racgp.org.au/your-practice/guidelines/redbook) [Accessed 15 September 2014].
13. Kubina N, Kelly J, Symington F. Navigating self management: a practical approach to implementation in Australian health care agencies. Melbourne: Whitehorse Division of General Practice, 2007. Available at [www.risen.org.au/CDSM/Docs/Navigating\\_self\\_management%20March%202008.pdf](http://www.risen.org.au/CDSM/Docs/Navigating_self_management%20March%202008.pdf) [Accessed 15 September 2014].
14. National Health and Medical Research Council. Summary guide for the management of overweight and obesity in Primary Care. Melbourne: NHMRC, 2013. Available at [www.nhmrc.gov.au/\\_files\\_nhmrc/publications/attachments/n57b\\_obesity\\_guidelines\\_summary\\_guide\\_131219.pdf](http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n57b_obesity_guidelines_summary_guide_131219.pdf) [Accessed 22 September 2014].
15. Colquhoun DM, Bunker SJ, Clarke DM, et al. Screening, referral and treatment for depression in patients with coronary heart disease: a consensus statement from the National Heart Foundation of Australia. *Med J Aust* 2013;198:483–84.
16. National Heart Foundation of Australia. Depression in patients with coronary heart disease: a practical tool for screening your patients. Canberra: NHF, 2013. Available at [www.heartfoundation.org.au/SiteCollectionDocuments/Depression-screening-support-tool.PDF](http://www.heartfoundation.org.au/SiteCollectionDocuments/Depression-screening-support-tool.PDF) [Accessed 17 October 2014].
17. NPS MedicineWise. Home medicines review. Sydney: NPS, 2012. Available at [www.nps.org.au/topics/how-to-be-medicinewise/managing-your-medicines/home-medicine-review](http://www.nps.org.au/topics/how-to-be-medicinewise/managing-your-medicines/home-medicine-review) [Accessed 15 September 2014].
18. National Heart Foundation of Australia. Improving adherence in cardiovascular care: a toolkit for health professionals. Canberra: NHF, 2011. Available at [www.heartfoundation.org.au/SiteCollectionDocuments/FAT-WEB\\_20130420.pdf](http://www.heartfoundation.org.au/SiteCollectionDocuments/FAT-WEB_20130420.pdf) [Accessed 15 September 2014].
19. NPS MedicineWise. Management of Hypertension – factsheet. Sydney: NPS, 2010. Available at [www.nps.org.au/\\_\\_data/assets/pdf\\_file/0011/111422/Clinical\\_guidance\\_Final\\_hypertension.pdf](http://www.nps.org.au/__data/assets/pdf_file/0011/111422/Clinical_guidance_Final_hypertension.pdf) [Accessed 15 September 2014].

**Preventive health (Activity ID: 14017)**

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 <http://gplearning.racgp.org.au>
- log into the *gplearning* website at <http://gplearning.racgp.org.au> and answer the following 10 multiple choice questions (MCQs) online
- 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 <http://gplearning.racgp.org.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.**

**CASE 1 – JAKE AND JOSH**

Jake and Josh are twins aged 20 years. Josh works on the family farm, which consists of livestock and crops. Josh will take over the farm as their father transitions into retirement. Jake is studying law and plans to live and work in the city. He has no plans to return to farm work or being involved in animal husbandry.

**QUESTION 1**

Which statement is correct regarding immunisation prophylaxis for Jake and Josh?

- Both Jake and Josh require the same immunisations.
- Neither Jake nor Josh requires immunisation prophylaxis to minimise occupational risks.
- Jake should be immunised for Q fever.
- Neither Jake nor Josh would benefit from an annual influenza vaccination.
- Josh should be immunised for Q fever.

**FURTHER INFORMATION**

The boys' father had a heart attack 1 year later at the age of 55 years, followed by a minor stroke and coronary artery bypass graft surgery. He retired early. Jake presents with symptoms suggestive of hay fever/allergy. His symptoms

worsened over the harvest period and have improved but have not resolved. The boys' mother, aged 58 years, has diagnosed allergies and was recently diagnosed with type 2 diabetes. Jake drinks beer most nights and smokes 20 cigarettes a day.

**QUESTION 2**

Which statement outlines the best management approach(s) for Jake?

- He should be encouraged to stop smoking.
- He may benefit from allergy testing/desensitisation.
- He should have cardiovascular screening appropriate for his age group and family history.
- He should have metabolic screening appropriate for his age group and family history.
- Answers A–D outline appropriate management approaches.

**CASE 2 – JULIANNE**

Julianne is 23 years of age and recently become sexually active. She presents to discuss contraception and cervical screening.

**QUESTION 3**

Which of the following statements regarding cervical screening is correct?

- The age at which Julianne became sexually active will determine whether a Pap smear is warranted.
- Pap smears are recommended annually for all women.
- Pap smears are recommended every 2 years for all women.
- Pap smears are recommended every 2 years for any woman who has ever had sex.
- Julianne does not require a Pap smear.

**CASE 3 – ANDREA**

Andrea is 46 years of age and presents complaining of longstanding vulval itching. Her problem was diagnosed as thrush by another GP. Despite multiple treatments with prescription and over-the-counter medications over the past year, the itching persists.

**QUESTION 4**

Which of the following statements regarding causes of vulval itch is correct?

- Lichen sclerosus and lichen planus do not cause vulval itching.
- Vulval intraepithelial neoplasia (VIN) and squamous cell carcinoma (SCC) are not differential diagnoses.
- Vulvovaginal candidiasis is still the most likely cause of Andrea's vulval itching.
- A number of dermatological causes could account for Andrea's presentation.
- Chlamydia is not a differential diagnosis.

**CASE 4 – COPD**

James, aged 43 years presents with a severe cold. He has a history of asthma, hypertension, hypercholesterolaemia and depression, and is on several medications. He currently smokes two packets of cigarettes a day. After taking a history and performing an examination you are concerned that he may have chronic obstructive pulmonary disease (COPD).

**QUESTION 5**

Which of the following statements is correct with regards to screening and diagnosis of asthma and chronic obstructive pulmonary disease (COPD)?

- Routine asthma screening using spirometry is recommended for asymptomatic individuals.
- Routine COPD screening using spirometry is recommended for asymptomatic individuals.
- Spirometry should only be undertaken in those with symptoms suggestive of asthma or COPD.
- Screening for the symptoms of COPD should be undertaken every 2 years in all smokers.
- Screening for the symptoms of COPD should be undertaken in all ex-smokers.

**CASE 5 – BILLY**

Billy, a new patient, is an Aboriginal and Torres Strait Islander man aged 21 years. He has come to see you about a cold that he cannot shake off. He wonders if giving up smoking might help.

**QUESTION 6**

Which of the statements below regarding preventive health activities for Billy is the most correct?

- Billy should be advised to have a pneumococcal vaccine (23vPPV).
- Opportunistic screening for gambling behaviours and problems could be undertaken at this visit.
- Billy should be provided with information, counselling and referral for smoking cessation.
- Answers A, B and C are correct.
- Answers B and C are correct but answer A is incorrect.

**CASE 6 – RICHARD**

Richard, 39 year of age, is a labourer who has no known cardiovascular disease (CVD). He is a smoker and a drinker. His most recent blood pressure reading was 149/81 mmHg, total cholesterol was 6.1 mmol/L and his high-density lipoprotein was 1.7 mmol/L.

**QUESTION 7**

Which of the following statements is correct regarding his absolute CVD risk?

- His absolute CVD risk can be calculated using the information presented.
- He does not meet the age criteria to have his absolute CVD risk calculated.
- His risk is low.
- He risk is moderate.
- He risk is high.

**FURTHER INFORMATION**

Richard presents again 10 years later, aged 49 years. You now calculate his absolute CVD risk, which is found to be 21%.

**QUESTION 8**

Which statement correctly identifies the implications of his score?

- He has approximately a one in five chance of having a cardiovascular event in the next 5 years.
- He has approximately a one in five chance of having a cardiovascular event in the next 10 years.
- He has approximately a one in five chance of having a cardiovascular event in the next 15 years.
- Richard is at moderate risk of having a cardiovascular event.
- Changes to Richard's lifestyle would make no difference to his cardiovascular risk.

**CASE 7 – HEATHER**

Heather, aged 79 years old, has been your patient for 37 years. She was recently discharged from hospital following a myocardial infarct (MI) and was advised to see you. She had several medication changes and additions.

**QUESTION 9**

Which statement regarding her management at this visit is correct?

- You can safely assume that cardiac rehabilitation has been organised.
- Heather is not eligible for a home medication review.
- Heather does not require additional educational information provide to her.
- You should review her discharge summary and update her electronic health record, noting her diagnosis and any medication changes .
- You should calculate her absolute CVD risk.

**FURTHER INFORMATION**

During her hospital stay Heather had a drug eluting stent implanted and had changes made to her medications.

**QUESTION 10**

Which statement below correctly outlines medications that Heather would be expected to be taking on discharge for secondary prevention?

- Aspirin, a statin, an angiotensin converting enzyme inhibitor (ACEI), a beta-blocker and a short-acting nitrate.
- Aspirin, clopidogrel, a statin, an ACEI or angiotensin II receptor blocker (ARB), a beta-blocker and a short-acting nitrate.
- Warfarin, an ACEI or ARB, a beta-blocker and a short-acting nitrate.
- Warfarin, a statin, an ACEI, a beta-blocker and a short-acting nitrate.
- Antiplatelet agents, an anticoagulant, a statin, an ACEI and a short-acting nitrate.

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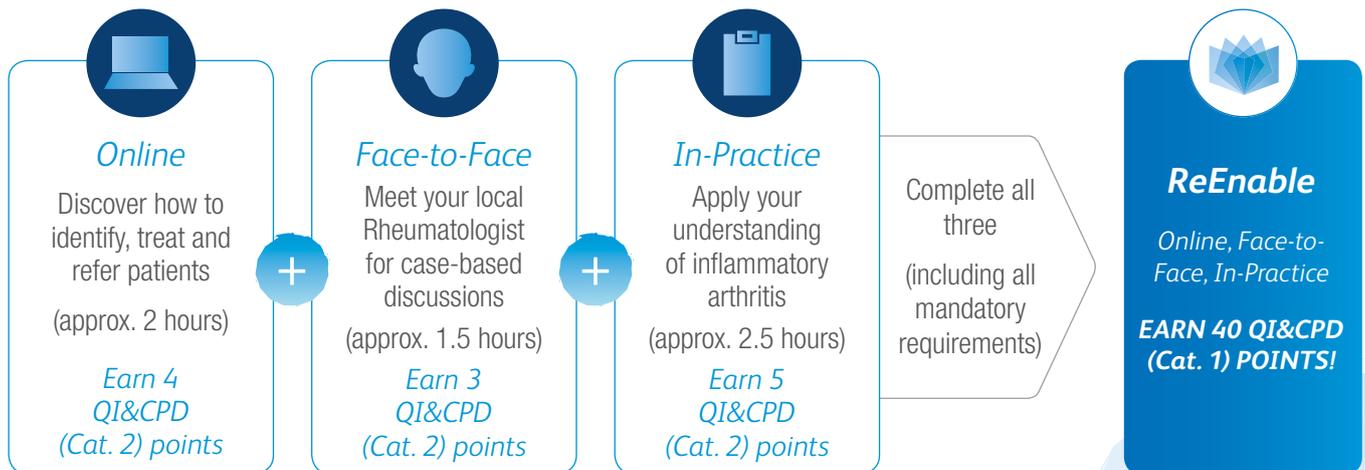


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