The Practitioner®

CPD study pack

January 2019;263(1822)

The study pack contains this month's CPD exercise plus all relevant articles:

- Diagnosing and treating mood disorders in older people
- Managing arrhythmias in coronary artery disease
- Investigating the underlying cause of erectile dysfunction

Fillable PDF templates*

- CPD exercise is a fillable PDF

CPD prompt and framework templates

- Journal article
- Case review
- Audit

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Practitioner CPD exercise

This Adobe fillable PDF form captures your answers and reflections to save in your personal development plan

Show impact
- Collection of data
- Practice audit to show improved care
- Case studies
- Implement a protocol
- Prescribing review
- Educational meetings
- Significant event audit
- Practice meeting
- Implement a new service
- Take on new role
- Significant event audits
- Change with feedback from patients and/or colleagues

Further options for sessional GPs
- Self audit of own practice:
  - Record-keeping
  - Referrals for investigations
  - Prospective condition based reviews
  - Random case analysis with colleagues
  - Review of telephone triage outcomes
- Update drugs in emergency bag

CPD Framework

Online all articles are associated with CPD prompts/frameworks to written reflection:

Adobe fillable PDFs
- Article review framework
- Case review framework
- Audit framework

Word documents
- Reflection on a journal article
- Stages of reflection on a situation
- Team reflection

MOOD DISORDERS IN OLDER PEOPLE

1 Which of the statements are true?

A Older people presenting with symptoms of depression should be assessed in the same way as younger people
True ☐ **False** ☐

B Depression and dementia have common aetiological causes and risk factors
True ☐ **False** ☐

C Mixed affective symptoms such as agitation or irritability are more common in younger than older people
True ☐ **False** ☐

D Depression is associated with poor outcomes for physical illness
True ☐ **False** ☐

2 Complete the statement.

Depression is not a normal part of

and, even if there are understandable

, this should not deter GPs from

(two words). (one word)

intervention is recommended, as a

duration of untreated illness improves the likelihood of

3 Older people with depression may not present with low mood as the predominant symptom. Give five examples of symptoms that may be potential indicators of depression in older patients.

4 List any changes to your clinical practice that you may make having read this article.

ARRHYTHMIAS IN CORONARY ARTERY DISEASE (CAD)

5 Which of the statements are true?

A Presyncope is a red flag symptom in patients with CAD
True ☐ **False** ☐

B A rate control strategy is recommended in patients with infrequent episodes of atrial fibrillation (AF)
True ☐ **False** ☐

C Beta-blockers are indicated for all patients with premature ventricular complexes in primary care
True ☐ **False** ☐

D Patients with NYHA class IV heart failure are less likely to benefit from an implantable cardioverter defibrillator
True ☐ **False** ☐

6 What are the three key elements in the management of atrial fibrillation?

»

CME

Time credits= Impact credits

7 Complete the statement.
In patients with severe LV impairment symptoms of

(two words)

or

should raise strong clinical suspicions of

(two words)

and urgent

(two words)

is required.

8 List any changes to your clinical practice that you may make having read this article.

ERECTILE DYSFUNCTION (ED)

9 Which of the statements are true?

A ED is associated with an increased risk of future cardiovascular events including after sexual activity
True ☐ **False** ☐

B Men presenting with lower urinary tract symptoms should be specifically questioned about ED
True ☐ **False** ☐

C Men with high CVD risk should be referred to a cardiologist before considering any treatment other than lifestyle adaptations
True ☐ **False** ☐

D PDE-5 inhibitors are contraindicated in patients with untreated hypotension or hypertension
True ☐ **False** ☐

10 List three anatomical abnormalities that can cause ED.

11 Complete the statement.
It is important to look for signs of hypogonadism such as

or loss of

(two words)

and examine the external genitalia for any

or

deformity.

12 List any changes to your clinical practice that you may make having read this article.

Answers

MOOD DISORDERS IN OLDER PEOPLE
1 **A** True **B** True **C** False **D** True **E** True

2 Ageing; triggers; offering treatment. Early; short; response

3 Physical symptoms; apathy; poor motivation; cognitive symptoms; agitation; retardation; fatigue; weight loss

ARRHYTHMIAS IN CORONARY ARTERY DISEASE (CAD)
5 **A** True **B** True **C** False (only for those with particularly bothersome symptoms of skipped or extra beats) **D** True

6 Thromboembolic risk assessment and stroke prevention; control of symptoms through either rate or rhythm control; management of associated comorbidities and risk factors

7 Rapid palpitations; syncope; ventricular arrhythmias; specialist assessment

ERECTILE DYSFUNCTION (ED)
9 **A** True **B** True **C** True **D** True

10 Peyronie's disease; post priapism; foreskin abnormalities

11 Gynaecomastia; body hair; penile; testicular

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Diagnosing and treating mood disorders in older people

Stageman W, Hay K, Allan CL. Diagnosing and treating mood disorders in older people.
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Diagnosing and treating mood disorders in older people

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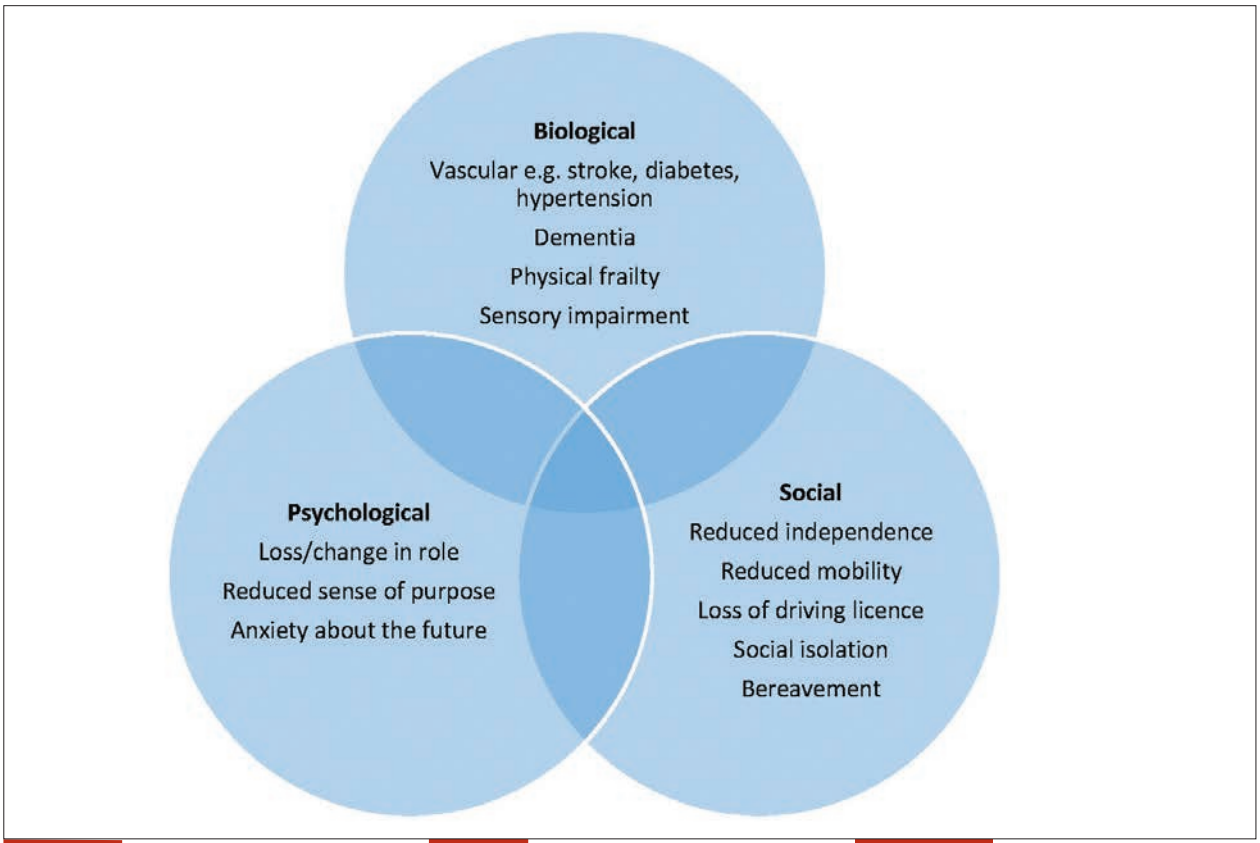

FIGURE 1 Biopsychosocial risk factors for late life depression

What are the main risk factors?

DEPRESSION IN OLDER ADULTS IS COMMON, WITH CLINICALLY SIGNIFICANT DEPRESSIVE SYMPTOMS affecting 15% of people over the age of 55,[1] and up to a third of people experiencing some depressive symptoms.[2] The prevalence increases in those over the age of 85 years in hospitals and in nursing homes.[1]

Depressive symptoms may be part of a recurrent depressive disorder, or experienced for the first time in later life as a result of changes in risk and resilience factors.

Depression may also be seen in the context of bipolar disorder; reviewing past history carefully and enquiring about past symptoms of mania is important. Bipolar disorder rarely presents for the first time in older adults[3] and a presentation of elevated mood and symptoms of mania should prompt investigations to exclude organic pathology and delirium.

RISK FACTORS

Risk factors for late life depression can be classified using a biopsychosocial framework. These risks are closely related and frequently overlap (see figure 1, above).

Cerebrovascular changes are common as people age and are associated with increased risk of depressive symptoms and depression.[4] Neuroimaging studies have repeatedly shown that cerebrovascular pathology, including white matter hyperintensities, can predispose, precipitate or perpetuate a depressive syndrome in the elderly.[5] Individuals with late life depression have greater white matter hyperintensities on structural imaging, and these are associated with poor outcomes.[6,7]

Diabetes mellitus and hypertension are particularly important risk factors for these end-organ changes in the brain.[8,9] Vascular risk factors not only increase the risk of depression, but also the risk of cognitive changes and dementia. There is a bidirectional relationship between depression and dementia, with common aetiological causes and shared risk factors.[1]

Physical illnesses, particularly those conditions associated with frailty, are important risk factors for depression. Conditions such as chronic or severe pain, Parkinson's disease and COPD, may have direct effects on the brain, or indirect effects on important psychosocial risk factors such as lack »

How should older patients be assessed?

Which patients should be referred?

of sleep, reduced mobility and social isolation. Additionally, the psychological and social effects of sensory impairment, particularly visual and hearing loss, can reduce communication and impact on wellbeing. Hearing loss can lead to low self-esteem, isolation, loneliness and depression.[10]

The ageing process requires ongoing psychological adaptation to changes in everyday life with alteration in roles and responsibilities (e.g. through retirement) and physical morbidity. Psychological adaptation to ageing, particularly when it is associated with loss of externally validating roles, can reduce an individual's sense of purpose and self-esteem.

There may be anxieties or fears over the future in relation to loss of independence, practicalities of obtaining support, financial pressures, or the dying process itself. Psychological adaptation may be harder for those who have historically responded poorly to stress or who have a history of multiple stressful life events.

As people age, illness is more common among their peers. The death of friends or family members (especially the loss of a spouse or partner) is a risk factor for depression.

Living alone and a dwindling social circle through death of friends or family or relocation can increase social isolation and loneliness. This may be compounded by reduced mobility and independence caused by musculoskeletal problems or stopping driving.

Changing needs of peers may require individuals to take on new caring roles, and while these may provide a greater sense of purpose, they can also be a source of stress contributing to risk for depression.

Bipolar disorder has been known to present for the first time as late as 86 years of age; risk factors are physical illness, cognitive impairment and previous depression.[11]

PRESENTATION

Identifying depression in an older patient may be a challenge for a number of reasons. It has a distinct presentation in late life and low mood may not be the predominant presenting symptom. Indeed, when asked directly, the older person may deny that they feel sad or low in mood.

Furthermore, the cognitive decline that can accompany depression may often be misdiagnosed as dementia.[12] It is therefore important to recognise other symptoms as potential indicators of depression in the older person.

The older person with depression may present with:[13]

- Physical symptoms
- Apathy
- Poor motivation
- Cognitive symptoms
- Agitation
- Retardation
- Fatigue
- Weight loss

These symptoms may trigger concerns of an underlying physical illness, however, the clinician should always consider depression as a cause of such symptoms in an older person. A recent study suggested that apathy symptoms may identify patients who are at increased risk of dementia.[14]

'A cognitive assessment should be completed in older people presenting with a possible mood disorder'

A further challenge in identifying depression in older people is posed when the patient has established cognitive impairment. A number of studies have found that depression is frequently comorbid with dementia, particularly vascular dementia, so it is essential to consider depression as a potential cause of any new symptoms in such patients.

Although less common, older people may also present with manic symptoms later in life. Mixed affective symptoms such as agitation or irritability are more common in older people than in those who present at a younger age. Understandably, this could be misdiagnosed as dementia in an older person. As such, in any older person presenting with a change in behaviour which seems atypical of dementia, mania should be considered a potential diagnosis. It is worth bearing in mind that the older person may also present with the typical manic symptoms seen in younger people such as elevated mood, poor sleep and risky and disinhibited behaviours.

Comorbidity

There is a correlation between low mood and:

- Chronic pain
- Any chronic physical illness
- Stroke
- Parkinson's disease
- Cognitive impairment (both as a cause and a symptom of depression)

Furthermore:

- Persistent severe depression is linked to the onset of dementia[15,16]
- There is a link between depression and worse outcomes for physical illness[17]
- There is a link between depression and increased mortality[17]

It is therefore essential to identify depression quickly and effectively, and to treat it adequately. If a patient has multiple comorbidities, a referral to a geriatrician may be helpful as a means of holistically approaching the patient's presentation, and in turn, this will be beneficial to the patient's mood.

ASSESSMENT

When an older person presents with possible symptoms of depression, the assessment should be similar to that of any younger person. A symptom check as per diagnostic criteria should be completed, bearing in mind the aforementioned predominance of biological symptoms in older people. The assessing clinician should also be vigilant for self-neglect and psychotic symptoms. Specific rating scales such as the Geriatric Depression Scale (GDS) are available to assess depressive symptoms in older people. The GDS is preferred to other commonly used tools such as the Patient Health Questionnaire-9 (PHQ-9), as it avoids questions about somatic symptoms (which in older people may be attributable to other causes). It is worth noting that the GDS is not a diagnostic tool but may be helpful in establishing severity of depressive symptoms.

A risk assessment in this group of people is vital as it will inform any management plan. The degree of mood disturbance may be impinging on the older person's functional ability, and as such, the individual may pose a risk to self and potentially others around the home. It is important to remember that many older people continue to drive and their ability and judgment may be impaired in this area.

Assessment of suicidality is essential: older people are far less likely to articulate their suicidal thoughts than younger people. The main risk factors for suicide in older people are social isolation and depression and they are more likely to use violent methods. It is worth bearing in mind that self-harm without underlying suicidal intent is rare in older people, therefore, any attempt at self-harm or suicide should be taken extremely seriously.

A cognitive assessment should also

be completed in older people presenting with a possible mood disorder. If there is a significant degree of impairment, information from a family member or another person may be helpful. On cognitive testing, you may find that a patient with an underlying dementia lacks insight into their memory loss, whereas an older person with a primary mood disorder maintains this insight. Another possible finding on cognitive testing may be preservation of higher function such as problem solving and speech but reduced concentration.

In older people, a medication review is essential as there are a number of drugs that may have been prescribed for comorbidities which may be contributing to their mood disturbance. Depression is a recognised side effect of certain beta-blockers, montelukast, corticosteroids and also proton pump inhibitors. Mania in older people should not be ignored and may indeed present later in life, with steroids being a relatively common cause of manic presentations.

If a patient presents with mood disturbance, either elevated or low mood, blood tests may help direct management should a contributory medical factor be identified. Suggested tests include:

- Full blood count
- Renal profile
- Thyroid function
- Vitamin B_{12} and folate
- Glucose

These tests may help identify potential causes of mood disturbance, as well as possible physical causes of somatic symptoms.

MANAGEMENT

Depression is not a normal part of ageing and, even if there are understandable triggers, this should not deter GPs from offering treatment. Indeed, early intervention is recommended, as a short duration of untreated illness improves the likelihood of response.[18] Management involves a stepped care approach.

For persistent subthreshold symptoms, or mild to moderate depression, low intensity psychological or psychosocial interventions should be considered. These could include sleep hygiene advice, behavioural activation, CBT, structured exercise, referral to a »

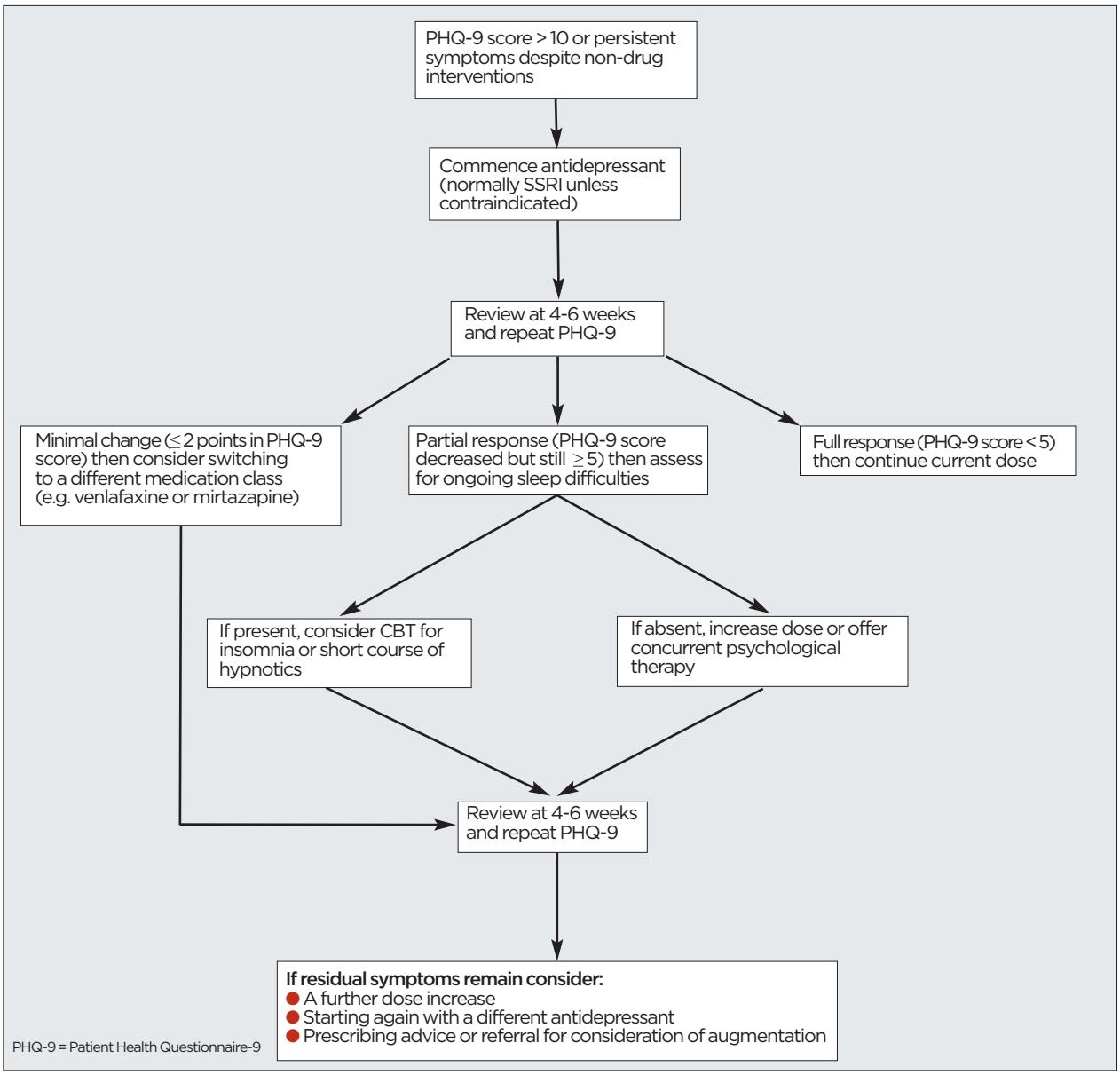

FIGURE 2
An example of a systematic approach to using pharmacological intervention in depression; adapted from ABC of Depression and Anxiety[29]

key points

SELECTED BY

Dr Phillip Bland
Former GP, Dalton-in-Furness, UK

Depression in older adults is common. Depressive symptoms may be part of a recurrent depressive disorder or experienced for the first time in later life as a result of changes in risk and resilience factors. Bipolar disorder rarely presents for the first time in older adults and a presentation of elevated mood and symptoms of mania should prompt investigations to exclude organic pathology and delirium.

Risk factors for late life depression can be classified using a biopsychosocial framework. There is an association between cerebrovascular pathology, vascular risk factors and depression. Physical illnesses, particularly those associated with frailty, are also important risk factors for depression. Conditions such as chronic or severe pain, Parkinson's disease and COPD may have direct effects on the brain, or indirect effects on important psychosocial risk factors such as lack of sleep, reduced mobility and social isolation. Visual or hearing loss can lead to low self-esteem, isolation, loneliness and depression.

Depression has a distinct presentation in late life and low mood may not be the predominant presenting symptom. Indeed, when asked directly, older patients may deny that they feel sad or low in mood. They may present with physical symptoms, apathy, cognitive symptoms, agitation, retardation, fatigue or weight loss. Cognitive decline associated with depression is often misdiagnosed as dementia. Moreover, depression is frequently comorbid with dementia, particularly vascular dementia, so it is essential to consider depression as a potential cause of any new symptoms in such patients.

Assessment of an older person presenting with possible symptoms of depression should be similar to that of any younger person. Assessment of suicidality is essential: older people are far less likely to articulate their suicidal thoughts. The main risk factors for suicide in older people are social isolation and depression. An assessment of cognition and a medication review should be carried out: depression is a recognised side effect of certain beta-blockers, montelukast, corticosteroids and PPIs. Blood tests include: FBC, renal profile, thyroid function tests, glucose, B_{12} and folate.

For persistent subthreshold symptoms, or mild to moderate depression, low intensity psychological or psychosocial interventions should be considered. These could include sleep hygiene advice, behavioural activation, CBT, structured exercise, referral to a befriending service or social prescribing. If symptoms persist or depression is moderate to severe, pharmacological or high intensity psychological therapies should be considered. SSRIs should generally be offered first line; sertraline and citalopram are often preferred because of the lower risk of interactions.

Referral to specialist mental health services may be indicated if there is: risk of suicide, risk of self-neglect, poor response to treatment, significant psychiatric comorbidity, psychotic symptoms, diagnostic difficulty (commonly differentiation between depression and early cognitive impairment) or new onset or undertreated bipolar disorder.

befriending service (such as one of those offered by Age UK, see Useful information box, p15) or social prescribing if available.[19] If symptoms persist or there is a moderate to severe burden, pharmacological or high intensity psychological therapies should be considered, see figure 2, p13.

When using antidepressant medication, assessing response systematically and utilising rating scales (such as the PHQ-9) have both been shown to reduce time to remission significantly as this prompts timely dose or medication change in those who fail to respond.[20,21]

SSRIs should generally be offered first line; sertraline and citalopram are often preferred because of their lower propensity for interactions. Be mindful that the risk of hyponatraemia with SSRIs increases with age and concurrent NSAID use. Combining antidepressants with NSAIDs raises the risk of bleeding in susceptible individuals or those on warfarin, and this includes the risk of brain haemorrhage.[22,23] Mirtazapine could be considered as an alternative.

Patients should be reassessed at four weeks after commencing medication. If there has been minimal change (2 points or less on PHQ-9), switching to a different class of antidepressant could be trialled. If there has been a partial response (PHQ-9 score decreased but still > 5) a dose increase could be considered. If there are ongoing sleep symptoms CBT for insomnia or a short course of hypnotics may help.

If residual symptoms remain despite dose optimisation of an antidepressant to which there was a partial response, augmentation could be considered, but this usually requires consultation with a psychiatrist.

The addition of lithium, quetiapine, aripiprazole and mirtazapine are common augmentation strategies. A systematic review of treatment resistant depression in older people found an overall response rate of 58% to antipsychotic augmentation and a 42% response rate to lithium.[24] Although not licenced, there is a growing body of evidence that aripiprazole is both an effective and well tolerated augmentation strategy in older people.[25] To reduce the risk of relapse, the medication(s) on which the individual achieved remission should be continued for at least a year in a first episode of depression, at least two years in a second, and perhaps indefinitely after a third.[26]

In those with moderate or severe depression and chronic physical health or complex needs collaborative care has been shown to be effective.[27] A care coordinator helps improve compliance with treatment and improves joint working between physical and mental health services.

In bipolar disorder treatment follows the same principles as for younger adults, although very few studies have been directed at older people. Generally, older people are more susceptible to adverse reactions, owing to end-organ sensitivity and impairment in both circulation and hepatic and renal clearance. This may be especially pertinent with lithium. Treatment doses are therefore generally lower than those used in younger adults and should be more carefully titrated.[28] The risk of switching to mania when using antidepressants without mood stabilisers is high in older people with bipolar depression and this should be avoided.

REFERRAL

Most patients with depressive disorders are treated in primary care. Referral to specialist mental health services may be indicated in the following cases:

- Risk of suicide
- Risk of self-neglect
- Poor response to treatment
- Significant psychiatric comorbidity
- Psychotic symptoms
- Diagnostic difficulty (commonly differentiation between depression and early cognitive impairment)
- New onset or undertreated bipolar disorder

Competing interests: None

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Useful information

MindEd
Educational resource on mental health
www.minded.org.uk

Royal College of Psychiatrists
Patient information leaflets
www.rcpsych.ac.uk/mental-health

Age UK Befriending services
www.ageuk.org.uk/services/befriending-services

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Managing arrhythmias in coronary artery disease

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FIGURE 1
Patient event monitor recording of atrial fibrillation during symptoms of palpitations

What are the common arrhythmias?

How should patients be investigated?

What are the management strategies?

CORONARY ARTERY DISEASE (CAD) IS THE LEADING CAUSE OF DEATH IN THE UK ACCOUNTING for approximately 15% of male deaths and 10% of female deaths. It also causes significant morbidity.[1] The broad condition of CAD encompasses presentation with acute coronary syndromes (ACS) and chronic stable disease. Important associated arrhythmias include atrial fibrillation (AF) and atrial flutter, ventricular tachycardia (VT) and ventricular fibrillation (VF), ventricular ectopy, and bradyarrhythmias. Arrhythmias occurring in hospital following admission with an ACS are associated with significant morbidity and mortality and will require immediate evaluation.[2,3]

Patients with a prior myocardial infarction (MI) or stable chronic coronary disease remain at risk of cardiac arrhythmias and particular importance must be attached to the care of those with impaired left ventricular systolic function and thus assessment of left ventricular function informs many management decisions. It is these groups that are of particular relevance to the primary care setting.

CLINICAL ASSESSMENT

Table 1, p18, outlines the key aspects of assessment common to all patients with CAD and suspected arrhythmias. A detailed history is vital to elucidate red flag symptoms necessitating urgent specialist assessment. Palpitations are common. However, patients often describe them in different ways and they can include irregular sensations in AF, rapid and regular palpitations of atrial flutter or VT, and skipped beats typical of ventricular ectopics. In AF, symptoms may be nonspecific and manifest as dyspnoea or fatigue.

Red flag symptoms in patients with CAD include syncope and presyncope, particularly in patients with concomitant left ventricular systolic dysfunction in whom rapid palpitations should raise strong suspicions for VT. Palpitations with severe chest pain and breathlessness also warrant urgent assessment.

Clinical examination will focus on the cardiovascular system particularly looking for signs of heart failure and murmurs suggestive of heart valve disease.

However, patients with CAD are often older with multiple comorbidities. A holistic approach is crucial to detecting and managing accompanying conditions that often precipitate arrhythmias including hypertension, obesity, diabetes mellitus, obstructive airways disease and thyroid dysfunction. Key aspects of clinical investigations and management are highlighted in reference to specific arrhythmias below.

ATRIAL FIBRILLATION

Assessment

AF is associated with increased mortality particularly due to heart failure and sudden death.[4,5] Early detection and management is therefore crucial. Undiagnosed AF is common, particularly in older populations, which overlap significantly with those with CAD. Although there are no specific studies in patients purely with CAD, broader studies of screening in older populations appear cost effective and use a combination of pulse palpation and single lead ECG.[6] The European Society of Cardiology guidelines recommend opportunistic screening using these methods in patients over 65 years of age, which encompasses many patients with CAD.[2]

The management of AF consists of three essential components:

- Thromboembolic risk assessment and stroke prevention
- Control of symptoms through either rate or rhythm control
- Management of associated comorbidities and risk factors

If an irregular pulse is detected, a 12-lead ECG should be carried out to confirm and document the presence of AF as well as identify the ventricular rate. Radial palpation should not be relied upon for assessment of ventricular rate. It is important to note that high ventricular rates will often result in an »

element of ST segment depression and this alone does not signify significant ischaemia. Moreover, it may cause a small elevation in cardiac troponins, even in people with no coronary atheroma. However, if ongoing angina is suspected then referral for repeated ischaemia evaluation may be required.[2,3]

Antithrombotic therapy

Thromboembolic risk should be assessed in all patients using the CHA_2DS_2VASc score (see table 2, opposite). Bleeding risk should also be assessed with particular emphasis on detecting and correcting modifiable risk factors (see table 2, opposite).

All patients with CAD will have a CHA_2DS_2VASc score of at least 1 and oral anticoagulation should therefore be considered.[2] Although both vitamin K antagonists (VKA) and direct oral anticoagulants (DOAC) are effective in stroke risk reduction, evidence suggests superior efficacy of DOACs particularly through lower risk of haemorrhagic stroke. Bleeding risk is also lower with DOACs and should therefore be considered first line.[2,7]

Importantly, patients with CAD are also prescribed antiplatelet agents. Combination therapy with anticoagulation significantly increases bleeding risk, particularly in the case of triple therapy with dual antiplatelet agents and oral anticoagulation where risk is increased by 79-134%.[8,9]

In patients with stable CAD without an ACS or percutaneous coronary intervention (PCI) in the preceding 12 months then oral anticoagulation alone should be used. In the context of ACS or recent (preceding 12 months) PCI then decisions on combination therapy should be undertaken in collaboration with the interventional cardiology team. A period of one to six months of triple therapy may be indicated, influenced by the specific stents used, coronary anatomy and patient specific bleeding risk. Dual

Table 1

Assessment of patients with suspected arrhythmias

History

Symptoms
- Palpitations
- Dyspnoea
- Chest pain
- Peripheral oedema
- Presyncope/syncope
- Exercise tolerance

Important comorbidities/ past medical history
- Hypertension
- Heart failure
- Valve disease
- Angina/ACS/PCI
- Thyroid dysfunction
- Obesity
- Diabetes mellitus
- Obstructive sleep apnoea
- Chronic airways disease
- Chronic kidney disease

Medications
- Anticoagulants
- Antiplatelets
- NSAIDs
- Heart failure drugs
- Antihypertensives

Social history
- Alcohol
- Smoking
- Exercise
- Occupation and driving

Examination

General
- Height, weight/BMI
- Oxygen saturations

Cardiovascular
- Pulse palpation
- BP
- JVP
- Murmurs
- Pulmonary congestion/ pleural effusions
- Peripheral oedema

Respiratory
- Chest hyperexpansion
- Expiratory wheeze

Thyroid
- Tremor
- Thyroid goitre

Investigations

Test/investigation	Indications/important considerations of tests
Blood tests: ● Full blood count ● Renal function/electrolytes ● Liver function ● Thyroid function	● Identify anaemia ● Guide drug dosing ● Inform bleeding risk ● Identify reversible causes
12-lead ECG	● Confirm diagnosis and assess ventricular rate ● Identify signs of ischaemia ● Identify conduction disease ● Identify signs of structural heart disease (e.g. BBB)
Ambulatory monitoring ● Continuous ECG monitoring ● External event recorders Including Zio patch and Kardia AliveCor device ● Implantable loop recorders	● Important to detect AF paroxysms ● Assess rate control ● Identify and assess burden of PVCs or NSVT ● Continuous monitoring of limited duration
Transthoracic echocardiography	● Assess LV systolic function ● Identify valve disease ● LVH as sign of hypertension ● LA size

ACS = acute coronary syndrome; BBB = bundle branch block; BMI = body mass index; BP = blood pressure; JVP = jugular venous pressure; LA = left atrium; LV = left ventricle; LVH = left ventricular hypertrophy; PCI = percutaneous coronary intervention

therapy with oral anticoagulation and clopidogrel should then be continued up to 12 months followed by anticoagulation alone. Prasugrel or ticagrelor are avoided in combination with anticoagulation given their increased risk of bleeding but DOACs are generally recommended in place of warfarin because of their lower bleeding risk.[2,10]

Rate vs rhythm control

Although there are no specific trials in patients with CAD, there is no clear evidence that a rhythm control strategy offers any significant long-term benefits.[11,12] The appropriate strategy is therefore determined primarily by the symptomatic burden of AF. In patients with well tolerated or infrequent episodes a rate control strategy is recommended. In those with paroxysmal AF a detailed correlation of contemporaneously recorded symptoms and arrhythmias during ambulatory recording is key, and in those with persistent AF cardioversion to assess the symptomatic benefit of a period of sinus rhythm may be required.

Management options for rate or rhythm control are outlined in table 3, p20. Rate control is based on slowing AV nodal conduction. As beta-blockers are indicated for many patients with CAD, particularly following an ACS, this should be considered first line. The appropriate initial target heart rate should be < 110. Evidence suggests that this lenient target, compared with a strict target of < 80 is equally effective and significantly easier to achieve.[13]

Importantly, although flecainide is commonly prescribed for patients with paroxysmal AF it is contraindicated in patients with CAD because of an increased risk of sudden death.[14] When clinically suspected, CAD should be excluded prior to commencing flecainide.

Patients with AF and heart failure with left ventricular systolic dysfunction should be referred to secondary care. AF may be the primary cause of heart failure often as a result of tachycardia mediated cardiomyopathy. This can be difficult to establish and is confirmed by demonstrating improved ejection fraction following a period of good rate control or sinus rhythm. In this setting, a rhythm control strategy is recommended. Calcium channel blockers are contraindicated. Beta-blockers and digoxin are used for rate control while amiodarone is used for rhythm control.

Studies have not specifically evaluated patients with CAD, but catheter ablation is increasingly used as part of a rhythm control strategy in patients refractory to pharmacological management and is effective in reducing AF recurrence.[2,15]

The recent CASTLE-AF trial evaluated catheter ablation compared with pharmacological therapy (rate or rhythm control) in patients with AF and heart failure with reduced ejection fraction. Nearly half, 46%, of patients had an ischaemic aetiology and catheter ablation was shown to result in significantly reduced rates of death or hospitalisation for heart failure. Catheter ablation should therefore particularly be considered in patients with severe symptoms and/or LV dysfunction.[16]

Risk factor modification

Risk factor modification is a crucial facet of CAD management and should include blood pressure control and lifestyle adjustments such as weight loss and exercise, which have been demonstrated to result in a significant reduction in AF burden and symptoms, as well as positive cardiac remodelling.[17] Studies of beta-adrenoceptor antagonists or renin-angiotensin-aldosterone system inhibitors have demonstrated reduced incidence of AF.[18]

Drugs in these classes are also indicated to reduce mortality following MI and in patients with left ventricular systolic dysfunction, and will therefore also be indicated in many patients with chronic CAD.[19]

Post coronary artery bypass grafting

AF occurs in up to 50% of patients following coronary artery bypass grafting (CABG). However, it is often self-limiting within the first 4-6 weeks.[10] Rhythm control is generally recommended in the early postoperative period and amiodarone is the most frequently used antiarrhythmic drug in this setting. This should be reviewed within six weeks of hospital discharge and can usually be stopped at this stage if sinus rhythm has been maintained. Ongoing arrhythmias should be managed with specialist input. There are no clear guidelines on anticoagulation in this patient group and decisions should be taken on a case by case basis considering ongoing bleeding and thromboembolic risk.[3]

ATRIAL FLUTTER

Atrial flutter is an organised atrial arrhythmia. The most common form observed in patients with CAD involves a band of tissue between the inferior vena cava and tricuspid annulus termed the cavotricuspid isthmus (CTI). This is identified by typical 'saw tooth' flutter waves visible in the inferior ECG leads. Catheter ablation involves creation of a linear lesion through the CTI thereby interrupting the reentry circuit and is both safe and effective.[20] Catheter ablation is therefore widely considered as the first-line treatment option. Importantly, atrial flutter and fibrillation often coexist and thromboembolic risk is considered similar. The same »

Table 2

Managing thromboembolic risk

Thromboembolic risk

CHA_2DS_2VASc	Score
Congestive heart failure/ LV systolic dysfunction	+1
Hypertension	+1
Age 75 or older	+2
Diabetes mellitus	+1
Prior **S**troke, TIA or systemic embolism	+2
Vascular disease (MI, peripheral arterial disease or aortic plaque)	+1
Age 65-74 years	+1
Sex: female	+1

Bleeding risk

Modifiable factors	Non-modifiable factors
● Hypertension	● Age
● Labile INR (if on VKA)	● Previous major bleeding
● Antiplatelet or NSAID use	● Stroke
● Excess alcohol	● Dialysis dependent renal disease or transplant
● Impaired renal function	● Cirrhotic liver disease
● Impaired liver function	● Malignancy
● Thrombocytopaenia	

Recommendations according to CHA_2DS_2VASc
0 (male) **1** (female) – Anticoagulation not recommended
1 (male) **2** (female) – Consider anticoagulation
2 (male) **3** (female) – Recommend anticoagulation

recommendations for antithrombotic therapy should therefore be followed.

Specific patient groups, particularly those who have previously undergone cardiac surgery or catheter ablation are susceptible to different forms of atrial flutter arising from both the left and right atria. These patients require specialist assessment.

However, the broad principles of initial management are the same for patients with any atrial flutter or AF and involve assessment of haemodynamic compromise, control of ventricular rate and stroke prevention.

VENTRICULAR ECTOPY

Premature ventricular complexes (PVC) and non-sustained VT (NSVT) are frequently observed in patients with an ACS, particularly during revascularisation for STEMI. However, PVCs are also frequently observed in primary care and are therefore commonly seen in patients with stable CAD. In this context reassurance is usually all that is required.

In patients with particularly bothersome symptoms of skipped or extra beats then pharmacological therapy with beta-blockers may be indicated. In patients with a very high burden of ectopy, severe symptoms or associated presyncope or syncope, and when associated with heart failure or significant ECG abnormalities (e.g. abnormal QRS morphology or bundle branch block) specialist assessment should be sought.

VENTRICULAR ARRHYTHMIAS

Ventricular arrhythmias in the context of CAD include monomorphic and polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF). These will often result in significant haemodynamic compromise or cardiac arrest resulting in sudden death. In the case of resuscitated cardiac arrest or sustained VT, management is undertaken in secondary care and may involve any, or a combination, of revascularisation, antiarrhythmic drug therapy, catheter ablation and device implantation. For patients in primary care, management should focus on identifying those at greatest risk of sudden death who will benefit from an implantable cardioverter defibrillator (ICD).

Primary prevention

Left ventricular ejection fraction (LVEF) $\leq$ 35% increases the risk of sudden death. All patients with CAD should therefore undergo assessment of LVEF usually by transthoracic echocardiography. This is particularly important at least six weeks following presentation with ST-elevation ACS.[19]

Recommendations for ICD implantation are derived from chronic heart failure guidelines and overlap with

Table 3

Management strategies for rate or rhythm control of atrial fibrillation (AF)

Therapy	Indication	Special considerations
Flecainide	**Contraindicated** in patients with CAD	Increased risk of sudden death
Beta-blockers	Rate control Indicated post ACS and in heart failure	Considered first line
Calcium channel blockers (diltiazem and verapamil)	Rate control	Contraindicated in heart failure with reduced ejection fraction. Avoid combination of verapamil and beta-blocker (risks severe bradycardia or AV block)
Digoxin	Rate control	Limited efficacy during exercise
Amiodarone	Maintenance of sinus rhythm	Requires careful counselling and checking/monitoring of TFTs, LFTs and lung function. Titrate to lowest effective dose
Sotalol	Maintenance of sinus rhythm	Confers risk of arrhythmia and sudden death Avoid in heart failure with reduced ejection fraction
Dronedarone	Maintenance of sinus rhythm (third line to amiodarone and sotalol)	Avoid in heart failure with reduced ejection fraction Requires close LFT monitoring. Avoid if AF becomes persistent
Catheter ablation	Maintenance of sinus rhythm: — Severe drug refractory symptoms — AF mediated LV dysfunction	Carries significant risk of complications and patient selection important to confer most benefit
AV node ablation and pacemaker implantation	Patients with severely symptomatic drug refractory AF unsuitable for/declined AF ablation Patients with heart failure and CRT in order to optimise biventricular pacing	May be useful in patients with ischaemia and symptoms related to difficult rate control

Table 4

ICD/CRT treatment options for heart failure with ejection fraction ≤ 35%, adapted from NICE TA134[21]

QRS duration (ms)	NYHA class I	NYHA class II	NYHA class III	NYHA class IV
< 120	ICD if there is a high risk of sudden cardiac death			ICD and CRT not indicated
120-149 (without LBBB)	ICD	ICD	ICD	CRT-P
120-149 (with LBBB)	ICD	CRT-D	CRT-P or CRT-D	CRT-P
≥ 150 (with or without LBBB)	CRT-D	CRT-D	CRT-P or CRT-D	CRT-P

ICD = implantable cardioverter defibrillator; CRT-D = cardiac resynchronisation therapy with ICD; CRT-P = cardiac resynchronisation therapy with pacing

recommendations for cardiac resynchronisation therapy (CRT).

QRS duration and morphology has been demonstrated to identify those who will benefit from CRT with those with QRS >150 ms and left bundle branch block most likely to benefit. However, left ventricular systolic dysfunction and heart failure are also associated with high rates of non-sudden cardiac death. Patients with severe heart failure (NYHA class IV) have a particularly poor prognosis and are therefore less likely to benefit from ICD implantation.

Table 4, above, outlines guidelines for cardiac device implantation but it should also be stressed that optimisation of medical therapy including renin-angiotensin-aldosterone inhibitors, angiotensin-receptor-neprilysin inhibitors and beta-blockers is of paramount importance and confers significant reduction in mortality.[19,21]

Secondary prevention

Patients with sustained ventricular arrhythmias require emergency assessment and secondary care management. In patients with severe LV impairment symptoms of rapid palpitations or syncope should raise strong clinical suspicions of ventricular arrhythmias and urgent specialist assessment is required.

The majority of patients with CAD who experience ventricular arrhythmias will require ICD implantation. A proportion of patients with an ICD will require antiarrhythmic drug therapy or catheter ablation to reduce the frequency of ICD shocks.

Those with sustained monomorphic VT without haemodynamic compromise and preserved LV systolic function may be managed with antiarrhythmic drug therapy or catheter ablation alone without requiring ICD implantation.[22]

Table 5

When to refer patients with arrhythmias

Arrhythmia	When to refer
Atrial fibrillation/flutter	● Symptomatic arrhythmia in whom rhythm control considered ● Symptoms of severe chest pain and/or dyspnoea ● Concomitant LV systolic impairment (new or preexisting) ● Guidance on appropriate antiplatelet combination therapy required ● New onset atrial flutter ● Ongoing arrhythmia six weeks post CABG
Ventricular arrhythmias/ heart failure	● Presyncope, syncope or rapid palpitations with known LV impairment ● Symptoms/signs of heart failure in patient with previous ACS ● New marked ECG abnormalities (e.g. new BBB)
Ventricular ectopy	● Severe symptoms or associated syncope ● High burden on ambulatory monitoring (> 10%) ● Associated LV systolic impairment ● New marked ECG abnormalities (e.g. new BBB)
Bradyarrhythmia	● Symptomatic sinus bradycardia despite reduction/cessation of rate limiting medications ● Second- or third-degree AV block

BRADYARRHYTHMIAS

Bradycardia secondary to advanced atrioventricular (AV) conduction block may occur in the context of STEMI. However, bradycardia in patients with stable CAD is most frequently related to rate limiting drugs such as beta-blockers resulting in sinus bradycardia with or without PR prolongation. In the absence of symptoms no intervention is required. The observation of second- or third-degree AV block requires specialist assessment.

CONCLUSION

Arrhythmias, particularly AF and VT/VF with associated risk of sudden death are common in patients with CAD. Identification of patients with AF is important particularly to institute appropriate anticoagulation for stroke prevention. Further management involving either rate control or rhythm control is determined primarily by patient symptoms as well as the effect of AF on LV systolic function and must incorporate a holistic approach to treat concomitant conditions and lifestyle factors. LVEF should be assessed in all patients with CAD to identify those at risk of sudden cardiac death who may benefit from ICD »

key points

SELECTED BY

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A detailed history is essential in patients with coronary artery disease (CAD) to elucidate red flag symptoms necessitating urgent specialist assessment. Red flags include syncope and presyncope, particularly in patients with concomitant left ventricular systolic dysfunction. Palpitations with severe chest pain and breathlessness also warrant urgent assessment.

Undiagnosed atrial fibrillation (AF) is common in older populations. In patients with stable CAD without an acute coronary syndrome (ACS) or percutaneous coronary intervention (PCI) in the preceding 12 months oral anticoagulation alone should be used. In the context of ACS or recent (preceding 12 months) PCI decisions on combination therapy should be undertaken in collaboration with the interventional cardiology team. Triple therapy for 1-6 months may be indicated, influenced by the specific stents used, coronary anatomy and patient specific bleeding risk. Dual therapy with oral anticoagulation and clopidogrel should then be continued up to 12 months followed by anticoagulation alone.

There is no clear evidence that a rhythm control strategy offers any significant long-term benefits in AF. The appropriate strategy is therefore determined primarily by the symptomatic burden of AF. Rate control is based on slowing AV nodal conduction. As beta-blockers are indicated for many patients with CAD, particularly following an ACS, this should be considered first line. The initial target heart rate should be < 110. Flecainide is commonly prescribed for paroxysmal AF. It is contraindicated in patients with CAD because of an increased risk of sudden death. Catheter ablation should therefore particularly be considered in patients with severe symptoms and/or LV dysfunction. AF occurs in up to 50% of patients post CABG, but is often self-limiting within the first 4-6 weeks.

Premature ventricular complexes are also frequently observed in primary care and are therefore commonly seen in patients with stable CAD. In this context reassurance is usually all that is required. In patients with particularly bothersome symptoms of skipped or extra beats then beta-blockers may be indicated.

Left ventricular ejection fraction (LVEF) ≤35% is a predictor of increased risk of sudden death. All patients with CAD should therefore undergo assessment of LVEF, usually by transthoracic echocardiography. This is particularly important at least 6 weeks following presentation with ST-elevation ACS. Most patients with CAD who experience ventricular arrhythmias will require an ICD.

Bradycardia in patients with stable CAD is most frequently related to rate limiting drugs such as beta-blockers resulting in sinus bradycardia with or without PR prolongation. In the absence of symptoms no intervention is required. The observation of second- or third-degree AV block requires specialist assessment.

implantation for primary prevention.

Evidence in this area is continually evolving. Screening for AF is likely to develop further and trials to identify the optimal anticoagulation strategies in combination with antiplatelet agents are currently ongoing. Risk stratification for sudden death remains challenging and current interest in the use of cardiac MRI and scar assessment may prove advantageous.

Competing interests: None

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Investigating the underlying cause of erectile dysfunction

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SPECIAL REPORT

Investigating the underlying cause of erectile dysfunction

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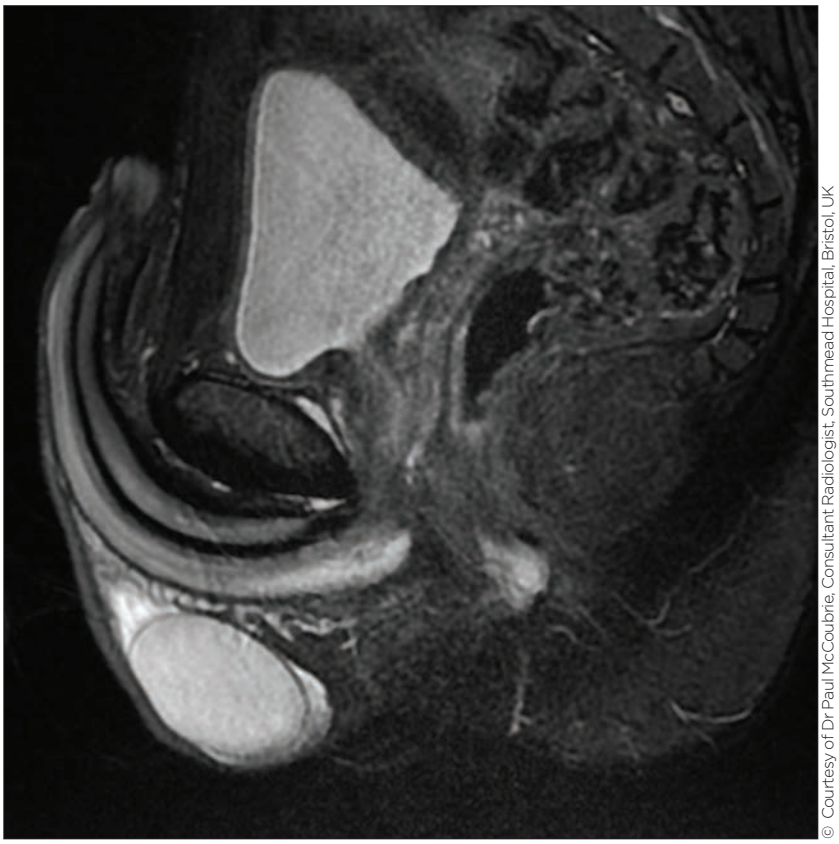

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FIGURE
MRI penis

How should patients be assessed?

What are the management approaches?

Which patients should be referred?

ERECTILE DYSFUNCTION (ED) IS THE PERSISTENT INABILITY TO ACHIEVE AND MAINTAIN AN ERECTION sufficient to permit satisfactory sexual performance. The term 'impotence' may also be used to describe ED.

ED has several possible organic and/or psychogenic causes. Common associations with ED are detailed in table 1, p24.[1] Rates of ED increase with age, with an estimated 70% of men aged 70 affected to some extent.[2]

Men may attend primary care with ED as a main presenting complaint or mention it during a consultation for unrelated concerns. Although increased awareness has destigmatised talking about ED, men can still feel embarrassed about discussing this complaint. A sensitive and nonjudgmental approach is essential to draw out important factors during history taking.

ED has been shown to be a significant predictor of underlying cardiovascular disease (CVD) which requires further investigation and as such ED should not be regarded as a condition which just impacts on quality of life.[3] All men should routinely be asked about ED during any assessment of cardiovascular risk. Men with established diagnoses of CVD, diabetes or lower urinary tract symptoms (LUTS) should be specifically questioned about ED.[4]

ASSESSMENT

A thorough history is essential to identify the most likely underlying ED aetiology and inform further investigations and management strategy. In addition to the onset and duration of symptoms important points in the history to clarify are:

- How does the patient define their ED? First clarify that the patient is actually complaining of an erection problem (sometimes men may use the term incorrectly) and ask the patient to quantify the loss of rigidity. Is there any ejaculatory or orgasmic dysfunction?
- Has the patient ever had erectile function?
- Are morning erections preserved or lost?
- Is the ED related to all sexual encounters or only in specific circumstances?
- Does the man have a partner and has there been any recent change in partner or behaviour?

Men should be asked about symptoms of hypogonadism such as loss of libido or low energy levels in addition to questions regarding their lifestyle. A brief assessment of exercise routines and capacity as well as a medication history may give an insight into the man's cardiovascular risk status.

However, we would recommend that all men presenting with ED should have their cardiovascular risk status formally evaluated using the QRISK prediction tool. ED has been shown to be an independent risk factor for CVD, »

Table 1

Potential causes of erectile dysfunction and associated underlying diseases of erectile dysfunction[6]

Vascular	Coronary artery disease Peripheral arterial disease Hypertension Hyperlipidaemia
Neurological	Post pelvic surgery or radiotherapy Peripheral neuropathy Parkinson's disease Multiple sclerosis Alzheimer's disease Spinal cord injury Autonomic neuropathy
Endocrine	Diabetes mellitus Metabolic syndrome Low testosterone (primary or secondary hypogonadism) Hypothyroidism/hyperthyroidism Cushing's syndrome
Drug induced	Antihypertensives Antidepressants Antipsychotics Antiandrogens Recreational drug use Anabolic steroids Alcohol
Psychosexual	Generalised Situational
Anatomical abnormalities	Peyronie's disease Post priapism Foreskin abnormalities

Table 2

Cardiac risk stratification, adapted from the European Association of Urology guidelines[6]

Low risk	Intermediate risk	High risk
Asymptomatic, < 3 risk factors for CAD (excluding sex)	≥ 3 risk factors for CAD (excluding sex)	High-risk arrhythmias
Mild, stable angina (evaluated and/or being treated)	Moderate, stable angina	Unstable or refractory angina
Uncomplicated previous MI	Recent MI (> 2, < 6 weeks)	Recent MI (< 2 weeks)
LVD/CHF (NYHA class I or II)	LVD/CHF (NYHA class III)	LVD/CHF (NYHA class IV)
Following successful coronary revascularisation	Non-cardiac sequelae of atherosclerotic disease (e.g. stroke, peripheral vascular disease)	Hypertrophic obstructive and other cardiomyopathies
Controlled hypertension		Uncontrolled hypertension
Mild valvular disease		Moderate to severe valvular disease

Key: CAD = coronary artery disease; CHF = congestive heart failure; LVD = left ventricular dysfunction; MI = myocardial infarction; NYHA = New York Heart Association

associated with a 25% increased risk of CVD. Consequently, ED has been included as a clinical variable in the most recent version of QRISK (QRISK 3), a validated algorithm to identify those at risk of heart disease and stroke.[5]

Other validated questionnaires such as the International Index of Erectile Function (IIEF) or Shorter Sexual Health Inventory for Men (SHIM) may facilitate history taking and establish a baseline against which responses to treatment can be assessed.[4,7]

It is important to look for signs of hypogonadism such as gynaecomastia or loss of body hair and examine the external genitalia for any penile or testicular deformity. Examining lower limb pulses may also highlight evidence of peripheral vascular disease.

Population-based studies have previously demonstrated an association between LUTS and sexual dysfunction.[8] If LUTS are elicited during the patient's history a digital rectal examination (DRE) should be performed as part of the man's assessment. Current NICE guidance also recommends performing a DRE in men with a history of prostate cancer and in men over 50 years of age.[6,9]

BMI, heart rate and blood pressure should be recorded for all patients, and if indicated by the history a cardiovascular or neurological examination may be performed.

Although the clinical history forms the bedrock of assessment in men with ED, further investigations in the form of blood tests may be appropriate. The European Association of Urology (EAU) guidelines recommend that initial blood tests include an HbA_{1c} or fasting glucose and lipid profile (if not recorded in the past 12 months). Early morning total testosterone levels should be measured in all patients.[6]

The Endocrine Society recommends that optimally clinicians investigating hypogonadism should measure fasting total testosterone concentrations on two separate mornings to confirm a low testosterone diagnosis. This is because testosterone concentrations may be subject to diurnal variations and can be suppressed by food or glucose intake.[10] If total fasting testosterone levels are low then repeat with FSH, LH and prolactin levels included.[9]

A PSA test is only indicated during the diagnostic work up of ED: if the DRE is abnormal or the man is over 50 years old and after counselling wishes to have a PSA test having weighed up the benefits and risks of the test, or if he is considering testosterone replacement

Table 3

Pharmacokinetics of PDE-5 inhibitors - adapted from NICE guidance[9]

	Sildenafil	Tadalafil	Vardenafil	Avanafil
Time taken before sexual activity	1 hour	At least 30 minutes	25–60 minutes	15–30 minutes
Duration of action	4–5 hours	Up to 36 hours	4–5 hours	Up to 6 hours
Effect of food intake	Rate of absorption reduced by 60 minutes when taken with food	None	Rate of absorption reduced by 60 minutes when taken with high-fat meals	Rate of absorption reduced by 75 minutes when taken with high-fat meals

therapy.[4] Any additional laboratory tests should be performed only if indicated, guided by clinical findings. It is worth considering carrying out a resting ECG in men with suspected CVD risk factors.

MANAGEMENT

ED is associated with an increased risk of future cardiovascular events including after sexual activity, even in men without cardiovascular symptoms at presentation.[11] The EAU guidelines recommend cardiac risk stratification at diagnosis in men seeking treatment.[6] Men with high CVD risk should be referred to a cardiologist for a review before considering any treatment other than lifestyle adaptations (see table 2, opposite).

All men with ED should be advised to review their lifestyle and improve their health; weight loss, smoking cessation, increasing exercise and reducing alcohol consumption are examples of factors which should be optimised.

Any reversible causes of ED should also be addressed. [6] If there is a clear drug-related cause consider the risk versus benefit to the patient's perceived quality of life and seek an alternative therapy, if appropriate, after shared decision making.

REFERRAL

The following groups of men should be referred directly for specialist management in secondary care after their ED diagnosis:

- Men with psychosexual dysfunction should be referred for psychosexual counselling, men with severe distress or a suspected underlying psychogenic cause may need to be referred to their local mental health services team[9]
- Men with hypogonadism should be referred to the secondary care endocrinology team for assessment and consideration of testosterone replacement[9]
- Men with severe or unstable CVD that would make sexual activity unsafe should be referred to cardiology[9]
- Young men who have always had ED, men who suffer ED after priapism (a persistent and painful erection lasting more than four hours) or ED as a consequence of trauma to the penis, genitals or spine should be referred to urology[9]

For men not referred directly to specialist services phosphodiesterase type-5 (PDE-5) inhibitors form the mainstay of oral treatment and can be started in the community. It is important to tell men prescribed PDE-5 inhibitors that sexual stimulation is necessary to enable an erection.

Common PDE-5 inhibitors and their time interval to efficacy are described in table 3, above. A trial period of six weeks is recommended to assess efficacy.[12] All men should be followed up after starting treatment as approximately 35% of patients may fail first-line treatment. Most commonly this is due to a lack of patient education regarding correct usage. If the first PDE-5 inhibitor is not effective at maximum dosage a change to one of the other PDE-5 inhibitors would be appropriate for a further six-week trial with counselling regarding usage.[12] It is important to check that patients are not taking versions of PDE-5 inhibitors that are sourced from the internet from non pharmacologically approved sources.

Commonly recognised side effects of PDE-5 inhibitors are headaches, flushing, indigestion, nasal congestion, blue vision and back pain.[9] Absolute contraindications and cautions for PDE-5 inhibitors are listed in table 4, above. This list is not exhaustive and prescribing information should be reviewed before administration.

Men with ED that has not responded to PDE-5 inhibitors should be referred for specialist review as further treatment options are available for those who wish to pursue them. The specialist expertise to deliver this service may be present within community facilities or secondary care depending on regional networks, competencies and protocols.

Vacuum erection devices, are a non pharmacological treatment option which may enable erections sufficient for intercourse in motivated patients and are an alternative first-line option in men in whom PDE-5 inhibitors are contraindicated or the elderly when clinicians are concerned about potential side effects or drug interactions.[6] »

Table 4

Generic contraindications and cautions for PDE-5 inhibitor use[6]

Contraindications	Cautions
● MI/stroke/arrhythmia in past 6 months ● Untreated hypotension or hypertension ● Unstable angina OR angina on sexual intercourse OR NYHA IV heart failure ● Concurrent use of nitrates ● Non anterior ischaemic neuropathy	● Adjust dose if used with an alpha-blocker ● Predisposition to priapism e.g. sickle cell disease, myeloma, leukaemia ● Anatomical penile deformity ● Active peptic ulcer ● Left ventricular outflow obstruction ● Vardenafil in elderly men

This list is not exhaustive and prescribing information should be reviewed before administration

key points

SELECTED BY

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Erectile dysfunction (ED) is the persistent inability to achieve and maintain an erection sufficient to permit satisfactory sexual performance. Rates of ED increase with age, with an estimated 70% of men aged 70 affected to some extent. ED is a significant predictor of underlying cardiovascular disease and is included in QRISK3.

In addition to the onset and duration of symptoms, important points in the history to clarify are: how the patient defines their ED, whether they have ever had erectile function, whether morning erections are preserved, the context of ED and recent sexual history. Men should be asked about symptoms of hypogonadism such as loss of libido or low energy levels. Cardiovascular risk should be evaluated.

GPs should look for signs of hypogonadism and examine the external genitalia for any penile or testicular deformity. Examination of lower limb pulses may provide evidence of peripheral vascular disease. If LUTS are elicited during the patient's history a digital rectal examination (DRE) should be performed as part of the man's assessment. NICE guidance also recommends performing a DRE in men with a history of prostate cancer and in men over 50 years of age. BMI, heart rate and BP should be recorded for all patients, and if indicated by the history a cardiovascular or neurological examination may be performed. Initial blood tests should include HbA_{1c} or fasting glucose, a lipid profile and fasting early morning total testosterone.

Patients should be stratified into cardiovascular risk categories. Those at high risk should be referred to cardiology before considering any treatment other than lifestyle adaptations. Men with suspected hypogonadism should be referred to endocrinology and counselling should be considered for men with psychosexual dysfunction. Medications known to cause ED should be reviewed and alternative therapy offered if appropriate. Lifestyle advice should include weight loss, smoking cessation, increased exercise and reduced alcohol intake.

Phosphodiesterase type-5 (PDE-5) inhibitors are first-line treatment and a trial period of 6 weeks is recommended to assess efficacy. Treatment failure is most commonly due to patients not being educated regarding correct usage. If the first PDE-5 inhibitor is not effective at maximum dosage a change to one of the other PDE-5 inhibitors would be appropriate for a further 6-week trial.

Men with ED that has not responded to PDE-5 inhibitors should be referred to a specialist. Vacuum erection devices are an option when PDE-5 inhibitors are ineffective or contraindicated, and in elderly patients where there are concerns about potential side effects or drug interactions. Unless there is a clear reversible organic or psychological cause, men with treatment-refractory ED should be referred to a urologist specialising in andrology, as should those with associated structural penile abnormalities such as Peyronie's disease and all those who develop ED after priapism or genital trauma.

Intracavernosal injections describe treatments for ED where vasoactive drugs are injected directly into the corpus cavernosum to increase blood flow into the penis and enable an erection. Alprostadil is currently the most common formulation used for this purpose. Patients started on this treatment (and if appropriate their partners) are taught how to prepare and administer the injection. Intracavernosal alprostadil is a highly efficacious treatment for ED.[13]

Men considering the treatment should be fully informed regarding potential complications, which include: penile pain, prolonged erections, priapism and penile fibrosis at injection sites. Penile pain is usually self-limiting after prolonged use but local anaesthetic can be considered to alleviate the pain from injections. Systemic side effects from intracavernosal injections are rare. Intracavernosal injections are contraindicated in men with bleeding disorders, a history of priapism or known hypersensitivity to alprostadil.[14]

Alprostadil may also be administered as either intraurethral or topical preparations. While such preparations are associated with lower rates of efficacy they offer reasonably well tolerated slightly less invasive alternatives.

The urology secondary care team have access to a full range of ED treatment options, including specialist expertise in whether combining treatments is appropriate and if necessary access to penile prostheses.

Men with ED refractory to treatment or complicated by concerns regarding structural abnormalities such as Peyronie's disease should always be referred to a urologist with a special interest in andrology.

Competing interests: None

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Useful information

British Association of Urological Surgeons
www.baus.org.uk/professionals/sections/erectile_dysfunction.aspx

NICE. Clinical Knowledge Summaries
https://cks.nice.org.uk/erectile-dysfunction#!scenario

British Society for Sexual Medicine Guidelines on the Management of Erectile Dysfunction in Men-2017
https://www.jsm.jsexmed.org/article/S1743-6095(18)30077-8/fulltext

We welcome your feedback

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CPD prompt and framework
Journal article

The Practitioner

Show impact
- Collection of data
- Practice audit to show improved care

- Case studies
- Implement a protocol
- Prescribing review
- Educational meetings
- Significant event audit
- Practice meeting
- Implement a new service
- Take on new role
- Significant event audits
- Change with feedback from patients and/or colleagues

Further options for sessional GPs
- Self audit of own practice:
  - Record-keeping
  - Referrals for investigations
  - Prospective condition based reviews
  - Random case analysis with colleagues
  - Review of telephone triage outcomes
- Update drugs in emergency bag

Time credits

Impact credits
Quick impact

Later impact

Later impact=

Total credits

Title and reference

Date

What have you learned?

How will this change your practice?

What research/actions will you take that will have further impact on practice?

Draft of your action plan

CPD prompt and framework
Case review

The Practitioner

Clinical case for review

Reason for selection of the case for reflection

Evidence base used to reflect on your practice

What went well?

What could have been done better?

What were the causes of the condition/event?

What learning needs arise from this case for (a) you and (b) for the practice?

Summary of any discussion of the case with peers

What changes/actions have resulted to your personal practice?

What changes/actions have resulted to the practice?

How/when will you review these changes?

Time credits	Impact credits	Total credits

CPD prompt and framework
Audit

The Practitioner

Title

Reason for the audit (target to improve or implement)

Define criterion (or criteria) to be met

Set the standard and its basis

Plan the audit process and any involvement of peers

Time credits **Impact credits** **Total credits**

Reflection on Table 1 results

What changes were implemented to improve results?

Reflection on Table 2 results and review with peers

Table 1

Results of data 1 analysis **Date of data collection:**

Criterion	Number sampled	Achievement (%)	Standard (%)
A			
B			
C			

Table 2

Results of data 1 and data 2 analyses **Date of data collection:**

Criterion	Number sampled Data 1	Achievement (%) Data 1	Number sampled Data 2	Achievement (%) Data 2	Standard (%)
A					
B					
C					