7
Cardiovascular Problems
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Hypertension

**GUIDELINE**


- Hypertension is one of the most common conditions treated in primary care in the United Kingdom and one of the most important preventable causes of death worldwide (Krause et al., 2011). It is the main risk factor for development of stroke and ischaemic heart disease and is strongly associated with the development of chronic kidney disease and cognitive impairment.
- *Hypertension,* defined as the presence of persistently raised blood pressure at or greater than 140/90 mm Hg (Box 7.1), affects more than a quarter of all UK adults and over half of those aged 65 years or more (Health and Social Care Information Centre, 2011). The adult prevalence of high blood pressure is even higher in other parts of the world, with over 40% of adults affected in Africa, for example (World Health Organisation [WHO], n.d.).
- Most people with hypertension have no symptoms or clinical findings on examination and their hypertension is identified incidentally or as a result of complications such as angina, myocardial infarction (MI), stroke, and arrhythmias.
- The rule of halves was described in the United States by Wilber and Barrow in 1972. They stated that half of hypertensives are not known to have a raised blood pressure (BP); of those with known hypertension, half are not on treatment and half of those on treatment are poorly controlled. The figures for detection and treatment of hypertension have improved in recent years, but this remains a useful reminder of the challenge posed by hypertension.

**BOX 7.1 Definition of Hypertension**

### Stage 1 Hypertension
- Clinic BP ≥140/90 mm Hg and subsequent ABPM or HBPM ≥135/85 mm Hg

### Stage 2 Hypertension
- Clinic BP ≥160/100 mm Hg and subsequent ABPM or HBPM ≥150/95 mm Hg

### Severe Hypertension
- Clinic BP ≥180/110 mm Hg

Note: Where a threshold or target level of blood pressure is given (e.g., 160/100 mm Hg), it means that action should be taken if the systolic is 160 or over or the diastolic is 100 or over.

ABPM, Ambulatory blood pressure monitoring; HBPM, home blood pressure monitoring.


**Detection**

- All adults should have their blood pressure measured at least every 5 years up to the age of 80 and at least annually thereafter (Hodgkinson et al., 2011).
- The 2011 NICE guidelines recommended a major shift in how blood pressure measurements are taken and hypertension diagnosed, centering on the use of ambulatory (ABPM) and home (HBPM) blood pressure monitoring to complement clinic measurements (Box 7.2). This is in part a response to the overtreatment of people with so-called white coat hypertension.
- The guidelines recommend the following steps to diagnose hypertension:
  1. If a clinic BP is over 140/90 mm Hg, take a second reading in the consultation.
  2. If the second reading is very different from the first, take a third reading.
Note that in patients with type 2 diabetes, a stricter target of less than 140/80 mm Hg is the aim (<135/75 mm Hg in those with microalbuminuria).

Three points should be made about these targets:
1. Any reduction in blood pressure carries benefit, even if the target is not reached (Czernichow et al., 2011).
2. The lower the blood pressure the greater the benefit (Ettehad et al., 2016).
3. These BP targets may change in light of more recent research recommending more intensive BP lowering for high risk patients (The SPRINT Research Group, 2015)

Practicalities of Blood Pressure Measurement

1. The patient should be seated, but in older patients and in patients with diabetes check the blood pressure both standing and sitting.
2. On the first occasion measure the BP in both arms. A significant difference is found in 20% of hypertensives. If there is a difference of more than 5 mm Hg, then use the arm with the higher reading for future measurements.
3. Measure the systolic and diastolic pressures to the nearest 2 mm Hg. If over 140/90 mm Hg, repeat the measurement toward the end of the consultation. If markedly different from each other, take at least one more. Take the average. Repeated readings by a nurse give the most reliable clinic results, occasional readings by a doctor the least (Little et al., 2002).
4. Timing. In mild uncomplicated hypertension, do not start treatment until three readings have been taken over a 3-month period. About 25% of blood pressures will settle in that time. Those that settle to below treatment levels need lifelong annual follow-up. If the initial diastolic is over 200/110 mm Hg or there is evidence of end organ damage, cardiovascular disease, or diabetes, three readings over 2 weeks would be more appropriate. Consider immediate treatment if the pressure is over 220/120 mm Hg.
5. Follow-up 6 monthly, once the patient is established on treatment. It is as good as monthly.

Workup

1. Check for a history of family and personal risk factors for stroke or coronary heart disease. Check whether relevant drugs (e.g., nonsteroidal antiinflammatory drugs [NSAIDs]) or excess alcohol are taken.
2. Examination, including:
   • fundi (essential only in severe hypertension) (van den Born, Hulsman, Hoekstra, Schlingemann, & van Montfrans, 2005);
   • femoral pulses;
   • palpation of kidneys and auscultation for presence of bruit;
   • signs of left ventricular hypertrophy.
3. Urinalysis for protein and blood

Blood Pressure Targets

• The target of hypertension treatment is to reduce clinic blood pressure levels to below 140/90 mm Hg in people aged under 80, and below 150/90 mm Hg in people aged 80 and over.
• Previous guidance focused on treating those aged under 80, but more recent evidence has shown that treatment is well tolerated and reduces total mortality and cardiovascular events (Beckett et al., 2008, 2011).
4. Blood:
   • Creatinine and electrolytes
   • Fasting blood sugar
   • Serum lipids
5. Look for left ventricular hypertrophy using electrocardiogram (ECG) and chest x-ray (CXR).
6. Calculate the patient’s 10-year cardiovascular disease (CVD) risk using a recognized calculator (e.g., the QRISK2 cardiovascular disease risk calculator, available at http://www.qrisk.org/). A 20% 10-year CVD risk means that the lower threshold for treatment applies and that primary prevention of CVD is indicated (see section below on Primary Prevention).

**Nondrug Treatment**

Nondrug treatment can lower the systolic pressure by 4 to 10 mm Hg (Stevens, Obarzanek, & Cook, 2001; Writing Group of the PREMIER Collaborative Research Group, 2003). It lowers the risk of CVD and should be offered to all with hypertension, whether or not drugs are being prescribed.

Consider the following:
1. **Smoking:** Ask about smoking. If appropriate offer advice and refer to the smoking cessation services. Stopping will not reduce the BP but it will lower the cardiovascular risk.
2. **Exercise.** Physical activity lowers the risk of developing hypertension and is an effective treatment for those with established hypertension. Brisk walking for 30 minutes every day is as beneficial as more vigorous exercise three times a week. After only 2 weeks of aerobic exercise, the mean fall in blood pressure is 5/4 mm Hg (Whelton, Chin, Xin, He, 2002).
3. **Weight:** Encourage weight loss if overweight (body mass index [BMI] >25 kg/m²). Overweight is a significant and independent predictor of the level of BP (Cox et al., 1996). A 10-kg weight loss promotes a reduction of 5 to 20 mm Hg (Chobanian, 2003).
4. **Alcohol:** There is a direct dose–response relationship between alcohol intake and risk of hypertension, particularly when alcohol intake exceeds two drinks per day (Xin et al., 2001). Support patients to reduce excessive consumption.
5. **Salt:** Reduce intake of salt to less than 5.8 g/day or less than 2.4 g sodium. A 2011 Cochrane review was unable to confirm whether reducing dietary salt had significant effects on mortality or cardiovascular morbidity (Taylor, Ashton, Moxham, Hooper, & Ebrahim, 2011) but a subsequent meta-analysis found a significant reduction in cardiovascular events (He & MacGregor, 2011), supporting long-standing public health recommendations to reduce salt consumption in the population. Common sources of salt are nuts, crisps, canned foods, table sauces, and bread. Many processed foods are high in salt, so avoid those with over 1.5 g per 100 g food. Also note that many labels use sodium rather than salt content: 1 g sodium = 2.5 g salt, so more than 0.6 g sodium per 100 g food is high.

6. **Dietary Approaches to Stop Hypertension (DASH) diet:** This is a diet rich in fruit, vegetables, and oily fish and low in sodium and total and saturated fats. It has been shown in a number of trials to reduce BP by up to 11/5 mm Hg (Appel et al., 1997). It is similar to a Mediterranean-style diet (Sacks & Campos, 2010).
7. **Coffee:** Coffee is known to acutely raise BP but a 2012 systematic review and meta-analysis found no significant effect on BP or the risk of hypertension (Steffen, Kuhle, Hensrud, Erwin, & Murad, 2012). However, standard advice remains to discourage excessive coffee drinking.
8. **Contraceptive pill:** Consider stopping but not until other adequate contraceptive measures are in place.
9. **Stress:** The relationship between stress and blood pressure is not well understood, yet anecdotally patients often blame a stressful life for their hypertension. This is an area of ongoing research, with some promising results for stress reduction interventions (Hughes et al., 2013).

**Drug Management**

**Primary Prevention**

- **Aspirin:** The use of low-dose aspirin (75 mg daily) in primary prevention is controversial, with ongoing debates about benefits (reducing cardiovascular events) versus risks (bleeding events) (Barnett, Burrell, & Iheanacho, 2010). At the time of writing, aspirin is not licensed for primary prevention in the United Kingdom and should not be routinely started, even in those with risk factors such as hypertension and diabetes (Scottish Intercollegiate Guidelines Network, 2010).
- **Statins:** Give statins if the patient has CVD or diabetes or has a risk of CVD that is sufficiently high. SIGN (2017) recommends a risk threshold of CVD of at or greater than 20% in the next 10 years as an indication for the introduction of statin therapy; however, the NICE guidance from 2014 suggests consideration of primary prevention strategies in those with over a 10% risk in the next 10 years, if lifestyle measures have not proved effective.

**Antihypertensives**

- About half of patients fail to take their antihypertensives as prescribed. Patients have many reservations about drug treatment (Benson & Britten, 2002). Getting them to voice those reservations gives clinicians a chance to alter any erroneous ideas they may have. A study of black Caribbean patients in London found these common misconceptions (Connell, McKevitt, & Wolfe, 2005):
  - Once the BP was controlled, they were cured and didn’t need the medication.
  - They could sense when their BP was raised and so could judge when they needed to take the medication.
- Commonly used drugs produce a similar average fall of 9.1/5.5 mm Hg at standard doses (Law, Wald, Morris, & Jordan, 2003). Most patients with hypertension therefore need more than one antihypertensive drug.
• Combining two drugs from different classes reduces the blood pressure five times more than doubling the dose of one drug (Wald et al., 2009).
• The timing of medication may also be important. There is a morning rise in blood pressure, in keeping with circadian rhythms. A Cochrane review found that advising patients to take their antihypertensives in the evening resulted in small gains in 24-hour BP reduction (mean of 1.7±1.4 mm Hg) (Zhao, Xu, Wan, & Wang, 2011). It is unclear, however, if this translates to a reduction in CV events.
• The choice of drugs should be influenced by age, comorbidity, adverse effects, possible synergistic effects between classes of drugs, and the individual’s response to each drug. Ethnic origin also influences the choice of medication; younger white patients tend to have high levels of renin and angiotensin II; older patients and those of African origin tend to have low renin levels and so respond less well to drugs that block the renin–angiotensin system, although the differences are thought to be small.
• Follow the scheme in Table 7.1, but do not persevere with a drug that has produced no benefit (i.e., a drop in BP of <5 mm Hg). Instead, switch to a drug with a different mode of action (e.g., from A to C or D). Persevere with a drug that shows some but inadequate benefit; it may be synergistic with a drug from a different group.
• Divide drugs into those that suppress the renin system (A [ACE inhibitors and angiotensin receptor blockers]) and those that work independently of it (C [calcium channel blockers] and D [diuretics]) (Brown et al., 2003). The following steps are recommended, but tailor them to the needs of the individual patient.
• Offer step 1 treatment to people aged under 80 with stage 1 hypertension and one or more of the following:
  1. Target organ damage
  2. Established cardiovascular disease
  3. Renal disease
  4. Diabetes
  5. 10-year cardiovascular risk equivalent to 20% or more

TABLE 7.1 Drug Management in Hypertension

<table>
<thead>
<tr>
<th>Age &lt;55 and Non-Black</th>
<th>Age ≥55 or Black</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>A or C or D</td>
</tr>
<tr>
<td>Step 2</td>
<td>A and C or D</td>
</tr>
<tr>
<td>Step 3</td>
<td>A and C or D</td>
</tr>
<tr>
<td>Step 4</td>
<td>Add spironolactone, or, if not tolerated, an alpha-blocker, or a beta-blocker, or another diuretic, or refer</td>
</tr>
</tbody>
</table>

A 2012 Cochrane review of Randomised Controlled Trials (RCTs) of treatment of mild hypertension in those without preexisting cardiovascular disease found that treatment did not reduce morbidity or mortality (Diao, Wright, Cundiff, & Gueyffier, 2012).
• Offer step 1 treatment to people of any age with stage 2 hypertension.
• Offer people aged under 55 years an ACE inhibitor or a low-cost angiotensin receptor blocker (ARB). If an ACE inhibitor is prescribed and not tolerated (e.g., due to cough), offer a low-cost ARB.
• Offer people aged over 55 years and black people of African or Caribbean family origin of any age a calcium channel blocker (CCB). If a CCB is not suitable (e.g., due to oedema or intolerance), or there is evidence/ risk of heart failure, offer a thiazide-like diuretic.

Drug Treatment of Patients With Other Medical Problems

Most individuals (approximately four of five individuals in one study [Barnett et al., 2012]) with hypertension will have additional chronic diseases.
1. **Angina:** Use a beta-blocker or a calcium channel blocker.
2. **Heart failure:** Use a diuretic, an ACE inhibitor, a beta-blocker, then an alpha-blocker.
3. **Diabetes:** Use an ACE inhibitor, a low-dose thiazide, an alpha-blocker, or calcium channel blocker.
4. **Smokers:** Do not use a beta-blocker.
5. **Migraine:** Use a beta-blocker and/or calcium channel blocker, then all other alternatives.
6. **Those active in sports:** Avoid beta-blockers.
7. **Raynaud syndrome:** Use a calcium channel blocker.
8. **Renal failure:** Get specialist advice. ACE inhibitors may improve renal function but will need to be given in lower dosage.
9. **Gout:** Avoid thiazides.
10. **Asthma:** Avoid beta-blockers.

Other Points About the Drugs

**Thiazides**

- Indapamide (2.5 mg daily) or chlorthalidone (12.5–25 mg daily) has been suggested by NICE to be preferential to bendroflumethiazide.
- Recheck creatinine and electrolytes 1 month after starting a thiazide then repeat annually. Repeat more frequently if the patient is unwell or is taking digoxin or another drug that might affect renal function. A rise of creatinine of up to 30% is acceptable provided it remains less than 200 μmol/L (Martin & Coleman, 2006).
- Diabetes is not a contraindication. In the Systolic Hypertension in the Elderly Program (SHEP) study, a thiazide was associated with the development of diabetes in an extra 4.3% of patients over 4 years, but it protected those who developed diabetes against an increase in cardiovascular mortality (Kostis et al., 2005).
• Ask specifically about erectile difficulties in men. The incidence in patients on thiazides is double that in those on placebo (17% versus 8%), but patients rarely volunteer this information.

**Calcium Channel Blockers**

• Use a long-acting preparation (e.g., diltiazem 120–180 mg slow release twice a day or verapamil 120–240 mg twice a day or 240–480 mg slow release daily). Their hypotensive effect is the same as nifedipine, with fewer side effects. Use brand names. Different generic products have different bioavailabilities.

• Use a dihydropyridine calcium channel blocker (e.g., nifedipine slow release oramlodipine), if:
  1. beta-blockers are also being given;
  2. there is peripheral vascular disease with skin ischaemia; it will not, however, help intermittent claudication;
  3. there is a risk of heart failure, which might be worsened by a non-dihydropyridine.

**ACE Inhibitors and Related Drugs**

• Starting them:
  1. The first dose should be taken at night. Even then, first-dose hypotension due to once-daily agents may not occur until 6 to 8 hours after the first dose, and may last for 24 hours.
  2. Recheck serum creatinine and electrolytes 1 week after starting the drug, and after any dose increase, then annually.

• A rise of serum creatinine of less than 30% is acceptable provided it remains under 200 μmol/L (Martin & Coleman, 2006). A greater rise suggests renal artery stenosis or chronic kidney disease. Above that, reduce or stop the drug and recheck weekly till the creatinine has returned to its previous level. Look for underlying renal disease.

• A rise of serum K⁺ to 5.5 to 5.9 mmol/L is acceptable but recheck more frequently. Stop the drug if the level reaches 6 mmol/L and refer (WHO, n.d.).

• Use them in patients with insulin-dependent diabetes; they improve insulin resistance (whereas thiazides and beta-blockers may worsen it).

• Avoid them in patients with peripheral vascular disease.

• Use an angiotensin II receptor antagonist in patients who need an ACE inhibitor but cannot tolerate it because of cough.

**Beta-Blockers**

• Perform a peak flow before and after starting treatment if the history suggests the possibility of chronic obstructive pulmonary disease (COPD). If there is a significant fall, stop the drug. However, cardioselective beta-blockers may be tolerated in mild to moderate reversible airway obstructions (Salpeter, Ormiston, & Salpeter, 2005).

• Warn the patient not to stop a beta-blocker suddenly. Even those without known coronary heart disease (CHD) have a fourfold increased risk of myocardial infarction or angina in the subsequent 4 weeks.

**Alpha-Blockers**

• Total cholesterol falls by an average of 4%, with a beneficial rise in high-density lipoprotein (HDL) cholesterol.

• Start with a low dose (e.g., terazosin 1 mg or doxazosin 1 mg), taken at night in case of first-dose hypotension.

• Use with caution in the elderly, who may experience continued postural hypotension.

**Poor Control**

• Gently ask about adherence. A question such as, “How difficult do you find it to take all of your tablets?” is more likely to elicit a truthful answer than “Do you ever forget to take them?”

• Consider the white-coat effect. If the BP seems to fluctuate or the patient seems tense, arrange for home readings (see Box 7.2).

• If neither of these applies, consider this to be resistant hypertension.

**Resistant Hypertension**

• Resistant hypertension is common, being found in approximately 10% of all treated patients.

• It is defined as hypertension not controlled by three drugs, at best tolerated doses, where the patient is taking them and the BP is raised at home as well as at the clinic.

• It is important, as patients with resistant hypertension are very high risk: They are 50% more likely to experience an adverse cardiovascular event compared to other hypertensive patients (Myat, Redwood, Qureshi, Spertus, & Williams, 2012).

• Before a diagnosis of resistant hypertension can be made, check that this is true resistance, not poor adherence or white-coat hypertension.

• New evidence suggests adding spironolactone as the 4th line medication (after A + C + D) in patients with an eGFR >45 ml/min (Williams et al., 2015).

• Resistant hypertension is likely to be multifactorial. Consider the following:
  1. Lifestyle factors: obesity, excess alcohol intake, excess dietary sodium, cocaine and amphetamines misuse; of these, obesity is the most common feature of patients with resistant hypertension. One study of over 45,000 primary care patients in Germany found that obese individuals (BMI >40 kg/m²) were more than five times more likely to need three antihypertensive drugs and three times more likely to require four antihypertensive drugs to achieve BP control compared with individuals with a normal BMI (≤25 kg/m²) (Sharma et al., 2004).
2. Medication-related causes: NSAIDs, selective COX-2 inhibitors, steroids, sympathomimetics (e.g., decongestants), oral contraceptives, and liquorice ingestion in sweets or chewed tobacco.

3. An underlying medical cause: Up to 10% of these patients have a previously undiagnosed secondary cause:
   - Renal disorders: the commonest overall cause and least amenable to treatment. Includes diabetic kidney disease, glomerulonephritis, chronic pyelonephritis, obstructive uropathy, and polycystic kidney disease.
   - Primary hyperaldosteronism: the commonest single cause of secondary hypertension, accounting for 5% to 13% of all cases of hypertension (Grasko, Nguyen, & Glendenning, 2010). Suspect if there is low potassium (K) and high sodium (Na), although in many patients these will be normal. The ratio of plasma aldosterone to renin will be raised and warrants referral to confirm the diagnosis and the underlying cause (Conn adenoma or idiopathic). Note that spironolactone, eplerenone, amiloride, and dihydropyridine calcium channel blockers (e.g., amlodipine) should be stopped before doing these tests (Grasko et al., 2010).
   - Obstructive sleep apnoea (OSA): ask about snoring, episodes of apnoea at night, and daytime sleepiness.
   - Vascular disorders: coarctation of the aorta (suspect if significant interarm difference in blood pressure). Check for radio-radial or radio-femoral delay; and renal artery stenosis (common in older hypertensives). Suspect if evidence of peripheral vascular disease. Check for abdominal bruit.
   - Thyroid diseases: hyperthyroidism usually increases systolic blood pressure, whereas hypothyroidism usually increases diastolic blood pressure.
   - Cushing disease: look for the typical clinical features (e.g., centripetal obesity, moon facies, abdominal striae).
   - Phaeochromocytoma: suggested by a history of episodic headaches, sweating, palpitations associated with an often dramatic rise of blood pressure. Check 24-hour urinary catecholamines.

   • If a cause for resistance is found that can be managed in primary care, continue management, even if it means proceeding to step 4. Otherwise, refer. Hypertension clinics are capable of controlling half of those referred with resistant hypertension.

**Referral**

• Refer patients in whom there is reason to suspect secondary hypertension: onset age under 40, fluctuating BP levels, evidence of renal disease, resistant hypertension.
• Refer urgently anyone with accelerated hypertension (grade IV retinopathy).

**Chest Pain of Recent Onset**

**GUIDELINE**


**GUIDELINES**


**Unstable Angina (Acute Coronary Syndrome)**

**GUIDELINE**

• Chest pain is very common, accounting for about 1% of all patient encounters in general practice, 5% of visits to the emergency department, and 25% of all emergency hospital admissions (Goodacre, 2005). These encounters represent just a fraction of the number of episodes of chest pain experienced in the community, for which medical attention is not always sought (Elliott, McAteer, & Hannaford, 2011).

• The challenge for GPs is to identify serious cardiac disease while also protecting patients from unnecessary investigations and hospital admissions.

• The 2010 NICE guidelines focus on the assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin. They present two separate diagnostic pathways: one for people with acute chest pain in whom an acute coronary syndrome is suspected; the other for those with intermittent stable chest pain in whom stable angina is suspected (Cooper et al., 2010). Medical history-taking and physical examination will determine which of these pathways to follow, as will be described in the following sections.
• Admit by emergency ambulance any patient with cardiac pain lasting >15 minutes despite GTN, with an abnormal or unavailable ECG. Start management immediately but do not delay transfer to hospital.

• While awaiting admission, give:
  1. GTN (e.g., three sprays over 15 minutes) if pain continues.
  2. Consider IV opioid if available: diamorphine 1 mg/min (maximum 5 mg) or morphine 2 mg/min (maximum 10 mg). Administer with IV antiemetic such as metoclopramide 10 mg or cyclizine 50 mg.
  3. aspirin 300 mg (unless allergic), written record of administration to go with patient;
  4. 12-lead ECG (fax ahead if possible), but do not do if it will delay hospital transfer;
  5. ACS if the ECG is normal;
  6. pulse oximetry (offer oxygen only if O₂ saturation is <94%; aim for 94%–98%);
  7. patient monitoring until diagnosis.

- Chest pain with associated nausea and vomiting, sweating, breathlessness
- Chest pain associated with haemodynamic instability
- New onset chest pain, or abrupt deterioration in previously stable angina, with recurrent pain occurring at rest or at significantly lower levels of activity, or that the frequency, duration, or severity of the attacks has substantially worsened
- Subsequent management depends on timing of presentation:
  1. If the patient is presenting with current chest pain, proceed with immediate management as outlined in Box 7.3.
  2. If the chest pain was in the last 12 hours, but the patient is currently pain free, do an ECG. Arrange emergency admission if the ECG is abnormal or if ECG is unavailable; if ECG is normal, arrange urgent same-day assessment.
  3. If the chest pain was 12 to 72 hours ago, arrange urgent same-day assessment.

Myocardial Infarction (MI)

• Defibrillation: defibrillate a patient who develops ventricular fibrillation while awaiting transfer to hospital. If no defibrillator is available, perform cardiopulmonary resuscitation while waiting for one to arrive. Out-of-hospital defibrillation has been shown to save lives.

• Primary PCI: patients with an ST-segment-elevation acute coronary syndrome should be treated immediately with primary percutaneous coronary intervention (PCI) (SIGN, 2016).

• When primary PCI cannot be provided within 120 minutes of ECG diagnosis, patients with an ST-segment-elevation acute coronary syndrome should receive immediate (prehospital or admission) thrombolytic therapy (SIGN, 2016).

- Thrombolysis: make an initial judgment about the patient’s suitability (see the upcoming contraindications). All patients with a typical history of MI and ST segment elevation or left bundle branch block (LBBB) should be considered for thrombolysis, regardless of age, if their quality of life warrants it, and if local policy does not prefer acute percutaneous coronary intervention (PCI).

- Thrombolysis is of benefit in the 24 hours after the onset of symptoms, with more benefit the sooner it is given. Thrombolysis involves hospital admission, whether or not it is given at home first.

• GPs need special training. It should be given outside hospital only if:
  1. there is strong clinical suspicion of acute myocardial infarction;
  2. chest pain, unrelieved by GTN spray, has been present for at least 20 minutes and for no more than 12 hours;
  3. the ECG shows ST elevation or LBBB;
  4. a defibrillator is available, because of the small but significant increase in risk of ventricular fibrillation (VF) after thrombolysis;
  5. no contraindications exist.

Contraindications to Thrombolysis
(Lip, Chin, & Prasad, 2002)

Absolute
1. Aortic dissection
2. Previous cerebral haemorrhage
3. Known cerebral aneurysm or AV malformation
4. Intracranial neoplasm
5. Any cerebrovascular accident in the last 6 months
6. Active internal bleeding (other than menstruation)
7. Streptokinase should not be given to a patient who has received it more than 4 days previously. The development of antibodies may reduce its effectiveness.

Relative
1. Uncontrolled hypertension (BP >180/110 mm Hg) or chronic severe hypertension
2. On anticoagulants or known bleeding diathesis
3. Trauma within past 2 to 4 weeks, including head injury and prolonged (>10 minutes) CPR
4. Aortic aneurysm
5. Recent (within 3 weeks) major surgery, organ biopsy, or puncture of a noncompressible vessel
6. Recent (within 6 months) GI, or GU, or other internal bleeding
7. Pregnancy
8. Active peptic ulcer

Cardiac Rehabilitation

• Most patients following MI will now routinely be enrolled in a cardiac rehabilitation programme. This should be a comprehensive package of support, including exercise, education, and psychologic support.
• Exercise. Encourage the continuation of exercise, which will continue to improve cardiac performance and survival and reduce the risk of another infarction by 20%. Exercise can be tennis, jogging, cycling, swimming, or circuit training. NICE (2013) recommends 20 to 30 minutes each day, increasing slowly so that it is sufficiently vigorous to make the patient slightly breathless.
• Smoking. Encourage a smoker to stop, with the news that stopping even at this late stage probably reduces mortality by 50%.
• Weight. Advise the obese to lose weight.
• Diet. Whether the patient needs to lose weight or not, recommend a Mediterranean diet. The elements of the diet that seem to be associated with a lower mortality are moderate alcohol, low meat intake, and high intake of vegetables, fruit, nuts, olive oil, and legumes.
• Alcohol. Check that safe limits are not being exceeded.
• Lipids. Arrange a test for serum lipids at 3 months postinfarction, unless the lipids were assessed within 24 hours of the onset of the infarct. Once blood has been taken, start a statin regardless of baseline value and continue it long term.
• Diabetes. Unless known to be diabetic, or to have had an assessment of glucose metabolism in hospital, check the fasting plasma glucose.
• Depression. Look again for depression. Even at 1 year, 25% are depressed.
• Other drugs.
  • Check that the patient continues to take an ACE inhibitor (or an angiotensin-11 receptor blocker if not tolerated).
  • A beta-blocker should be taken for at least 12 months post MI in people without left ventricular dysfunction or heart failure, unless contraindicated. A beta-blocker should be carried on indefinitely in those with left ventricular dysfunction.
  • Dual antiplatelet therapy is usually advised for up to 12 months after an MI. This is usually aspirin plus a second antiplatelet drug such as clopidogrel, prasugrel, or ticagrelor. Aspirin is usually carried on lifelong, clopidogrel can be used for those intolerant of aspirin.
  • Recommend an annual influenza immunization.
• Check that the patient has a cardiology follow-up appointment for an assessment of the coronary arteries and suitability for invasive treatment (PCI), as well as for an assessment of left ventricular function. The initial screening to assess coronary blood flow is likely to be an exercise (stress) ECG. Patients with a negative stress test are unlikely to require coronary artery surgery, and so an unnecessary angiogram can be avoided.

Other Factual Advice

• Sedentary workers may return to work at 4 to 6 weeks, light manual workers at 6 to 8 weeks, and heavy manual workers at 3 months following MI.

• Do not fly for 2 weeks, and then only if able to climb one flight of stairs without difficulty. A more cautious policy is to advise against flying for 6 weeks.
• Drivers should not drive for 1 month, but no longer are required to notify the Driver and Vehicle Licensing Agency (DVLA). They should inform their insurance company. Heavy Goods Vehicle (HGV) and Public Service Vehicle (PSV) licence holders must notify the DVLA, and may only continue vocational driving after individual assessment.

Stable Angina

GUIDELINES

• Angina is the main symptom of myocardial ischaemia and is usually caused by atherosclerotic coronary artery disease restricting blood flow (and therefore oxygen delivery) to the heart muscle.
• Stable angina is common, affecting about 8% of men and 3% of women aged 55 to 64 years and about 14% of men and 8% of women aged 65 to 74 years in England (O’Flynn et al., 2011). The diagnosis is clinical, based on the history.
• The history is of pain or constricting discomfort that typically occurs in the front of the chest, but can radiate to the neck, shoulders, jaw, or arms (NICE, 2011). It is usually brought on by physical exertion or emotional stress, but some people can have atypical symptoms such as gastrointestinal discomfort, breathlessness, or nausea.
• Stable angina is unlikely if the pain is:
  • continuous or very prolonged;
  • unrelated to activity;
  • brought on by breathing in;
  • associated with dizziness, palpitations, tingling, or difficulty swallowing,
  • in such cases, consider other causes of chest pain such as gastrointestinal or musculoskeletal pain.
• The NICE guidelines present a table to help estimate the likelihood of coronary artery disease in someone presenting with symptoms of angina, but such tables are not yet routinely used in general practice. Put simply, the likelihood of angina increases with age and with the presence of additional cardiovascular risk factors:
• Smoking
• Hypertension
• Diabetes
• Family history of CHD (first-degree relative: male <55 years/female <65 years)
• Raised cholesterol (>6.5 mmol/L) and other lipids
As well as these risk factors, also assess for:
1. BMI;
2. hypo/hyperthyroidism;
3. anaemia/polycythaemia;
4. arrhythmias;
5. valvular disease (any aortic systolic murmur needs assessment for aortic stenosis);
6. depression and social isolation;
7. physical activity levels.

**WORKUP—INVESTIGATIONS**

1. Haemoglobin
2. Thyroid function tests (TFTs)
3. Fasting blood sugar
4. Fasting lipid profile
5. Resting ECG. A normal ECG does not exclude the diagnosis but an abnormal ECG makes it more likely. ECG features consistent with coronary artery disease include:
   - pathological Q waves;
   - LBBB;
   - ST segment and T wave abnormalities (e.g., flattening or inversion).

**Drug Treatment of Stable Angina**

*(Bass & Mayou, 2002)*

- Patients require optimal drug treatment with one or two antiangina drugs as necessary to treat symptoms plus drugs for secondary prevention of cardiovascular disease.
- Explain the purpose of the drug treatment, why it is important to take the drugs regularly, and how side effects of drug treatment might affect the person's daily activities.
- Titrate the drug dosage against the person's symptoms up to the maximum tolerable dosage.
- Review the person's response to treatment, including any side effects, 2 to 4 weeks after starting or changing drug treatment.

**Cardiovascular Risk Reduction**

- Encourage lifestyle change (i.e., diet, exercise, smoking, alcohol).
- Consider aspirin 75 mg daily. Clopidogrel is preferred if patient has peripheral arterial disease or if aspirin not tolerated. Dual therapy is only recommended in stable angina after PCI for up to one year.
- Offer statin treatment (Atorvastatin 80 mg) and treat high blood pressure in line with NICE guidelines.
- Consider ACE inhibitors or low-cost ARB for people with stable angina and diabetes, previous MI, CKD or LV systolic dysfunction.

**Selecting Drugs**

1. Offer either a beta-blocker (e.g., atenolol 100 mg, metoprolol 50–100 mg twice daily, or bisoprolol 5–20 mg) or calcium channel blocker (use rate limiting CCB [e.g., diltiazem], unless heart failure or heart block, in which case use amlodipine) as first line treatment.
   - If either is not tolerated, switch to the other.
   - If either is ineffective, switch to the other or combine both (NB: avoid verapamil in combination with beta-blocker).
2. If the person cannot tolerate β-blockers or calcium channel blockers or if both are contraindicated, consider monotherapy with one of the following:
   - A long-acting nitrate
   - Ivabradine
   - Nicorandil
   - Ranolazine
3. If not satisfactorily controlled on two drugs, refer for consideration of revascularization

**Revascularization**

- Coronary intervention such as percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) surgery should only be considered if symptoms are not optimally controlled with medical treatment.
• Refer to cardiology for further assessment, which may include noninvasive investigations and/or coronary angiography.
• Coronary angiography is the traditional investigation for establishing the nature, anatomy, and severity of CHD. It is an invasive investigation and carries a mortality risk of around 0.1% for elective procedures (SIGN, 2018).
• The main purpose of revascularization is to improve the symptoms of stable angina:
  - CABG surgery and PCI are effective in relieving symptoms, but repeat revascularization may be necessary after either procedure, and the rate is lower after CABG surgery.
  - Stroke is an uncommon complication of each of these two procedures (the incidence is similar).
  - There is a potential survival advantage with CABG surgery for some people with multivessel disease.
  - In those for whom both percutaneous and surgical revascularization is feasible, percutaneous revascularization is a more cost-effective approach.

Noncardiac Chest Pain

• Less than half of patients referred to cardiac clinics and accident and emergency (A&E) departments with chest pain are found to have coronary artery disease (Bass & Mayou, 2002). Many of these patients continue to suffer pain and long-term functional impairment. Their management is often inadequate once the diagnosis of cardiac disease is excluded.
• A clinical prediction rule called the Marburg Heart Score (MHS) assists GPs in ruling out chest pain caused by CHD (Haasenritter et al., 2012). Based on five findings from history and examination, the score has a high negative predictive value, but further research is needed to assess its use in routine practice.
• Be clear from the history, investigations, and specialist assessment that the pain is noncardiac. Then resist requests for further cardiac referral.
• Try to make a positive diagnosis of the cause of the pain: musculoskeletal disorders, panic attacks, reflux, depression.
• If a clear diagnosis is possible, treat accordingly (e.g., with NSAIDs for musculoskeletal pain, a PPI for reflux, antidepressants for depression).
• If no clear diagnosis is possible, attempt an explanation along the following lines: that the symptoms are real; that they do not arise from the heart or from any other specific illness; that the brain normally disregards sensations that it receives from all over the body, but that for some reason, in this patient, it is sensitized to sensations from the chest. This explanation might lead on to discussion of how such sensitization may have occurred. The patient may volunteer the memory of some event that seemed to trigger the chest pain.
• Ask who else in the family is worried about the pain and try to see them as well.

Heart Failure

GUIDELINE


• Heart failure is a complex syndrome of symptoms and signs, caused by structural or functional abnormalities of the heart (NICE, 2010b). It is often divided into heart failure due to left ventricular systolic dysfunction (LVSD, associated with a reduced left ventricular ejection fraction) or heart failure with a preserved ejection fraction (HF-PEF). Most of the evidence on treatment is for heart failure due to LVSD. The most common causes of heart failure in the United Kingdom are coronary artery disease and hypertension, and many patients have had a myocardial infarction in the past.
• Both the incidence and prevalence of heart failure increase steeply with age, and the prevalence is expected to rise in the future as the result of an ageing population, improved survival of people with ischaemic heart disease, and more effective treatments for heart failure (Owan et al., 2006).
• The prognosis remains poor, despite improvements in outcomes in the past decade. In one community-based study, 10-year survival rates ranged from 12% in those with multiple-cause heart failure to around 31% in those with LVSD (Taylor, Roalfe, Iles, & Hobbs, 2012).

Diagnosis

• Making the diagnosis of heart failure can be challenging. Patients may present with a range of symptoms, including fatigue, breathlessness, and ankle swelling; and they may also have other conditions such as CHD, diabetes, hypertension, and COPD, which can confuse the picture (SIGN, 2016a).
• Careful history-taking and examination will help to guide further assessment. The following clinical signs will add to the suspicion of heart failure, though none are diagnostic in isolation:
  • Raised jugular venous pressure (JVP)
  • Presence of a third heart sound (S3)
  • Basal crepitations
• Tachycardia
• Peripheral oedema
• Initial investigations should help to rule out other causes of symptoms and should include full blood count, fasting glucose, thyroid function tests, serum urea and creatinine, urinalysis, ECG, and chest x-ray. NICE and SIGN guidelines also now recommend the widespread use of serum natriuretic peptides as an aid to diagnosis of heart failure.

**Serum Natriuretic Peptides**

- B-type natriuretic peptide (BNP) and N-terminal-pro-BNP (NT-pro-BNP) are peptide hormones produced in the heart. They are raised with increasing left ventricular volume and pressure and concentrations tend to rise with NYHA class (Table 7.2). Very high levels indicate a worse prognosis and should prompt urgent referral.
- If the test is negative, heart failure is very unlikely (i.e., it has high sensitivity). False negatives may be caused by obesity and by drugs used to treat heart failure (i.e., diuretics, ACE inhibitors, ARBs, and beta-blockers).
- If the test is positive but prompts referral for echo (i.e., it has relatively low specificity). False positives occur with cardiac ischaemia from any causes (e.g., LVH, tachycardia, hypoxia) and in some other chronic conditions (e.g., diabetes, CKD, COPD, cirrhosis) (Al-Mohammad et al., 2010).

**How to Use B-Type Natriuretic Peptide**

- The first question to ask of someone presenting with possible heart failure is, “Have you previously had an MI?” If so, refer urgently (within 2 weeks) for echo and specialist assessment. There is no need to check BNP in this situation.
- If, however, there is no history of MI, the NICE guidelines suggest checking BNP and referring to cardiology if the level is raised:
  - If very high (BNP >400 pg/mL [116 pmol/L] OR NT-pro-BNP >2000 pg/mL [236 pmol/L]): refer urgently (to be seen within 2 weeks)
  - If raised (BNP 100–400 pg/mL [29–116 pmol/L] OR NT-pro-BNP 400–2000 pg/mL [47–236 pmol/L]): refer within 6 weeks
  - If levels are normal (BNP <100 pg/mL [29 pmol/L] OR NT-pro-BNP <400 pg/mL [47 pmol/L]), a diagnosis of heart failure is unlikely in an untreated patient, and alternative diagnoses should be sought.

**Management of Confirmed Chronic Heart Failure**

Pharmacologic management of heart failure depends on whether the person has LVSD or preserved ejection fraction (most research has been done on the former), and each will be considered in turn in this section. First, there are a number of general measures that apply to both forms of heart failure.

**General Measures**

- **Information:** Explain what is going on and what to do if things get worse.
- **Weight:** Encourage to reduce weight, if obese. Recommend daily weighing at roughly the same time and in similar clothes. Patients should report if they gain more than 2 kg over 2 days. Use diuretics flexibly, guided by weight (Arroll, Doughty, & Andersen, 2010).
- **Diet:** Advise a diet with no added salt.
- **Fluid:** Restrict fluid intake to 2 L/day, unless losing fluids from sweat, diarrhoea, or vomiting.
- **Alcohol:** Keep alcohol intake low. In alcoholic heart disease recommend complete cessation.
- **Smoking:** Stop smoking; it causes vasoconstriction. Refer to the smoking cessation services.
- **Exercise:** Rest in the acute phase, but exercise once stable. Regular low intensity physical activity improves mortality and morbidity. Refer to the heart failure rehabilitation service if this facility exists.
- **Sexual activity:** Be prepared to broach sensitive issues with patients, such as sexual activity, as these may not be raised by patients.
- **Vaccination:** Recommend influenza vaccination annually and pneumococcal vaccination once.
- **Air travel:** Air travel will be possible for the majority of patients with heart failure, depending on their clinical condition at the time of travel.
- **Driving regulations:** Advise patients to check with the DVLA, particularly drivers of large goods vehicles (https://www.gov.uk/government/organisations/driver-and-vehicle-licensing-agency).
- **Depression:** Check for depression. It is present in one third and should be treated as thoroughly as if it was not associated with heart failure. Warn patients not to buy St. John wort over the counter (OTC) because of its interaction with prescribed medication, specifically digoxin and warfarin.
- **NSAIDs:** Stop NSAIDs (including COX-2 inhibitors) if taken, unless they are essential. Warn the patient not to buy them OTC.

**TABLE 7.2 New York Heart Association Classification of Heart Failure Symptoms**

| Class I—asymptomatic | No limitations of ordinary activity |
| Class II—mild | Slight limitation of physical activity but comfortable at rest |
| Class III—moderate | Marked limitation of physical activity. Comfortable at rest but symptomatic on less than ordinary physical activity |
| Class IV—severe | Inability to carry on any physical activity without discomfort. Symptoms present at rest |
• **Carers:** Identify the carer or carers and involve them in the management.

**Chronic Heart Failure (CHF) With Left Ventricular Dysfunction**

Left ventricular systolic dysfunction, assessed by measuring the left ventricular ejection fraction by echocardiography, refers to impaired left ventricular pump (contractile) function. Most evidence to guide management of heart failure is in this group of patients, which make up around half of all heart failure cases (Arroll, Doughty, & Andersen, 2010).

• All patients should be offered both ACE inhibitors and beta-blockers, unless there are contraindications.

**ACE Inhibitors/Angiotensin-II Receptor Blockers**

• ACE inhibitors increase the ability to exercise, improve well-being, and prolong life in all degrees of heart failure (Dargie & McMurray, 1994).

• ARBs (e.g., candesartan and valsartan) may be used in patients intolerant of ACE inhibitors (e.g., with ACEI-related cough).

• If there is a high probability of chronic heart failure (CHF), start treatment before waiting for echo, unless valve disease is suspected, in which case wait.

• Exclude the absolute contraindications of allergy to ACE inhibitors and pregnancy.

• Start at a low dose and titrate upward at short intervals (e.g., every 2 weeks) until the optimal tolerated or target dose is achieved.

• Measure serum urea, creatinine, electrolytes, and eGFR when starting an ACE inhibitor and after each dose increment. Also check blood pressure.

• ACE inhibitors (and ARBs) should not normally be started if the pretreatment serum potassium concentration is significantly above the normal reference range (typically >5.0 mmol/L). Stop ACEI/ARB therapy if the serum potassium concentration rises to above 6.0 mmol/L and other drugs known to promote hyperkalaemia have been discontinued.

• If there is a drop in eGFR at or over 25% or an increase in plasma creatinine at or over 30%, investigate other causes of a deterioration in renal function such as volume depletion or concurrent medication (e.g., NSAIDs). If no other cause is found, stop the ACEI/ARB or reduce dose to previously tolerated level.

• Advise the patient to take the first dose before going to bed to reduce the effect of first-dose hypotension.

• Once the dose is stable, repeat creatinine and electrolytes at 3 months then 6 monthly or sooner if further dose titration is necessary or there is intercurrent infection.

• Warn the patient to avoid dehydration, for instance in gastroenteritis or in hot weather, as this can cause a sudden deterioration in renal function. If the weight drops by more than 1 kg because of dehydration, the patient should stop the diuretic and drink more fluid.

**Beta-Blockers**

• All patients with LVSD should be started on a licensed beta-blocker (e.g., bisoprolol, carvedilol, nebivolol) once heart failure is controlled, provided there are no absolute contraindications (e.g., heart block or asthma).

• In patients already taking another beta-blocker (e.g., atenolol for angina) who develop heart failure, advise switching to one of the above. A study in British general practice found that switching to bisoprolol or carvedilol, mainly from atenolol, was the single most valuable manoeuvre in patients with left ventricular dysfunction (Mant et al., 2008).

• NICE guidelines suggest you should not be put off prescribing beta-blockers in individuals with what were previously considered to be relative contraindications, such as older adults and those with peripheral vascular disease, erectile dysfunction, diabetes mellitus, interstitial pulmonary disease, and COPD without reversibility.

• Take a “start low, go slow” approach. Start at a low dose (e.g., bisoprolol 1.25 mg daily or carvedilol 3.125 mg twice daily), and double the dose every 2 to 4 weeks until the target dose is reached (bisoprolol 10 mg daily, carvedilol 25 mg twice daily).

• Monitor heart rate, BP, and clinical status at each dose titration. If the pulse is less than 50 and symptoms have worsened, halve the dose. Stop and seek specialist advice if symptoms have become severe.

• If at any stage it is necessary to stop the beta-blocker, tail it off gradually to avoid rebound ischaemia and arrhythmias.

**Diuretics (Other Than Spironolactone)**

• Diuretics should be routinely used for the relief of congestive symptoms and fluid retention in patients with heart failure, and titrated (up and down) according to need following the initiation of first line heart failure therapies. Their only function is symptom relief.

• Avoid overtreatment, which can lead to dehydration and renal dysfunction, particularly with loop diuretics. Hypokalaemia is a common cause of dizziness and lightheadedness.

• Monitor blood pressure and renal function after 4 weeks and then 6 monthly.

• Use a loop diuretic (e.g., furosemide or bumetanide) in moderate to severe heart failure. They will cause less hypokalaemia and impotence than a high-dose thiazide (e.g., bendroflumethiazide). Increase the dose gradually until diuresis occurs.

• Once stable, reduce the dose to as low as possible without oedema or breathlessness occurring. Alternatively, consider stopping or reducing to a thiazide diuretic.

• The addition of a thiazide diuretic or a potassium sparing diuretic such as spironolactone (or both) to loop diuretics can be useful if pulmonary or ankle oedema persists,
because the different classes of diuretic are thought to have an additive effect. However, even in short-term combined use, the diuresis can be excessive and can cause hypokalaemia. Check creatinine and electrolytes after 3 days.

**Second Line Pharmacologic Management**

- If a patient remains symptomatic despite optimal treatment with ACE inhibitor and beta-blocker, refer for specialist advice on second line treatment which might include:
  - A mineralocorticoid receptor antagonist (MRA), such as spironolactone or eplerenone;
  - an ARB;
  - sacubitril/valsartan (angiotensin receptor neprilysin inhibitor) - stop ACE inhibitor and ARB, continue beta-blocker and MRA;
  - hydralazine plus nitrate (especially for a patient of African or Caribbean descent with moderate to severe heart failure);
  - ivabradine;
  - digoxin.

**Mineralocorticoid Receptor Antagonists**

- Hyperkalaemia is a potentially fatal adverse effect and is most likely in those with impaired renal function. Only give spironolactone if the creatinine is less than 220 μmol/L.
- Warn the patient to avoid salt substitutes with a high potassium content. Warn males about gynaecomastia.
- If K⁺ reaches 5.5 to 5.9 mmol/L or creatinine rises to 200 μmol/L, reduce the dose and monitor closely. If those levels are exceeded, stop and seek specialist advice.

**Ivabradine**

- Ivabradine was approved by NICE in November 2012 and is an option for patients:
  - with symptomatic stable heart failure (NYHA class II to IV) with LVSD and an ejection fraction of 35% or less;
  - who are in sinus rhythm with a resting heart rate above 75 bpm;
  - who are also receiving standard therapy including beta-blockers, ACE inhibitors, and aldosterone antagonists.
- Ivabradine should be initiated by a heart failure specialist (e.g., specialist nurse) with access to a multidisciplinary heart failure team.

**Digoxin**

- Consider for patients in sinus rhythm if still symptomatic despite all the above. See the upcoming section on atrial fibrillation for the use of digoxin in patients with AF.

**Heart Failure With Preserved Ejection Fraction (HF-PEF)**

- As noted, it is thought that up to 50% of patients with heart failure have a preserved ventricular ejection fraction. It has the same symptoms and signs as heart failure with LVSD, and carries a similar mortality risk but has a less clear evidence base for treatment (Jong, McKelvie, & Yusuf, 2010).
  - Patients with HF-PEF are more likely than those with LVSD to be older, female, have hypertension, and atrial fibrillation but are less likely to have coronary artery disease.
  - NICE recommends the use of diuretics for symptomatic relief.
  - ACE inhibitors and beta-blockers should be considered, particularly when there are other compelling indications for their uses (such as coronary artery disease, hypertension, and diabetes mellitus).
  - If atrial fibrillation is present, digoxin may be added for rate control, and anticoagulation should be considered.

**Grounds for Admission**

1. Severe symptoms (e.g., severe dyspnoea or hypotension)
2. Acute myocardial infarction
3. Severe complicating medical illness (e.g., pneumonia)
4. Inadequate social support
5. Failure to respond to treatment
6. Uncontrolled arrhythmia

**Referral for Specialist Review**

- Refer patients to the specialist multidisciplinary heart failure team for:
  - the initial diagnosis of heart failure;
  - the management of:
    1. severe heart failure (NYHA class IV);
    2. heart failure that does not respond to treatment;
    3. heart failure that can no longer be managed effectively in the home setting.
- Consider specialist review:
  - when renal function continues to deteriorate or deteriorates rapidly;
  - when there are concerns about low blood pressure;
  - when the patient is pregnant or considering pregnancy.

**End-Stage Heart Failure**

- Issues of sudden death and living with uncertainty are pertinent to all patients with heart failure. The opportunity to discuss these issues should be available at all stages of care.
- Deciding on prognosis in heart failure is difficult and cardiologic advice may be needed. The palliative needs of patients and carers should be identified, assessed, and managed at the earliest opportunity. There should be access to professionals with palliative care skills within the heart failure team.
- Nonessential treatment should be stopped.
- The focus should be on symptom control (see the upcoming discussion).
Breathlessness

- Morphine is the most effective palliation. Give a low oral dose (2.5–5 mg 4-hourly), as peak plasma levels are higher in heart failure.
- If breathless at rest then consider oxygen therapy at home (40%–80% if there is no COPD).
- Refer for relaxation, breathing exercises, and anxiety management if these are available.

Weakness and Fatigue

- This is usually secondary to a low cardiac output.
- Drug reduction. Consider reducing ACE inhibitors and beta-blockers, and diuretics if there is dehydration.
- Depression. Avoid tricyclics; SSRIs may be of value.
- Social factors. Increase social care if possible and ensure support for the carer(s).

Anorexia/Nausea

- Digoxin. Check serum level and renal function.
- Consider increasing diuretics if there is hepatic congestion.
- Diet. Advise the patient to have small, appetizing meals more frequently. Allow small amounts of alcohol before meals.
- Antiemetics. Use levomepromazine. Avoid cyclizine.

Oedema

- Mobilization is advisable in theory but rarely practicable at this stage.
- Diuretics. Avoid increasing diuretics at this stage unless doing so gives symptomatic relief. They are unlikely to have much effect on the oedema.
- Be cautious about advising the patient to raise the feet. This may increase the venous return to the heart and worsen the dyspnoea.
- Compression stockings/bandages. These may increase tissue damage and are uncomfortable.

Acute Pulmonary Oedema

- When called urgently to the patient in extremis, get someone else to dial for an ambulance while you:
  1. sit the patient up with the legs down;
  2. give oxygen if available;
  3. give a GTN tablet or spray sublingually (for its immediate vasodilator effect);
  4. give intravenous (IV) diamorphine 1 mg/min up to 5 mg, or IV morphine 2 mg/min up to 10 mg, mixed with metoclopramide 10 mg IV; do not use cyclizine as it raises systemic arterial pressure;
  5. give a loop diuretic (IV furosemide 50 mg or IV bumetanide 1 mg).
- Look for causes of the heart failure that may need treatment in their own right, especially myocardial infarction.
- Admit once the patient is sufficiently stable, for fuller assessment and for continuing treatment.

- Note: Digoxin and aminophylline should not be used in acute failure outside hospital.

Right Heart Failure

Acute Right Heart Failure

- The clinical picture of low output state, no pulmonary oedema, and a raised JVP may be due to massive pulmonary embolism or to acute right heart failure as in a right ventricle myocardial infarction, or acute cor pulmonale.
- Admit urgently. Position the patient however they are the most comfortable. Do not give morphine; respiratory depression and hypotension are very likely to follow. Do not give diuretics as they may precipitate shock.

Chronic Cor Pulmonale

- Treat the lung disease by considering:
  1. antibiotics for exacerbations of infection;
  2. inhaled bronchodilators and steroids via a spacing device;
  3. physiotherapy;
  4. long-term oxygen therapy;
  5. nasal intermittent positive pressure ventilation for those with thoracic deformities and obstructive sleep apnoea.
- Admit readily anyone with an acute exacerbation. Oxygen will dilate the pulmonary vessels and so reduce the pulmonary artery pressure. The danger of hypercapnoea makes this hazardous in the acute situation at home.
- Use diuretics carefully as they may reduce renal blood flow further. They are only required if oedema becomes troublesome.
- Use digoxin only if the patient is in atrial fibrillation.
- Do not start ACE inhibitors outside hospital.

Palpitations and Arrhythmias

- Palpitations are a common presentation in general practice and a frequent reason for cardiology referrals. They often cause considerable distress and anxiety for the patient; however, more often than not they are benign, with less than half of patients with palpitations suffering from an arrhythmia and not every identified arrhythmia being of clinical or prognostic significance (Mayou, Sprigings, Birkhead, & Price, 2003).
- Furthermore, not all arrhythmias present with palpitations. Some may cause no symptoms, while in other cases symptoms include tachycardia, bradycardia, chest pain, breathlessness, lightheadedness, and fainting (syncope) or near fainting.
- As well as arrhythmias, palpitations can be caused by structural heart diseases, psychosomatic disorders, systemic diseases, and effects of medical and recreational drugs (Raviele et al., 2011).
• Careful history-taking and physical examination are important, but further investigation is invariably required (Hoefman et al., 2007).

**History**

• Ask what the patient means by *palpitations*. It may be more like a pulsatile tinnitus or a chest discomfort.
• Ask about the rate, frequency, and duration of palpitations, as well as exacerbating and relieving factors. The patient may be able to tap out the rate on the consulting desk.
• Are there any associated symptoms, such as sweating, breathlessness, or chest pain? If any of these are present and the patient has palpitations at that time, refer to hospital immediately by 999 ambulance.
• Ask about smoking, alcohol, caffeine consumption, and use of illicit substances such as cocaine, ecstasy, and amphetamines.
• Ask about general well-being. In particular, assess for any life stressors and ask about personal or family history of anxiety or heart problems.

**Examination**

• Assess for tremor, which may indicate thyrotoxicosis or anxiety. Ask the patient to hold the arms outstretched in front with the palms down and to spread the fingers.
• Assess pulse rate and rhythm and check blood pressure. Is the pulse rate regularly irregular, suggesting ectopic beats, or irregularly irregular, suggesting AF or atrial flutter?
• Examine the heart, assessing for evidence of structural heart disease (e.g., murmurs, abnormal heart sounds, or signs of heart failure).

**Investigations**

• Blood tests should include full blood count (to exclude anaemia), urea and electrolytes, and thyroid function tests.
• Arrange a 12-lead ECG. Abnormalities to look for include:
  - atrial fibrillation;
  - second- and third-degree AV block;
  - signs of previous myocardial infarction;
  - left ventricular hypertrophy and left ventricular strain patterns;
  - left bundle branch block;
  - abnormal T wave inversion and ST segment changes;
  - signs of preexcitation (short PR interval and delta waves);
  - abnormal QTc interval and T wave morphology.

**Next Steps**

• There is no validated risk stratification tool that is widely used in practice, although some authors have proposed a traffic light system to guide further management (Wolff & Cowan, 2009).

• Patients with the following features should be referred *urgently* to cardiology:
  - Palpitations with syncope/near syncope
  - Palpitations during exercise
  - Family history of inheritable heart disease/sudden arrhythmic death syndrome (SADS)
  - High-risk structural heart disease
  - High-degree atrioventricular block
• Patients with the following features should be referred routinely to cardiology or to local palpitations assessment service:
  - Palpitations with associated symptoms
  - Abnormal ECG
  - History suggestive of recurrent tachyarrhythmia
  - Structural heart disease
• Patients with the following features may not require referral:
  - History in keeping with extrasystoles/ectopic beats
  - Normal ECG
  - No family history
  - No evidence of IHD or structural heart disease

**Ectopic Beats in a Normal Heart**

• Explain their benign nature to the patient.
• Advise against caffeine, fatigue, smoking, and alcohol.
• *Beta-blockers* should be used only if the patient is unable to tolerate the ectopics.
• *Frequent ventricular ectopics (>100/hour)* may be grounds for referral. Patients may have a prolapsing mitral valve, or the older patient may have unsuspected ischaemic heart disease.
• For those still troubled by their symptoms, consider referral for cognitive behavioural therapy (CBT) or similar (Mayou, Sprigings, Birkhead, & Price, 2002).

**Atrial Fibrillation**

• Atrial fibrillation (AF) is the most common disorder of heart rhythm with a prevalence that increases with age from about 6% in people aged 65 to 74, to 12% in people aged 75 to 84, and 16% in people aged 85 and over (Fitzmaurice et al., 2007).
• It is associated with increased risk of stroke and heart failure, with higher mortality as a result.
• AF also increases the risk of sudden cardiac death (Chen, Sotoodehnia, & Bůžková, 2013).
• Oral anticoagulation can reduce the risk of stroke by two-thirds (Aguilar & Hart, 2005). However, current
evidence suggests that only about half of patients with AF identified as being at high risk are receiving anticoagulants (Holt et al., 2012).

- The older you are, the more dangerous AF becomes, with strokes being more common and more disabling. Yet undue use of anticoagulation is highest in older people, who have most to gain from treatment (Hobbs et al., 2011).
- Primary care has considerable potential to further reduce the risk of stroke in AF, by identifying high-risk patients and lowering the threshold for offering anticoagulation, as recommended in guidelines.

Identification of Atrial Fibrillation

- The SAFE study in 2005 showed that opportunistic screening (by pulse palpation followed by ECG if pulse irregular) of patients aged over 65 is cost effective for detecting AF (Hobbs et al., 2005). Opportunistic screening is endorsed by the European Society of Cardiology Guidelines.
- Some have gone further, suggesting that a national screening programme should be introduced, using this approach, for all patients over 65 (James & Campbell, 2012).
- Screening of such patients while attending annual influenza vaccination clinics has been piloted, but with mixed results (Gordon, Hickman, & Pentney, 2012; Rhys, Azhar, & Foster, 2013).

WORKUP OF ATRIAL FIBRILLATION

1. History of alcohol intake (either chronic or bingeing)
2. Blood pressure (half of all cases of AF are hypertensive)
3. TFTs, creatinine, and electrolytes
4. Examine for heart failure, valvular heart disease, congenital heart disease, or acute pericarditis or myocarditis
5. ECG (looking for ischaemic heart disease [IHD], left ventricular (LV) strain, and delta waves)
6. NICE recommend performing an echocardiogram in patients for whom:
   - a baseline echocardiogram is important for long-term management;
   - a rhythm control strategy that includes cardioversion is being considered;
   - there is a high risk or a suspicion of underlying structural/functional heart disease (such as heart failure or heart murmur) that influences subsequent management (e.g., choice of antiarrhythmic drug);
   - refinement of clinical risk stratification for antithrombotic therapy is needed.

- Admit if rapid AF is associated with:
  1. chest pain;
  2. hypotension;
  3. more than mild heart failure.
- Refer urgently if seen within 48 hours of the onset of AF and the patient is a candidate for cardioversion (see the upcoming discussion).

Cardioversion Versus Rate Control

- Rate control is recommended as the treatment of choice in most patients. It has advantages over rhythm control: fewer hospital admissions, fewer adverse drug reactions, possibly a lower mortality (Atrial Fibrillation Follow-up Investigation of Rhythm Management [AFFIRM], 2002), and it is more cost effective (Hagens, Vermeulen, & TenVergert, 2004).
- Referral for cardioversion is more appropriate for certain patients, either because it is more likely to succeed than in others (the first three categories) or because sinus rhythm offers greater chance of clinical benefit than rate control (the last two categories):
  - Younger patients
  - Those presenting for the first time with lone AF
  - Those with AF secondary to a cause that has now been treated
  - Those who are symptomatic
  - Those with congestive heart failure (restoration of sinus rhythm may improve LV function)

The decision as to whether to use chemical or electrical cardioversion will be made in discussion between the patient and cardiologist. Vernakalant is a new intravenous antiarrhythmic agent approved for cardioversion of AF of 7 days duration or less. Other agents include ibutilide, flecainide, propafenone, and amiodarone.

- Rate control is more appropriate in those in whom cardioversion is less likely to succeed:
  - Those over 65 years old
  - Those with structural heart disease or CHD
  - Those whose AF is longstanding (e.g., >12 months)
  - Those in whom previous attempts at cardioversion have failed or been followed by relapse
  - Those in whom an underlying cause (e.g., thyrotoxicosis) has not yet been corrected
  - Those with a contraindication to antiarrhythmic drugs

- Aim for lenient (HR <110 bpm) rather than strict (HR <80 bpm) rate control, unless patient is symptomatic.
- Use:
  1. a beta-blocker first line (e.g., atenolol, bisoprolol, metoprolol);
  2. a rate-limiting calcium channel blocker second line (e.g., diltiazem or verapamil); avoid both in heart failure, and avoid the combination of verapamil and a beta-blocker;
  3. digoxin if still symptomatic. If digoxin is contraindicated, add diltiazem to the beta-blocker. The usual concern about causing bradycardia or AV block is less of a problem in AF since that is the aim of the treatment.

Risk Stratification for Anticoagulation

- A risk factor-based approach to stroke risk stratification has been promoted for many years (e.g., CHADS2
Anticoagulation

- For more than half a century, warfarin has been the primary medication used to reduce the risk of thromboembolic events in patients with atrial fibrillation (Mega, 2011).
- Research has confirmed that warfarin is superior to antiplatelet therapy (e.g., aspirin) in the management of AF in the community, even in older people (Mant et al., 2007). Aspirin should no longer be used for stroke prevention in AF; such patients should be reviewed.
- Despite its clinical efficacy, warfarin has several limitations, including drug interactions and the need for regular blood monitoring (INR should be controlled to between 2 and 3) and dose adjustments. See the accompanying table for contraindications to warfarin.
- As a result, alternative anticoagulants that are at least as efficacious but easier to administer have been developed and widely promoted.
- Four novel oral anticoagulant agents (NOACs) have been approved by NICE as alternatives to warfarin in nonvalvular AF. These are dabigatran, rivaroxaban, apixaban, and edoxaban.
- Dabigatran is a direct thrombin inhibitor and the other three are factor Xa inhibitors.
- The main advantages of these agents are that they are fixed dose, less susceptible to interactions, and do not require monitoring.

Assessment of Bleeding Risk

- Before starting anticoagulation, an assessment of bleeding risk should be undertaken. Major bleeding, especially intracranial haemorrhage (ICH), is the most feared complication of anticoagulation therapy and confers a high risk of death and disability (Connolly et al., 2011).
- Several different guidelines recommend the use of the HAS-BLED score, which has been validated in many cohort studies and correlates well with ICH risk (Table 7.4) (Cairns, Connolly, McMurtry, Stephenson, & Talajic, 2001; Camm et al., 2010; Lip et al., 2011).
- A HAS-BLED score of 3 or more indicates a bleeding risk (ICH or requiring admission) on anticoagulation over the next year sufficient to justify caution with anticoagulation.
- A score of 3 or above does not exclude patients from receiving anticoagulation, but would require extra caution to control bleeding risks (e.g., closer monitoring of renal function and INR, better management of hypertension).

### TABLE 7.3 CHA2DS2-VASc

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>H</td>
<td>Hypertension</td>
</tr>
<tr>
<td>A₂</td>
<td>Age ≥75</td>
</tr>
<tr>
<td>D</td>
<td>Diabetes</td>
</tr>
<tr>
<td>S₂</td>
<td>CVA or TIA</td>
</tr>
<tr>
<td>V</td>
<td>Vascular disease</td>
</tr>
<tr>
<td>A</td>
<td>Age &gt;65</td>
</tr>
<tr>
<td>Sc</td>
<td>Sex category (i.e., female)</td>
</tr>
</tbody>
</table>

**TOTAL SCORE**

- **0** = low risk (0.8% annual stroke rate)
- **1** = moderate risk (1.75% annual stroke rate)
- **≥2** = high risk (>2.7% annual stroke rate)
- **3** = 3.2%; **5** = 6.7%; **7** = 9.6%

### TABLE 7.4 The HAS-BLED Bleeding Risk Score (Pisters et al., 2010)

<table>
<thead>
<tr>
<th>Letter</th>
<th>Clinical Characteristic</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>Abnormal renal and liver function (1 point each)</td>
<td>1 or 2</td>
</tr>
<tr>
<td>S</td>
<td>Stroke</td>
<td>1</td>
</tr>
<tr>
<td>B</td>
<td>Bleeding</td>
<td>1</td>
</tr>
<tr>
<td>L</td>
<td>Labile INRs</td>
<td>1</td>
</tr>
<tr>
<td>E</td>
<td>Elderly (over 65)</td>
<td>1</td>
</tr>
<tr>
<td>D</td>
<td>Drugs or alcohol (1 point each)</td>
<td>1 or 2</td>
</tr>
</tbody>
</table>

**TOTAL**

Hypertension is defined as systolic blood pressure >160 mm Hg.
Abnormal kidney function is defined as the presence of chronic dialysis or renal transplantation or serum creatinine ≥200 mmol/L.
Abnormal liver function is defined as chronic hepatic disease (e.g., cirrhosis) or biochemical evidence of significant hepatic derangement (e.g., bilirubin >2 × upper limit of normal, in association with aspartate aminotransferase/alanine aminotransferase/alkaline phosphatase >3 × upper limit normal, etc.).
Bleeding refers to previous bleeding history and/or predisposition to bleeding (e.g., bleeding diathesis, anemia).
Labile INRs refers to unstable/high INRs or poor time in therapeutic range (e.g., <60%).
Drugs/alcohol use refers to concomitant use of drugs, such as antiplatelet agents, nonsteroidal antiinflammatory drugs, or alcohol abuse.
INR, International normalized ratio.
Left Atrial Appendage Closure

- The left atrial appendage (LAA) is considered the main (though not the only) site of thrombus formation leading to ischaemic stroke in people with AF.
- Minimally invasive techniques have been developed to occlude the LAA orifice and thereby reduce stroke risk, with results equivalent to warfarin (Holmes et al., 2009).
- At present, this procedure is only considered for patients in whom oral anticoagulation is contraindicated.

Paroxysmal Atrial Fibrillation

- Paroxysmal atrial fibrillation (PAF) is defined as recurrent (two or more) episodes of AF that terminate spontaneously in less than 7 days, and usually less than 24 hours.
- Tailor the treatment to the severity.
- The patient with infrequent attacks and no serious symptoms, or attacks that can be averted by avoiding a precipitating cause (e.g., alcohol or caffeine), either give:
  - no drug treatment, or
  - a pill-in-the-pocket, to be taken at the onset of an attack, if the patient is suitable (see the accompanying box).
- The patient with more frequent attacks or more severe symptoms:
  - receives a standard beta-blocker.
• If that is ineffective or not tolerated, refer for specialist assessment. Other medications such as sotalol, flecainide, propafenone, or amiodarone may be considered. Dronedarone, which is structurally related to amiodarone, has been approved for the treatment of paroxysmal or persistent AF, but only under specialist initiation and supervision. It should not be given to patients with moderate or severe heart failure, and should be avoided in patients with less severe heart failure, if appropriate alternatives exist.

• NICE warns of the danger of initiating drugs other than standard beta-blockers for paroxysmal AF in general practice without specialist advice because of the risk that the drug will itself cause ventricular arrhythmias. This is most likely in those with underlying heart disease. In practice the patient will often have been assessed already by a cardiologist and had previous experience of an antiarrhythmic drug without adverse effects, obviating the need for re-referral.

NICE RECOMMENDATIONS FOR THE SUITABILITY OF A PATIENT FOR A “PILL-IN-THE-POCKET”

1. Infrequent episodes (e.g., between once per month and once per year)
2. Sufficiently reliable to use the treatment correctly
3. No structural heart disease, coronary heart disease, or left ventricular dysfunction
4. Satisfactory baseline state: systolic blood pressure >100 mm Hg, resting heart rate >70 bpm

• If a paroxysm becomes persistent (arbitrarily defined as lasting at least 7 days), refer for consideration of cardioversion. Refer immediately if the patient develops heart failure or hypotension.

Catheter Ablation

• The European Society of Cardiology (ESC) guidelines recommend that catheter ablation (by pulmonary vein isolation) be considered as a first line therapy for AF rhythm control in selected patients (i.e., those with PAF with a preference for interventional treatment and a low-risk profile for procedural complications) (Kirchhof et al., 2016).

• Studies have shown promising results (Nielsen et al., 2012), but the procedure carries the risk of invasive complications. In one study, complication rates were 0.6% for stroke, 1.3% for tamponade, 1.3% for peripheral vascular complications, and around 2% for pericarditis (Arbelo et al., 2012).

Anticoagulation

A patient with paroxysmal AF should be considered for antithrombotic treatment according to the presence of risk factors (e.g., as assessed by CHA2DS2-VASc). The risk of stroke, although less well defined in paroxysmal AF, is considered to be the same as in persistent or permanent AF, regardless of the number and severity of paroxysms (Friberg, Hammar, & Rosenqvist, 2010).

Atrial Flutter

• Atrial flutter should be approached in a similar way to atrial fibrillation, with a focus on:
  1. rate control;
  2. rhythm control;
  3. prevention of thromboembolic complications.

• It is much less common than AF and usually less well tolerated, often presenting with palpitations.

• Episodes of atrial flutter and AF can occur in the same person.

• Refer all suspected cases for specialist assessment.

• Radio-frequency catheter ablation is first line treatment (Sawhney & Feld, 2008).

Paroxysmal Supraventricular Tachycardia

• If the patient is seen during the attack, get the patient to perform a Valsalva manoeuvre. This can be described to the patient as trying to breathe out forcefully while keeping the mouth closed and nose pinched, or simulating straining on the toilet (Whinnett, Afzal Sohaib, & Wyn Davies, 2012).

• Apply carotid sinus massage except where the patient:
  1. is elderly;
  2. has ischaemic heart disease;
  3. is likely to be digoxin toxic;
  4. has a carotid bruit;
  5. has a history of transient ischaemic attacks.

  Note: The British National Formulary recommends ECG monitoring during carotid sinus massage.

• Admit if the attack continues and there is no clear history of previous attacks which have terminated themselves. Even if there is such a history, keep the patient at the surgery until the attack indeed terminates.

• Record an ECG and give the patient a copy.

• If SVT is diagnosed from the patient’s history or if the attack has terminated before admission was needed:
  • Refer for specialist confirmation and initiation of treatment. Referral should be urgent if attacks are associated with chest pain, dizziness, or breathlessness.
  • Catheter ablation is the first line definitive management option for SVT.

• Drug treatment is reserved for minimizing symptoms while awaiting catheter ablation or for those who decline catheter ablation or in whom the procedure carries an unacceptably high risk.

• Discuss with a cardiologist before starting an antiarrhythmic drug (e.g., sotalol, flecainide, verapamil), while awaiting the cardiology appointment.

• A baseline ECG, with a further ECG before each dose increase, is recommended to look for prolongation of the QT interval, an indicator that there is a risk of drug-induced torsade de pointes. A final decision on
CHAPTER 7  Cardiovascular Problems

the most appropriate drug will depend on the electrophysiology in the individual patient.

- Instruct the patient in the use of the Valsalva manoeuvre, and check that he or she is not smoking or misusing alcohol or caffeine.

Ventricular Tachycardia

- VT associated with loss of consciousness or hypotension is a medical emergency requiring immediate cardioversion.
- Call 999 emergency ambulance.
- Remember ABCs (airway, breathing, circulation), oxygen, ECG monitoring.
- If the patient is conscious but in extremis, consider IV lidocaine 100 mg while waiting for the ambulance.
- After discharge, prophylaxis will be needed. If amiodarone is chosen, 6-monthly TFTs and LFTs are necessary.

Sick Sinus Syndrome

This requires admission for pacing. Drugs are likely to make symptoms worse because of the variability of the rhythms.

Bradycardia

- Refer all patients with a bradycardia, other than sinus bradycardia, even if asymptomatic. A pacemaker is likely to be needed and may be life saving. Untreated second degree and complete atrioventricular (AV) block have a mortality of 25% to 50% in the first year after diagnosis. For this reason, even asymptomatic patients with a rate of 40 or below should be paced.
- Admit a patient in acute AV block with hypotension due to the bradycardia. Give IV atropine if available while waiting for the ambulance.

PATIENT SUPPORT GROUPS

- Arrhythmia Alliance: www.heartrhythmcharity.org.uk/
- Atrial Fibrillation Association: www.atrialfibrillation.org.uk/
- Sudden Adult Death Trust: www.sadsuk.org/
- Cardiac Risk in the Young: www.c-r-y.org.uk/

Prophylaxis of Infective Endocarditis

- Do not offer antibiotics to prevent ineffective endocarditis (IE) for any of the following procedures:
  - Any dental procedure
  - An obstetric or gynaecologic procedure, or childbirth
  - A procedure on the bladder or urinary tract
  - A procedure on the oesophagus, stomach, or intestines
  - A procedure on the airways (including ear, nose, and throat and bronchoscopy)
- People with the following cardiac conditions should be regarded as being at risk of developing IE:
  - Acquired valvular heart disease with stenosis or regurgitation
  - Valve replacement
  - Structural congenital heart disease, including surgically corrected or palliated structural conditions, but excluding isolated atrial septal defect, fully repaired ventricular septal defect or fully repaired patent ductus arteriosus, and closure devices that are judged to be endothelialized
  - Hypertrophic cardiomyopathy
  - Previous infective endocarditis
- People at risk of IE should be offered clear and consistent information about prevention, including:
  - the benefits and risks of antibiotic prophylaxis, and an explanation of why antibiotic prophylaxis is no longer routinely recommended;
  - the importance of maintaining good oral health;
  - symptoms that may indicate IE and when to seek expert advice;
  - the risks of undergoing invasive procedures, including nonmedical procedures such as body piercing or tattooing.
- In people at risk of IE, it is important to investigate and treat promptly any episodes of infection to reduce the risk of endocarditis developing.
- If a person at risk of IE is receiving antimicrobial therapy because of undergoing a gastrointestinal or genitourinary procedure at a site where there is a suspected infection, offer an antibiotic that covers organisms that cause infective endocarditis.

PATIENT INFORMATION

There are a range of resources available through the British Heart Foundation website at www.bhf.org.uk. Their helpline (0300 330 3311) is open Monday through Friday, 9 am to 5 pm.

The Prevention of Cardiovascular Disease

- Antibiotic prophylaxis aims to reduce the incidence of infective endocarditis.
- The NICE guidance represented a major shift in advice on antibiotic prophylaxis.
Cardiovascular disease (CVD) describes disease of the heart and blood vessels caused by the process of atherosclerosis and predominantly affects people older than 50 years (NICE, 2014a). There are significant gender differences in cardiovascular risk, including biologic differences associated with pregnancy and menopause (Parikh, 2011), as well as differences in behavioural risk factors (Huxley & Woodward, 2011).

Worldwide, the two most important modifiable cardiovascular risk factors are smoking and abnormal lipids (SIGN, 2017).

The next most important are hypertension, diabetes, psychosocial factors (including deprivation), and abdominal obesity, but their relative effects vary in different regions of the world.

Risk factors tend to cluster and their effects are multiplicative, not additive. The presence of one or more of the following increases the chance that other risk factors will also be present: hypertension, raised cholesterol, inactivity, obesity, smoking, and glucose intolerance (Perry, Wannamethee, & Walker, 1995).

Strategies for the prevention of cardiovascular disease can be divided into primary and secondary prevention. Primary prevention is concerned with preventing the occurrence of cardiovascular disease in those currently unaffected. Secondary prevention relates to delaying or reversing the progression of disease in those already affected. While targeting those at highest risk is known to be most cost effective, most countries recognize the importance of a multifaceted approach, adopting both population-based and more targeted interventions (Rose, 1981).

Primary Prevention

For the primary prevention of CVD in primary care, a systematic strategy should be used to identify people who are likely to be at high risk, now considered to be a 10%, 10-year risk after lifestyle modification. The NICE (2014) guidelines remain controversial, reflecting uncertainty in the data that underpins them (Otto, 2016).

The role of GPs and practice nurses is, first and foremost, to support patients to make and sustain lifestyle changes where appropriate and to help them make an informed decision regarding statins if they are at increased CVD risk. This should be based on an individualised assessment of the likely benefits and harms of statins.

The following groups of people should be assumed to be at high risk based on clinical history alone and do not require risk assessment with a scoring system:
- People who have had a previous cardiovascular event (angina, MI, stroke, transient ischaemic attack, or peripheral arterial disease)
- People with diabetes (type 1 or 2) over the age of 40 years
- People with familial hypercholesterolaemia

CVD risk should be estimated using information already contained in the patient’s records, and using a chart or computer programme based on epidemiologic data such as the US Framingham heart study data (Anderson, Odell, Wilson, & Kannel, 1991), according to:
- age;
- sex;
- lifetime smoking habit;
- blood pressure (if treated use pretreatment level);
- serum lipids if known. If treated use pretreatment level.

There are a number of different validated resources available to calculate 10-year risk of cardiovascular disease, but NICE now recommends the use of QRISK2 assessment tool.

Asymptomatic individuals should be considered at high risk if they are assessed as having a 10% or greater risk of a first cardiovascular event over 10 years. Such individuals warrant intervention with lifestyle changes and consideration for drug therapy, to reduce their absolute risk.

NICE now recommends that the QRISK2 tool is used up to and including age 84 years. Consider people aged 85 and older to be at increased risk of CVD because of their age alone.

A further consideration when assessing someone’s CVD risk is the presence of xanthelasmata. A 2011 study found that xanthelasmata predict an increased risk of CV events, particularly in women, even if serum lipid levels are normal (Christoffersen et al., 2011). The authors suggest xanthelasmata may be a sign of an increased propensity to deposit lipid in soft tissues and is therefore a cutaneous marker of atheroma. There is no such increased risk with arcus corneae.

Lifestyle Changes

Stop smoking. Someone who smokes 20 cigarettes a day or less and stops has a risk of CHD 10 years later almost the same as in one who has never smoked. Recovery is, however, less the longer the person has smoked (Doll, Peto, Boreham, & Sutherland, 2004). Nicotine replacement therapy increases the rate of quitting by 50% to 70%, regardless of the setting and independent of whether support, other than brief advice, is offered (Stead, Perera, Bullen, Mant, & Lancaster, 2008).

Alcohol. Keep alcohol intake within safe limits. Current UK recommendations are 14 units of alcohol or less per week for both men and women.

Take exercise. Encourage the patient to incorporate exercise into his or her daily life, rather than rely on visiting a gym or playing football at the weekend. Simple advice from the GP is unlikely to change behaviour. A more supportive programme is needed.

Manage social isolation. Depression and lack of social support are associated with an increased risk of cardiovascular disease (Bunker et al., 2003), as is the combination of high workload and low autonomy at work (Aboa-Eboule et al., 2007).

Control weight. Central obesity, rather than a raised BMI, carries the greater cardiovascular risk and can be assessed by measuring the waist circumference, with a single cutoff point for each sex. Men with a waist circumference
over 94 cm and women over 80 cm are likely to have other risk factors for cardiovascular disease, and men with a waist circumference over 102 cm (women over 88 cm) are 2.5 to 4.5 times as likely to have other major cardiovascular risk factors (Han et al., 1995). Small losses of weight are possible in primary care if advice on diet and exercise is accompanied by a behavioural programme.

- **Diet.** Encourage patients to:
  - reduce their meat and fat intake;
  - eat oily fish at least twice a week;
  - eat bread, pasta, and potatoes as sources of carbohydrate;
  - eat at least five portions a day of fruit or vegetables;
  - use olive oil and rape seed margarine instead of butter.

**Intensive Management When a 10-Year Cardiovascular Disease Risk of At Least 10% Is Detected**

**Lipid Lowering**

- NICE now recommends offering atorvastatin 20 mg for the primary prevention of CVD to people who have a 10% or greater 10-year risk of developing CVD
- Check TFTs, creatinine, LFTs, and fasting sugar. Note that elevated liver enzymes are not a contraindication to the use of a statin. They are not associated with subsequent statin-induced toxicity (Chalasani et al., 2004).
- Check baseline lipids. NICE no longer recommends a fasting test.
- NICE recommends annual medication reviews for patients and consideration of an annual, nonfasting, non-HDL cholesterol blood test to inform the discussion. Aim for a reduction of greater than 40% in non-HDL cholesterol in both primary and secondary prevention.
- Strongly encourage physical activity in all those taking statins. In combination, mortality risk is greatly reduced in those with dyslipidaemia (Kokkinos, Faselis, Myers, Panagiotakos, & Doumas, 2013).
- Warn the patient to report muscle pain or weakness; if present, check creatine kinase (CK) and TSH (Lasker & Chowdhury, 2012).
  - If CK is 10 times or above upper limit of normal then STOP immediately. This is a risk of rhabdomyolysis.
  - If CK is 5 times or below normal, this is rarely clinically significant and is often related to exercise.
  - If CK is not significantly raised, suggest rechallenging with a statin at a lower dose or switching statin (e.g., 10 mg atorvastatin is equivalent to 40 mg simvastatin). If myalgia recurs, try a nonstatin treatment (i.e., ezetimibe 10 mg).
- Continue indefinitely unless adverse effects occur.
- Be aware of interactions:
  - When macrolides (e.g., erythromycin and clarithromycin) have to be prescribed, stop the statin and restart 1 week after the macrolide is finished.
  - Avoid itraconazole and ketoconazole (contraindicated with simvastatin).
  - Reduce statin dose (max 20 mg simvastatin) with amiodipine, diltiazem, verapamil, and amiodarone.
  - On subsequent visits, check that the patient is taking the statin. A study from Liverpool found that a quarter of patients took their statin less than 80% of the time and had a higher mortality (Howell, Trotter, Mottram, & Rowe, 2004).

**Blood Pressure Control**

- Check blood pressure annually.
- Treat if levels are sustained at 140/90 mm Hg or above.
- Aim for a level of below 140/90 mm Hg, or in those with type 2 diabetes below 140/80 mm Hg (and 130/80 mm Hg if microalbuminuria or proteinuria is present).

**Talking to the Patient About Absolute Risks When Taking a Statin**

NICE have produced guidance on the components of good patient experience in adult NHS services, including recommendations on the communication of risk (NICE, 2014b). Shared decision making is particularly important in the context of CVD prevention, as the benefits to individual patients (of taking a statin, for instance) may be small and are not without the risk of harm. NICE have developed a patient decision aid which provides absolute benefits and harms of taking a statin (NICE, 2014c). Adequate time should be set aside for shared decision making using aids such as this. Discussions should be documented and can be re-visited in the future if patients change their minds.

**Secondary Prevention**

Consider the following measures in those with cardiovascular disease, diabetes, chronic kidney disease, or primary hyperlipidaemia.

- **Recommend lifestyle changes** as for primary prevention.

**Lipid-lowering measures:**

- Order the tests recommended under primary prevention and give the same warnings.
- Offer a statin regardless of baseline cholesterol level.
- Start atorvastatin 80 mg in people with CVD. Use a lower dose if there are potential drug interactions, high risk of adverse effects or patient preference.
- Recheck the serum lipids after 3 months. If TC ≥ 4 mmol/L or LDL ≥ 2 mmol/L consider intensifying treatment. If targets are still not reached, add ezetimibe. Other options are a fibrate (but with a warning about the increased risk of rhabdomyolysis), nicotinic acid or an omega-3 fatty acid.
- If adverse effects occur consider reducing the statin dose, or changing to another statin, or using ezetimibe, a fibrate, or nicotinic acid.
Familial Hypercholesterolaemia

GUIDELINE

- Familial hypercholesterolaemia (FH) is important because it carries a very high risk of premature cardiovascular morbidity and mortality. It is generally asymptomatic so is easily missed in general practice (Gill, Harnden, & Karpe, 2012).
- It is present in about 1 in 500 of the population in the United Kingdom and for men carries a 50% risk of a major coronary event by the age of 50. In women the risk is 30% by the age of 60. This risk is far higher than would be predicted from the cholesterol level alone and requires intensive therapy (high-dose statins and ezetimibe).
- Suspect FH as a possible diagnosis in adults with:
  - a total cholesterol level greater than 7.5 mmol/l and/or
  - a personal or family history of premature CHD (an event before 60 years in an index individual or first-degree relative).
- NICE recommends systematically searching primary care records for people:
  - younger than 30 years, with a total cholesterol concentration greater than 7.5 mmol/l and
  - 30 years of older, with a total cholesterol concentration greater than 9.0 mmol/l
- If suspicion of FH is raised:
  - Recheck the serum lipids with a fasting specimen.
  - Check for other causes of raised cholesterol (e.g., hypothyroidism, excess alcohol consumption).
  - Ask about the family history across three generations. Accept that the patient may need to return with these details after consultation with the family.
  - Ask about symptoms of CVD.
  - Examine for evidence of CVD or skin or tendon manifestations of hyperlipidaemia.
- Make a clinical diagnosis of FH according to the Simon Broome criteria:
  - FH exists if the total cholesterol is above 7.5 mmol/L or the LDL cholesterol is above 4.9 mmol/L AND tendon xanthoma are present (in the patient or a first or second degree relative).
  - FH is possible if:
    1. the total cholesterol is above 7.5 mmol/L or the LDL cholesterol is above 4.9 mmol/L AND there is a first degree relative with a myocardial infarction before the age of 60 or a second degree relative with a myocardial infarction before the age of 50; or
    2. the total cholesterol is above 7.5 mmol/L or the LDL cholesterol is above 4.9 mmol/L in the patient AND also in a first or second degree relative (or

Annual Workup at a Coronary Heart Disease Prevention Clinic

- **Blood pressure.** Treat if levels are sustained at 140/90 mm Hg or above. Aim for a level of below 140/90 mm Hg, or in those with diabetes below 130/80 mm Hg.
- **Antiplatelet therapy.** Recommendations for antiplatelet therapy vary in different scenarios. These are now largely specialist-led decisions.
- **ACE inhibition.** Prescribe an ACE inhibitor to all patients with coronary heart disease. Titrate to maximum tolerated doses and continue long term.
- **Prescribe a beta-blocker to all those who have had an MI.** Start them in patients whose MI was in the last 5 years and who therefore missed the opportunity to have them started in the acute stage. Patients at the highest risk benefit most from beta-blockade (e.g., those aged >50 with angina, hypertension, or heart failure).
- **Eplerenone.** This aldosterone antagonist is now preferred to spironolactone in patients who have LV dysfunction following MI.

- **Check:**
  1. fasting sugar;
  2. total cholesterol;
  3. urine protein;
  4. BP;
  5. weight, height, and BMI.
- **Action to be taken when a new abnormal result is found:**
  - **Fasting sugar:**
    1. If fasting sugar is 7 mmol/L or higher and the patient has symptoms (thirst, polyuria, lethargy), this is diabetes.
    2. If fasting sugar is 7 mmol/L or higher and the patient has no symptoms, repeat after 1 week. If still 7 mmol/L or higher, diabetes is confirmed.
    3. If fasting sugar is between 6.1 and 6.9 mmol/L inclusive, check blood sugar 2 hours after a 75-g glucose drink (=394 mL of the new Lucozade formulation which contains 73 kcal/100 mL):
      a. If greater than 11 mmol/L, diabetes is confirmed.
      b. If less than 7.8 mmol/L, this is impaired fasting glycaemia (IFG). Warn as for IGT (see the upcoming discussion).
      c. If between 7.8 and 11 mmol/L inclusive, this is impaired glucose tolerance (IGT). Warn that it carries an increased risk of diabetes, which can be reduced by exercise and diet. Recheck fasting sugar annually.
  - **Proteinuria.** If + or more, repeat after 1 week on the first morning specimen. If still + or more, send mid-stream urine for culture and sensitivities, serum creatinine and electrolytes, and an albumin to creatinine ratio (ACR) on a single urine sample. If the urine ACR is above 30 or there is haematuria, or the creatinine is raised, this is significant proteinuria (different thresholds apply in diabetes).
Referral is needed:
1. To a specialist with expertise in familial hypercholesterolaemia for all with a definite or possible clinical diagnosis. Further investigation will include DNA testing for relevant mutations and family screening;
2. To a cardiologist if there are symptoms of CHD. Consider referral if the patient has no symptoms but there is a family history of CHD in early adult life or the patient has two or more other risk factors for CHD.

Cholesterol lowering in a patient with a confirmed diagnosis of FH:
- Give a high-intensity statin (e.g., atorvastatin 40 mg daily)
- Aim for a LDL cholesterol over 50% below the pre-treated level
- If the target is not met, increase the statin to the maximum licensed dose, provided it is tolerated, and/or add ezetimibe.
- Re-refer if the target is not met.
- Review annually or sooner. Stress that lifestyle changes are even more important in someone with FH than in the general population.

References


