Drugs used in the prevention and management of hypertension, hyperlipidaemias and ischaemic heart disease

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Abstract
This is an A to Z list of drugs important in the care of ischaemic heart disease, hypertension and hyperlipidaemias. Their major mechanisms of action, the key pharmacokinetic principles essential for their safe use and important adverse effects are explained. Each class of drug is also given context for effective clinical use.

Keywords β-adrenoceptor blockers; angina pectoris; antiplatelet therapy; bempedoic acid; calcium channel blockers; fibrates; fibrinolytics; hypertension; inclisiran; lipid disorders; nitrates; sinus node inhibitor; statins; vasodilators

Key points

- Antihypertensive medications target three main mechanisms to lower blood pressure: the renin–angiotensin system, the autonomic nervous system and locally active mediators
- Oxygen demand to the myocardium is typically reduced by direct action on the heart reducing heart rate and contractility, by reducing preload by dilatation of the venous system, or by reducing afterload by decreasing arterial resistance
- Statins remain the cornerstone of low-density lipoprotein (LDL)-cholesterol reduction, with decreases in circulating LDL-cholesterol of up to 50%. Myalgia can be an issue. Statins are recommended for both primary and secondary prevention of cardiovascular disease
- Fibrinolytic agents are predominantly used in the acute management of ischaemic stroke when patients present early, and are still used in very rare circumstances in ST segment elevation myocardial infarction when there is no access to primary percutaneous coronary intervention
- Clopidogrel, prasugrel and ticagrelor bind irreversibly to P2 receptors on platelets, inhibiting platelet activation. They are used in acute coronary syndromes in conjunction with aspirin when specific criteria are met

Adenosine triphosphate citrate lyase (ACL) inhibitor

Mechanism: bempedoic acid inhibits ACL upstream of β-hydroxy β-methylglutaryl-coenzyme A (HMG-CoA) reductase. It also increases hepatic low-density lipoprotein (LDL) receptors, in turn decreasing circulating LDL. It is given in addition to other therapies when they are insufficient to produce adequate LDL reduction.[1]

Adverse effects: anaemia should be monitored, and there is a risk of gout and limb pain.

α-adrenoceptor antagonists

Mechanism: α-adrenoceptor antagonists act on postsynaptic α1-adrenoceptors to relax smooth muscle in arterioles and venous capacitance vessels. Examples include doxazosin and prazosin.

Adverse effects: postural hypotension from peripheral venous pooling can be troublesome. Headache, lethargy, dizziness, nausea and urinary frequency or incontinence can occur.

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β-adrenoceptor blockers

β-adrenoceptor blockers can be used in the management of hypertension and angina.

Mechanism: competitive antagonism of β-adrenoceptors reduces cardiac output and renin release. This results in vasodilation and a decrease in plasma volume.

β_1-adrenoceptor selective agents are 'cardioselective', but this selectivity decreases at higher doses. Non-selective drugs act on both β_1- and β_2-adrenoceptors. Pindolol is a partial agonist at β-adrenoceptors, resulting in less bradycardia and some peripheral vasodilation.

Vasodilation can also be produced by drugs with an antagonist action on α-adrenoceptors (labetalol, carvedilol) or by promoting endothelial nitric oxide production (e.g. nebivolol). This may be more beneficial in the management of hypertension.

Pharmacokinetics: lipophilic drugs (propranolol, metoprolol) are absorbed well by the gut and metabolized by the liver (to different extents in different individuals), so individual dosing is important to maximize the benefit. Modified-release (MR) preparations are preferred as the half-lives are short.

Hydrophilic drugs (e.g. atenolol) are not absorbed as well orally; however, they have more predictable plasma concentrations and longer half-lives. They are excreted unchanged in the urine.

Adverse effects:

- Acute left ventricular failure can occur with high doses in patients with impaired left ventricular function. They can also worsen intermittent claudication and Raynaud phenomenon. Excessive bradycardia can cause syncope.
- β_2-adrenoceptor antagonism can cause bronchospasm in people with asthma. This is less of an issue in chronic obstructive pulmonary disease as there is little reversibility in airway narrowing.
- Gluconeogenesis is reduced, which increases the risk of hypoglycaemia in people with insulin-dependent diabetes. They can also mask the symptoms associated with hypoglycaemia.
- β-adrenoceptor blockers can raise concentrations of triglycerides (triacylglycerols) and decrease high-density lipoproteins.
- Sudden withdrawal in chronic use leads to high catecholamine sensitivity causing tachycardia and palpitations, from an upregulation of β-adrenoceptors.
- Bradycardia and hypotension are well-known consequences of the concurrent use of β-blockers and non-dihydropyridine calcium channel blockers.[3]

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor antagonists

Mechanism: arterial vasodilation is caused by limiting the effects of angiotensin II on vascular smooth muscle and its ability to increase sympathetic tone. These drugs also decrease aldosterone production, which promotes renal salt and water loss.

For further details, see the article on drugs for heart failure and arrhythmias in *Medicine* 54:8).

Aspirin

Mechanism: irreversible inhibition of cyclooxygenase 1 in platelets prevents the generation of thromboxane A2 from arachidonic acid. It inhibits aggregation over the platelets' lifespan of 7–10 days. Loading doses achieve a rapid onset of platelet inhibition.

Adverse effects: dyspepsia, nausea, gastric ulceration and bleeding are the most common adverse effects.

Bile acid-binding resins

Mechanism: the resins colestyramine, colesevelam and colestipol limit cholesterol reabsorption from the gut. The liver compensates by removing LDL-cholesterol from the circulation to continue to maintain bile salt synthesis.[2]

Adverse effects: the unpalatable taste and texture of the medication can cause discontinuation. These drugs can also cause constipation. They interfere with digoxin, warfarin and levothyroxine absorption so those drugs should be taken 1 hour before or 4 hours after a resin.

Calcium channel blockers

Calcium channel blockers can be used in the management of hypertension and angina.

Mechanism: arterial vasodilation is achieved by blocking the influx of calcium via transmembrane L-type channels in the smooth muscle cells of resistance vessels; this reduces afterload and myocardial oxygen demand. When L-type channels in the myocardium are antagonized, there is a reduction in heart rate and contractility.[3]

The dihydropyridine group (amlodipine, nifedipine) act mainly on vascular smooth muscle, whereas the non-dihydropyridine (verapamil, diltiazem) group have important actions on the myocardium. These bind to different subunits on L-type channels. Non-dihydropyridines decrease exercise heart rate and can be effective monotherapy for angina.

Pharmacokinetics: MR preparations are preferred as most drugs in this class have a short half-life. Amlodipine has the longest half-life at 1–2 days (Table 1).

Adverse effects: dihydropyridines produce adverse effects such as flushing, headache, ankle oedema and reflex tachycardia. Adverse effects are reduced by using MR preparations. Non-dihydropyridines are negatively inotropic and can cause decompensation in heart failure. Their negatively chronotropic effects can cause significant bradycardia and they should not generally be used with β-adrenoceptor blockers.

Centrally acting antihypertensives

Selective imidazoline receptor agonist (moxonidine)

Mechanism: moxonidine stimulates imidazoline I_1 receptors in the medulla, decreasing sympathetic outflow and lowering blood pressure without reflex tachycardia. It has a long duration of action.

Adverse effects: dry mouth, nausea, fatigue, dizziness and headache can be seen.

Centrally acting α_2-adrenoceptor agonists (methyldopa, clonidine)

Mechanism: methyldopa is a pro-drug that is metabolized in the nerve terminal into a neurotransmitter analogue. Clonidine acts directly on the receptors. This decreases sympathetic outflow and increases vagal activity. Methyldopa is most often used in pregnancy.

Adverse effects: decreased sympathetic activity can cause postural or exertional hypotension. Ejaculatory failure can affect some men. Drowsiness is also reported.

Methyldopa causes a reversible positive Coombs test. Sudden withdrawal of clonidine can cause reflex tachycardia, sweating and anxiety.[3]

Clopidogrel, ticagrelor and prasugrel

Mechanism: these agents bind irreversibly to purinergic P2 receptors on platelets, reducing the binding of adenosine diphosphate (ADP), which is an initiator of platelet aggregation. Clopidogrel can be given as an alternative to aspirin when it is not tolerated, and is better at preventing strokes (Figure 1).

Pharmacokinetics (Table 2): ticagrelor is an active parent drug, whereas clopidogrel and prasugrel have active derivatives.

Calcium channel blockers

Drug	$t_{½}$ (hours)	Modified-release	Negative inotropic effect	Vasodilator	Bradycardia	Dose reduction	Pregnancy	Breastfeeding
Dihydropyridines								
Amlodipine	30–60	No	No	+++	No	L	?A (3)	A
Felodipine	12–25	Yes	No	+++	No	L	A	
Isradipine	2–6	No	+	+++	No	L	?A (3)	A
Lacidipine	7–8	No	+	+++	No	L	?A (3)	A
Lercanidipine	3–5	No	+	+++	No	L, R	A	A
Nicardipine	1–12	Yes	+	+++	No	L, R	?A (3)	A
Nifedipine	2–4	Yes	+	+++	No	L	?A (3)	
Non-dihydropyridines								
Diltiazem	2–5	Yes	++	++	Yes	L, R	A	?A
Verapamil	2–5	Yes	+++	+	Yes	L	A	

Modified-release: formulation available to prolong effect.
Negative inotropic effect: when present, avoid in heart failure.
Vasodilator: comparative degree of vasodilator action.
Bradycardia: reduces heart rate at rest and on exercise.
Dose reduction: reduce dose or avoid in liver (L) impairment, or reduce dose in renal (R) impairment.
Pregnancy: avoid (A), or avoid unless essential in third trimester (?A (3)) as it can inhibit labour.
Breastfeeding: manufacturer advises avoid (A) as no information is available, or avoid (?A) unless there is no suitable alternative.
$t_{½}$, plasma half-life.

Table 1

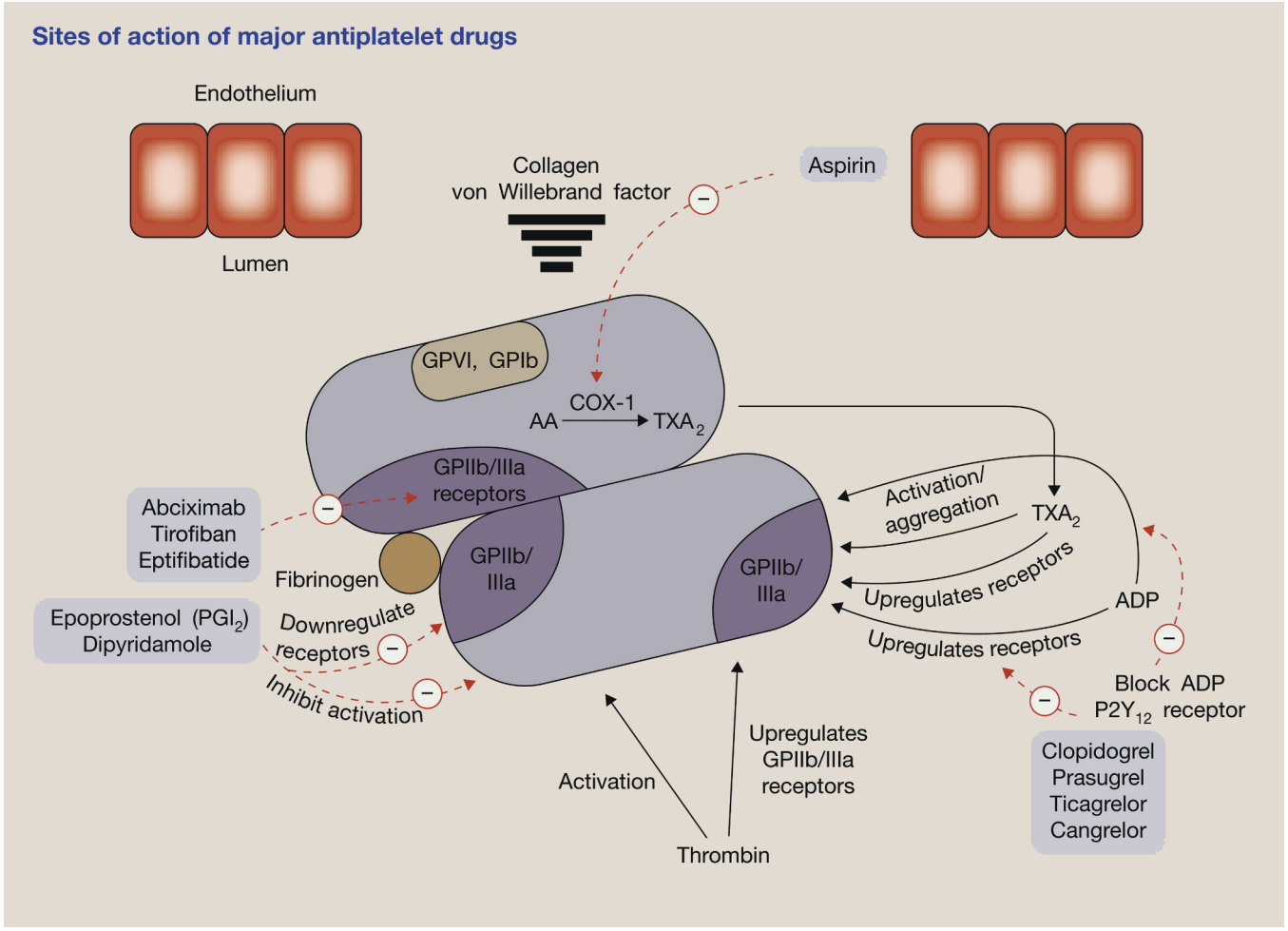

Figure 1 AA, arachidonic acid; COX, cyclooxygenase; PGI_2, prostacyclin; TXA_2, thromboxane A2. Reproduced from Waller DG, Sampson T. Medical pharmacology and therapeutics, 5th edn. Elsevier Health Sciences, 2017, with permission from Elsevier.

Oral antiplatelet drugs

Drug	$t_{½}$ (hours)	Dose reduction	Pregnancy	Breastfeeding
Aspirin	3–20	L, R	A (3)	A
Clopidogrel	5–8	L	A	A
Dipyridamole	12			C
Prasugrel	8 days	L	A	A
Ticagrelor	7	L	A	A

Dose reduction: avoid in severe liver (L) impairment; avoid in severe renal (R) impairment.
Pregnancy: avoid (A) or avoid in the third trimester (A (3)).
Breastfeeding: avoid (A); apply caution (C) and use only if benefits outweigh risks.

Table 2

Prasugrel has a more predictable activation and platelet inhibition. Clopidogrel has less reliable activation by the liver.

Adverse effects: there is a high risk of bleeding, particularly with prasugrel. Ticagrelor can cause breathlessness.

Dipyridamole

Mechanism: dipyridamole inhibits phosphodiesterase type 5, which in platelets increases intracellular concentrations of cyclic adenosine monophosphate (cAMP) and reduces the activation and expression of surface glycoprotein (GP) IIb/IIIa receptors. This reduces platelet aggregation (Figure 1).

Adverse effects: include gastrointestinal upset, myalgia, dizziness, headache, flushing, hypotension and tachycardia.

Diuretics

Thiazide diuretics

Mechanism: these inhibit Na^+/Cl^- co-transporters in the distal convoluted tubule and early collecting duct.

Adverse effects: hyponatraemia and hypokalaemia can occur; renal function should be checked within 2 weeks of starting treatment and less frequently thereafter. Prolonged hypokalaemia can lead to impaired glucose tolerance by inhibiting insulin release. Thiazide diuretics also increase plasma lipids, linked to increased dose.

Loop diuretics

Loop diuretics are discussed in more detail in the article on drugs for heart failure and arrhythmias in *Medicine* 54:8. They have a short duration of action so are not first-line choices for hypertension. They can be useful if volume expansion is contributing to hypertension.

Potassium-sparing diuretics

Potassium-sparing diuretics are discussed in more detail in the article on drugs for heart failure and arrhythmias in *Medicine* 54:8. Spironolactone is most useful to treat hyperaldosteronism and resistant hypertension.

Ezetimibe

Mechanism: a specific cholesterol absorption inhibitor acting in the small intestine, ezetimibe can reduce plasma cholesterol by 15% and LDL cholesterol by 20%. There are data to suggest that it reduces cardiovascular events when taken with a statin after an acute coronary syndrome.

Adverse effects: it can produce diarrhoea, headache and angioedema.

Fibrates

Mechanism: fibrates activate the gene transcription factor PPAR-α, which encodes proteins that control lipoprotein metabolism. Fibrates increase fatty acid uptake in the liver, heart and skeletal muscle, where they are altered to limit their availability for triglyceride synthesis. As a result, plasma triglycerides fall by 50%, and circulating high-density lipoprotein (HDL) increases by up to 20%.[2]

Adverse effects: gastrointestinal disturbance, rashes, pruritus, dizziness and headache can occur. Myalgia and myositis are uncommon but more likely in combination with statins or in renal impairment.

Fibrinolytics

Mechanism: alteplase is a recombinant tissue-type plasminogen activator (rt-PA) and tenecteplase is a modified rt-PA. They increase activated plasminogen, which is converted into the enzyme plasmin, which in turn degrades fibrin.

Pharmacokinetics: fibrinolytics are given intravenously or occasionally intra-arterially. Tenecteplase is given as a bolus. Alteplase is given as an infusion and should be followed by a heparin infusion to prevent vessel re-occlusion. The half life is 30–60 minutes.

Adverse effects: severe haemorrhage occurs in 1% of treatments and can be reversed with fresh frozen plasma or antifibrinolytic drugs. Hypotension can occur, in which case the infusion must be stopped.

GPIIb/IIIa receptor antagonists

Mechanism: abciximab is a human chimera monoclonal antibody to GPIIb/IIIa receptors. It binds irreversibly to GPIIb/IIIa receptors and blocks fibrinogen binding, reducing platelet aggregation by >90%. It is given intravenously with heparin and

aspirin to prevent ischaemic complications during percutaneous coronary intervention.

Tirofiban and eptifibatide bind reversibly to and block GPIIb/IIIa receptors. They can also be given intravenously with heparin and aspirin to prevent early myocardial infarction in individuals with unstable angina or non-ST elevation myocardial infarction.

Adverse effects: there is a higher risk of bleeding in individuals who are elderly or of low body weight; risk can be reduced by adjusting the dose to the body weight. Thrombocytopenia also occurs. Abciximab can cause nausea, vomiting, hypotension, bradycardia and headache.

Hydralazine

Mechanism: smooth muscle relaxation causes arterial vasodilation. Smooth muscle relaxation is thought to be caused by activation of guanylate cyclase raising intracellular guanosine monophosphate. Hydralazine is used in the treatment of pre-eclampsia.

Adverse effects: individuals who metabolize hydralazine slowly (slow acetylators) have a higher risk of dose-related systemic lupus erythematosus-type syndrome. This is reversible on drug withdrawal. Vasodilatory effects such as headache, dizziness and flushing can also occur. Sympathetic activation can cause reflex tachycardia.

Inclisiran

Mechanism: this is a small interfering ribonucleic acid that inhibits PCSK9 gene expression in the liver. The result is an increase in LDL receptor expression and increased LDL uptake from the plasma. Plasma concentrations are reduced by 30–50%. It is administered by subcutaneous injection every 6 months.[4]

Adverse effects: include musculoskeletal pain, diarrhoea and hypertension.

Ivabradine

Mechanism: this acts on the sinoatrial node, with inhibition of sodium and potassium influx through f-channels in diastole delaying the spontaneous depolarization of these cells. It has no effect on myocardial contractility. Reduced heart rate can be of benefit in heart failure.

Adverse effects: f-channels are found in the eye, so visual symptoms can occur at higher doses; these resolve on stopping the drug. Headache, dizziness and ventricular ectopics are reported. Stopping the drug can reverse any bradycardia, and ivabradine is not recommended if the heart rate is <60 beats per minute.

Minoxidil

Mechanism: minoxidil opens adenosine triphosphate (ATP)-sensitive potassium channels in vascular smooth muscle, hyperpolarizing the cell membrane and closing voltage-gated calcium channels. This causes smooth muscle relaxation and vasodilation.

Adverse effects: hirsutism restricts the use of minoxidil to men. Activation of the renin–angiotensin–aldosterone system can lead to salt and water retention, resulting in peripheral oedema. Minoxidil is a powerful vasodilator and can cause flushing, headache and reflex tachycardia.

Concurrent use of an ACE inhibitor or β-blockers and loop diuretics is required to minimize the adverse effects.

Nicorandil

Mechanism: this opens ATP-sensitive potassium channels, hyperpolarizing the cell membrane and inhibiting the opening of voltage-dependent calcium channels. This relaxes the vascular smooth muscle. It also causes nitric oxide production leading to venodilatation.

Adverse effects: headache is seen in up to 50% of patients. Dizziness and mouth and anal ulcers also occur.[5]

Nicotinic acid and derivatives

Mechanism: nicotinic acid and its derivative acipimox act to reduce lipolysis and the availability of free fatty acids, limiting triglyceride and very-low-density lipoprotein (VLDL) synthesis. Nicotinic acid can reduce plasma triglycerides by 35% and LDL by 15%, and increases HDL by 25%. A randomized control trial failed to show a reduction in myocardial infarction or stroke in patients taking nicotinic acid.[2]

Adverse effects: cutaneous vasodilation with flushing and itching can occur; however, symptoms can be reduced by gradually increasing the dose or taking the drug with food or with low-dose aspirin. It can cause glucose intolerance at higher doses. Hepatotoxicity requires monitoring. Gastrointestinal upset, headache and dizziness are also frequent.

Nitrates

Mechanism: the metabolites of glyceryl trinitrate (GTN) and isosorbide mononitrate (ISMN) release nitric oxide. This activates enzymes in the vascular endothelium to reduce the availability of calcium to vascular smooth muscle, therefore dilating venous capacitance vessels, large systemic arteries and coronary arteries. This decreases cardiac preload and afterload, and increases blood supply to an ischaemic myocardium when there is coronary artery vasoconstriction.

Pharmacokinetics: GTN is rapidly absorbed by the gut but metabolized into an inactive substance by first-pass metabolism. GTN works within 1 minute to relieve angina and provides prophylaxis for up to 30 minutes.

Buccal tablets show a slower release with longer duration of action. Transdermal patches can deliver the drug at a rate to maintain stable concentrations in the blood. Intravenous infusion is short-acting but can be titrated against relief of pain when treating unstable angina.

ISMN is absorbed orally and does not undergo first-pass metabolism; therefore it provides a predictable and prolonged response.

Adverse effects: venodilatation can cause postural hypotension, dizziness, syncope and reflex tachycardia. Arterial dilatation can

cause headache and flushing. Tolerance to therapeutic effects can be managed with a 'nitrate-low' period each day, achieved by asymmetrical dosing. Hypotension can be aggravated by phosphodiesterase inhibitors.[5]

ω-3 ethyl esters

Mechanism: these are long-chain polyunsaturated acids, which are substrates for the enzymes that produce triglycerides. They competitively block triglyceride synthesis, resulting in decreased circulating triglycerides and VLDL. However, they increase both HDL and LDL.

They can also decrease plasma fibrinogen, limiting thrombogenesis, and alter prostanoid synthesis in platelets, which inhibits platelet aggregation. In addition, they can enhance nitric oxide-mediated vasodilatation and have an antiarrhythmic action.

Adverse effects: gastrointestinal disturbance and prolonged bleeding time occur.

PCSK9 inhibitors

Mechanism: evolocumab and alirocumab are subcutaneously injected monoclonal antibodies. They inhibit the enzyme PCSK9, which degrades LDL receptor expression on liver cell surfaces. This increases LDL expression in the liver and decreases circulating LDL by 45–70%. Data show a reduction in cardiovascular events when used with a statin. They are currently recommended as primary or secondary prevention in familial hypercholesterolaemia and as an addition to statin therapy in individuals with a very high risk of cardiovascular disease.

Pharmacokinetics: PCSK9 inhibitors have a half-life of 11–20 days and are therefore injected every 2 weeks.

Adverse effects: irritation and pain occur at site of infection. Nasopharyngitis, arthralgia and back pain are also seen.

Ranolazine

Mechanism: ranolazine blocks late sodium transit into myocytes, leading to a decrease in intracellular calcium. This decreases diastolic wall tension, decreasing oxygen demand, and improves blood flow to the intramyocardial blood vessels. It is effective in ischaemic myocardium.

Adverse effects: gastrointestinal disturbance, lethargy, headache and dizziness are reported. Prolonged QT segments can occur, but are not associated with an increase in arrhythmias. Electrocardiographic monitoring is advised if ranolazine taken with other QT-prolonging medicines.

Statins (Table 3)

Mechanism: inhibition of HMG-CoA reductase catalyses the rate-limiting step in hepatic cholesterol synthesis. Reduced intrahepatic cholesterol results in hepatic cholesterol uptake so circulating LDL falls. A modest rise in HDL of 5% is expected.

Drugs for lipid disorders

Drug	$t_{½}$ (hours)	Dose reduction	Pregnancy	Breastfeeding
Statins				
Atorvastatin	30	L	A	A
Fluvastatin	1	L	A	A
Pravastatin	1	L, R	A	A
Rosuvastatin	20	L, R	A	A
Simvastatin	2	L, R	A	A
Fibrates				
Bezafibrate	1–5	L, R	A	A
Ciprofibrate	27–28	L, R	A	A
Fenofibrate	20–27	L, R	A	A
Gemfibrozil	1–2	L, R	A	A
Resins/absorption inhibitors				
Colesevelam	–		C	C
Colestipol	–		C	C
Colestyramine	–		C	C
Ezetimibe	22	L	A	A
Nicotinic acid				
Acipimox	1	R	A	A
Nicotinic acid	1–2	L	A	A

Dose reduction: avoid in active or severe liver (L) impairment; reduce dose or avoid in renal (R) impairment.
Pregnancy: avoid (A); use with caution (C) as it can cause fat-soluble vitamin deficiency.
Breastfeeding: manufacturer advises avoid (A); use with caution (C) as it can cause fat-soluble vitamin deficiency.
$t_{½}$, plasma half-life.

Table 3

Statins also stabilize atherosclerotic plaques and decrease inflammatory cell infiltrates.[2]

Adverse effects: liver function tests can be abnormal in the first few weeks. A rise to >3 times the upper limit of normal should lead to withdrawal. Myalgia, myositis and rhabdomyolysis are important adverse effects. These are more common when taken with fibrates or nicotinic acid. Low-dose intermittent rosuvastatin can be effective in those experiencing muscle symptoms. ◆

KEY REFERENCES

1 Ray KK, Bays HE, Catapano AL, et al. Safety and efficacy of bempedoic acid to reduce LDL cholesterol. *N Engl J Med* 2019; **380:** 1022–32.

2 Cholesterol Treatment Trialists' collaboration. Efficacy and safety of LDL lowering therapy among men and women: meta-analysis of individual data from 174,000 participants in 27 randomised trials. *Lancet* 2015; **385:** 1397–405.

3 Poultry NR, Prabhakaran D, Caulfield M. Hypertension. *Lancet* 2015; **386:** 801–12.

4 Ray KK, Wright RS, Kallend D, et al. Two phase 3 trials of inclisiran in patients with elevated LDL cholesterol. *N Engl J Med* 2020; **382:** 1507–722.

5 Husted SE, Ohman EM. Pharmacological and emerging therapies in the treatment of chronic angina. *Lancet* 2015; **386:** 691–701.

FURTHER READING

Sheppard JP, Martin U, McManus RJ. Diagnosis and management of resistant hypertension. *Heart* 2017; **103:** 1295–302.

Reed GW, Rossie JE, Cannon CP. Acute myocardial infarction. *Lancet* 2017; **389:** 197–210.

Pavasini R, Camici PG, Crea F, et al. Anti-anginal drugs: systematic review and clinical implications. *Int J Cardiol* 2018; **283:** 55–63.

Pfisterer ME, Zellweger MJ, Gersh BJ. Management of stable coronary artery disease. *Lancet* 2010; **375:** 763–72.

Sabatine MS, Giugliano RP, Keech AC, et al. Evolocumab and clinical outcomes in patients with cardiovascular disease. *N Engl J Med* 2017; **376:** 1713–22.

TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 68-year-old man presented with muscle aches. He had ischaemic heart disease, hypertension and diabetes. He was taking aspirin, atorvastatin, metformin and ramipril. His creatinine kinase concentrations were normal. The atorvastatin was stopped and after 2 weeks the aches improved.

What is the next-step drug to trial in his lipid management?

A. Evolocumab (PCSK9 inhibitor)
B. Fenofibrate (fibrate)
C. Intermittent low-dose rosuvastatin
D. Nicotinic acid
E. ω-3 ethyl ester

Question 2

A 59-year-old woman presented with resistant hypertension. She was taking amlodipine, candesartan and indapamide at maximum doses.

On clinical examination, her blood pressure was 151/94 mmHg (matching her home monitoring results) and her heart rate was 59 beats/minute. There was some mild ankle swelling. Renal function was normal. She was provided with extensive lifestyle advice.

Which additional antihypertensive medication is most likely to help improve her blood pressure?

A. Hydralazine
B. Minoxidil
C. Moxonidine
D. Nebivolol
E. Spironolactone

Question 3

A 74-year-old man presented with a 6-month history of chest tightness radiating into the left arm on exertion, and resolving with rest, when walking his dog.

On clinical examination, he looked well. His blood pressure was 151/89 mmHg and heart rate 58 beats/minute. Heart sounds were normal. He was booked for an outpatient CT coronary angiogram.

In addition to aspirin and GTN spray what medication should be added?

A. Amlodipine
B. Bisoprolol
C. Diltiazem
D. Ivabradine
E. Nicorandil