Hypertension: GP guide to recommended treatment

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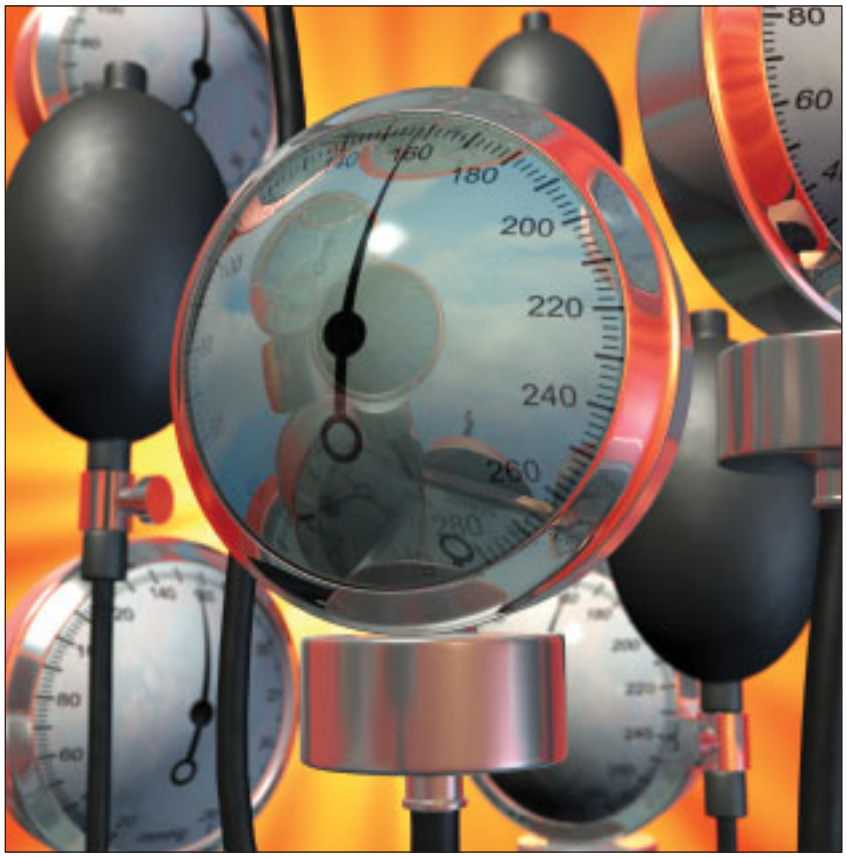

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Our Drug review of hypertension discusses the properties of the wide range of antihypertensive agents available and their recommended use, both alone and in combination, followed by sources of further information in Resources.

The management of hypertension is a long-term undertaking, often in asymptomatic people. The objective is to reduce cardiovascular morbidity without impairing quality of life.

A wide range of antihypertensive drugs is available. The proportional reduction in CHD events and stroke for a given reduction in blood pressure, an approximate halving of risk for each 10mmHg diastolic reduction, is similar to that expected from observational studies.[1,2] If differences in blood pressure lowering are taken into consideration, the five main classes of antihypertensive drugs – thiazides, calcium-channel blockers (CCBs), ACE inhibitors, angiotensin-II receptor blockers (ARBs) and beta-blockers – are similarly effective in preventing CHD and stroke.[1]

Contemporary blood pressure targets are rigorous, particularly in those with diabetes, chronic kidney disease or evidence of cardiovascular damage. To achieve these targets, individuals often require more than one drug and most will require two or more in combination. The different classes of antihypertensive drugs at standard doses, or the same multiple of standard doses, lower blood pressure to a similar extent.

Combining drugs from different classes has an additive effect on blood pressure reduction while the dose-response of an individual drug is modest.[3] Overall, combining drugs has an incremental effect much greater than can be achieved with dose titration and is associated with fewer side-effects.

The initial challenge is to select the therapy most appropriate for the individual to be treated. To facilitate the best therapeutic regimens in the majority of people with hypertension, *ie* those with uncomplicated

hypertension, the British Hypertension Society (BHS) in collaboration with the National Institute for Health and Clinical Excellence (NICE) has proposed a simple ACD algorithm (see Figure 1).[4] This is based on the observation that younger Caucasians (aged <55 years) tend to have elevated plasma renin activity and respond well to drugs that block the renin angiotensin system, *ie* ACE inhibitors or ARBs (A drugs), while older Caucasians (aged ≥55 years) and people of Afro-Caribbean origin have a low renin form of hypertension and respond preferentially to CCBs or diuretics (C or D drugs). Thereafter, drugs are combined in a logical manner as necessary to achieve blood pressure targets.

Many people with hypertension have concomitant conditions. A sound starting point in such patients is consideration of drug therapy in terms of compelling indications, possible indications, possible contraindications or compelling contraindications (see Table 1).[5] Thus, although beta-blockers are no longer recommended as first-line therapy in uncomplicated hypertension (see below), these drugs have compelling indications in angina and post-MI and are useful in tachyarrhythmias such as atrial fibrillation. In contrast A drugs (ACE inhibitors or ARBs) are contraindicated in young women planning to become pregnant.

Thiazide and thiazide-like diuretics

These drugs have formed the cornerstone of the management of hypertension for several decades. Control of hypertension may be difficult without the use of a diuretic.

Thiazide and thiazide-like diuretics, *eg* indapamide, act on the nephron mainly in the peripheral part of the distal tubule to increase sodium excretion. The evidence base for these drugs in the management of hypertension is strong. Thiazide or thiazide-like diuretics were used in most of the studies that established the benefit of treating high blood pressure, albeit at doses far in excess of those currently recommended.[6]

Antihypertensive effects are seen at low doses with little additional blood pressure lowering from higher doses when used as monotherapy. At higher doses, metabolic side-effects are much more marked. Current recommendations are to use low-dose thiazide or thiazide-like diuretic regimens.

Side-effects

These are mainly metabolic (see Table 2). Efforts should be made to avoid hypokalaemia; a potassium-sparing diuretic can be co-administered – potassium supplements lack effect and are difficult to take.

Indications

Compelling indications include older people, isolated systolic hypertension, heart failure and secondary stroke prevention. Although a history of gout is a compelling contraindication, thiazide or thiazide-like diuretics may sometimes be necessary to control blood pressure in people with gout, ideally in combination with allopurinol. While traditionally avoided in patients with diabetes, effectiveness in blood pressure lowering outweighs any adverse effect.

Calcium-channel blockers

CCBs are among the most widely used drugs in cardiovascular medicine with roles not only in hypertension but also in angina and (for some CCBs) tachyarrhythmias. Large-scale prospective outcome studies have demonstrated beneficial cardiovascular protection with CCBs at least equivalent to other antihypertensive agents.[1]

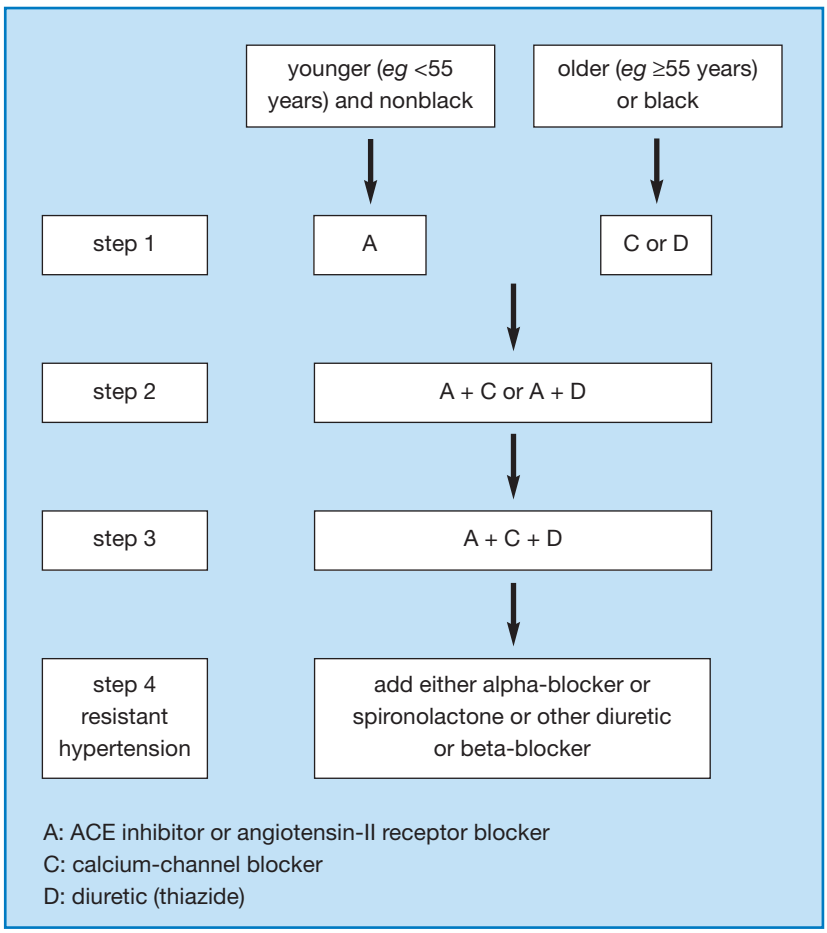

Figure 1. BHS/NICE recommendations for managing hypertension[4]

Class of drug	Compelling indications	Possible indications	Caution	Compelling contraindications
Alpha-blockers	benign prostatic hypertrophy		postural hypotension, heart failure	urinary incontinence
ACE inhibitors	heart failure, left ventricular dysfunction, post-MI or established CHD, type 1 diabetic nephropathy, 2° stroke prevention	chronic renal disease, type 2 diabetic nephropathy, proteinuric renal disease	renal impairment, PVD	pregnancy, reno-vascular disease
ARBs	ACE inhibitor intolerance, type 2 diabetic nephropathy, hypertension with LVH, heart failure in ACE-intolerant patients, post-MI	LV dysfunction post-MI, intolerance of other anti-hypertensive drugs, proteinuric renal disease, heart failure	renal impairment, PVD	pregnancy, reno-vascular disease
Beta-blockers	MI, angina	heart failure	heart failure, PVD, diabetes (except with CHD)	asthma/COPD, heart block
Calcium-channel blockers: dihydopyridine	older people, ISH	older people, angina		
rate limiting	angina	MI	combination with beta-blockade	heart block, heart failure
Thiazide-like diuretics	older people, ISH, heart failure, 2° stroke prevention			gout

Table 1. Compelling and possible indications and contraindications for major antihypertensive agents (BHS IV)

CCBs promote vasodilator activity (and reduce blood pressure) by reducing calcium influx into vascular smooth muscle cells by interfering with voltage-operated calcium channels (and to a lesser extent receptor-operated channels) in the cell membrane.

Interference with intracellular calcium influx is also important in cardiac muscle, cardiac conduction tissue and gastrointestinal smooth muscle. In cardiac tissues, CCBs have potential for negative inotropic, chronotropic and dromotropic activity while the gastrointestinal effects predispose to constipation.

These effects vary with different agents according to ability to penetrate cardiac and other tissues, relative affinity for calcium channels in different tissues and the influence of reflux cardiac stimulation secondary to peripheral vasodilatation.

Although often considered as a single class, CCBs can be subdivided according to structural and functional distinctions.

- dihydropyridine derivatives, *eg* amlodipine, lercanidipine (Zanidip), nifedipine
- phenylalkylamine: verapamil
- benzothiazepine derivative: diltiazem.

Dihydropyridine derivatives have pronounced peripheral vasodilator properties and intense reflex cardiac stimulation overcomes any direct cardiac effects. Verapamil and diltiazem are also vasodilators but the balance of actions is such that these drugs

have noticeable cardiac effects including reduced heart rate (rate-limiting CCBs).

Side-effects (see Table 3)
Headache, flushing and tachycardia are seen particularly with rapid-onset and short-acting agents. Such side-effects usually decline with time and are less common with rate-limiting CCBs.

Ankle oedema and gum hypertrophy are long-term effects. These can arise many months after treatment initiation and may be more common with long-acting agents. Earlier formulations of some dihydropyridines, such as nifedipine capsules, have a rapid onset of action, unpredictable effects on blood pressure and are accompanied by reflex tachycardia and activation of the renin-angiotensin system. Angina can be precipitated. These formulations have no place in the management of hypertension, even in the emergency setting. Long-acting dihydropyridine drugs, such as amlodipine, or other CCBs in once-daily formulations are preferred.

Indications
Compelling indications include hypertension in older people, isolated systolic hypertension (dihydropyridines) and angina (rate-limiting CCBs). Possible indications include angina (dihydropyridines) and post-MI (rate-limiting CCBs). Caution is required with concomitant rate-limiting CCBs and beta-blockers since diltiazem and verapamil share many properties with beta-blockers, *eg* AV blockade; fatalities have been reported with such combinations. Compelling contraindications for rate-limiting CCBs are heart block and heart failure.

ACE inhibitors

These drugs competitively inhibit the activity of angiotensin-converting enzyme (ACE) to prevent formation of the active octapeptide angiotensin II from angiotensin I, the inactive decapeptide. This occurs in blood and tissues including kidney, heart, blood vessels, adrenal gland and brain. Angiotensin II is a potent vasoconstrictor, promotes aldosterone release, facilities sympathetic activity and has other potentially harmful effects on the cardiovascular system.

Reduction in blood pressure secondary to vasodilatation following ACE inhibition is greatest when the renin-angiotensin system is stimulated, *eg* following diuretic therapy or in renal artery stenosis, but ACE inhibitors also lower blood pressure when there is normal or low activity of the renin-angiotensin system. Inhibition of ACE also leads to accumulation of kinins including bradykinin, which promotes vasodilator activity and may contribute to the overall effectiveness of ACE inhibitors.

Properties
- peripheral vasodilatation
- reduced cardiac contractility

Side-effects
- Dihydropyridines
  - headache and flushing due to peripheral vasodilatation
  - tachycardia and palpitation secondary to reflex activation of the sympathetic nervous system
  - swelling of ankles and occasionally hands due to disturbance of haemodynamics of microcirculation (preferential precapillary arteriolar vasodilatation)
  - gum hypertrophy
- Rate-limiting CCBs
  - bradycardia and atrioventricular conduction delay due to direct cardiac effects
  - constipation with verapamil

Table 3. Properties and side-effects of calcium-channel blockers

Large-scale prospective outcome trials have demonstrated benefits in cardiovascular protection with ACE inhibitors equivalent to those with other antihypertensive drugs.[1] Furthermore, cardiovascular and renal protective effects beyond that to be expected from blood pressure reduction have been reported in various groups of patients at high risk.[7]

Side-effects (see Table 4)
ACE inhibitors are well tolerated. Dry cough is a class effect that is not improved by a change of ACE inhibitor. Although common, this side-effect is rarely intolerable for the individual but may trouble close associates and alternatives are available; withdrawal for cough should be a considered decision, not a reflex response.

Impairment of renal function may arise in patients with bilateral renal artery stenosis, which should be suspected if there is evidence of peripheral vascular disease. Renal failure, reversible on discontinuation of the ACE inhibitor, may be precipitated. However, ACE inhibitors can protect renal function in patients with chronic renal failure and hypertension.

To avoid the risks of abrupt reduction in renal function and hyperkalaemia, serum creatinine and potassium should be measured before and soon after (within two weeks) of starting an ACE inhibitor. Only if serum potassium rises above the reference range or serum creatinine rises by more than 20 per cent

Properties • act in distal convoluted tubule to increase sodium excretion and urine volume • maximum effect at low doses
Side-effects • hypokalaemia due to urinary potassium loss; fall in serum potassium >0.3mmol per litre with low-dose thiazide or thiazide-like diuretics raises suspicion of primary hyperaldosteronism if serum sodium is in high-normal range: *refer for investigation* • hyperuricaemia due to interference with renal clearance of uric acid: *risk of acute gout* • hyperglycaemia possibly related to hypokalaemia: *risk of new-onset diabetes* • hypercalcaemia due to reduced renal clearance of calcium • erectile dysfunction by an unknown mechanism • thrombocytopenia and skin rashes: *rare*

Table 2. Properties and side-effects of thiazide and thiazide-like diuretics

(and is above the reference range) need the ACE inhibitor be discontinued.

ACE inhibitors should be avoided in women of child-bearing potential because of the danger of fetal renal maldevelopment. The risk of new-onset diabetes appears to be less with ACE inhibitors than with many other classes of antihypertensive drugs.

Indications

Compelling indications include heart failure, left ventricular dysfunction, post-MI or established CHD, type 1 diabetic nephropathy and secondary stroke prevention in combination with a thiazide-like diuretic. Possible indications include chronic renal disease (with caution, close supervision and specialist advice when there is established and significant renal impairment), type 2 diabetic nephropathy and proteinuric renal disease. Caution is advised if there is renal impairment or peripheral vascular disease because of the association with renovascular disease.

Compelling contraindications are pregnancy and renovascular disease, although ACE inhibitors are sometimes used in renovascular disease under specialist supervision.

Angiotensin-II receptor blockers

ARBs have haemodynamic properties similar to those of ACE inhibitors but are better tolerated. ARBs antagonise the action of angiotensin II in a highly selective manner at the angiotensin II AT_1 receptor which mediates all the classical effects of angiotensin II, *eg* vasoconstriction, aldosterone release, sympathetic activation and other potentially harmful effects on the cardiovascular system.

Large-scale prospective outcome trials have demonstrated benefits in cardiovascular protection with ARBs equivalent to those with ACE inhibitors and other antihypertensive drugs.[1] ARBs may also offer particular benefits in patients with type 2 diabetes complicated by hypertension and nephropathy, and in heart failure.[7]

Side-effects (see Table 5)

ARBs are reported to have a side-effect profile difficult to distinguish from that of placebo in clinical trials but adverse effects can occur rarely. These are similar to those with ACE inhibitors, but cough is not a feature since bradykinin metabolism is not inhibited.

Indications

Compelling indications include ACE inhibitor intolerance, type 2 diabetic nephropathy, hypertension with left ventricular hypertrophy, heart failure in ACE inhibitor-intolerant patients and post-MI infarction. Possible indications include left ventricular dysfunction post-MI infarction, intolerance of other antihypertensive drugs, proteinuric renal disease, chronic renal disease and heart failure. ARBs may be beneficial in chronic renal failure but should only be used with caution, close supervision and specialist advice when there is established and significant renal impairment.

Caution is advised in renal impairment and in peripheral vascular disease because of the association with renovascular disease. Compelling contraindications are pregnancy and renovascular disease, although ARBs are sometimes used in patients with renovascular disease under specialist supervision.

Alpha-blockers

Alpha-blockers are usually used as add-on drugs in difficult-to-treat hypertension or when other drugs are poorly tolerated.[5] Commonly used alpha-blockers act selectively at postganglionic $alpha_1$-receptors. Selective blockade of peripheral $alpha_1$-receptors leads to vasodilatation and hence reduction in blood pressure.

All alpha-blockers exhibit the phenomenon of tachyphylaxis, *ie* loss of the antihypertensive effect of low doses, *eg* doxazosin 1-2mg daily, within one to two weeks, overcome by increasing the dose. The maintenance dose of doxazosin should be at least 4mg daily.

Alpha-blockers have a favourable effect on atherogenic lipid profiles suggesting particular benefits in cardiovascular protection. This was not confirmed in the one outcome trial in which doxazosin was used as a first-line option.[8] Monotherapy with alpha-blockers may be associated with an increased risk of heart failure.

Side-effects (see Table 6)
These drugs are not particularly well tolerated. The main adverse effect of serious concern is first-dose postural hypotension. Although this is less of a problem with doxazosin, profound hypotension can occur when higher doses of the standard formulation, *eg* doxazosin 4mg daily, are initiated instead of the sustained-release formulation (Cardura XL). Noncardiac effects include an action on the bladder neck and alpha-blockers are used to treat benign prostatic hypertrophy, but this action can predispose to urinary incontinence, particularly in women.

Indications
The only compelling indication for alpha-blockers is benign prostatic hypertrophy. These drugs should be used with caution in patients with a history of postural hypotension or heart failure. A compelling contraindication is urinary incontinence.

Properties • vasodilatation • reduction in circulating angiotensin-II and aldosterone
Side-effects • dry, irritant cough in about 15% (10% men and 20% women) attributable to accumulation of bradykinin: *not dose related and can occur with low dose* • angioedema also attributable to kinin potentiation: *rare but potentially fatal* • hyperkalaemia due to potassium retention mediated by reduction of aldosterone: *rare except in renal impairment* • first-dose hypotension if renin-angiotensin system activated: *rare in essential hypertension* • impairment of renal function: caution if bilateral renal artery stenosis suspected • taste disturbance: *rare* • skin rashes: *very rare*

Table 4. Properties and side-effects of ACE inhibitors

Beta-blockers

Beta-blockers have been used widely in the management of angina and certain tachyarrhythmias as well as in hypertension. The mode of action in lowering blood pressure remains controversial. Conventionally, the antihypertensive action of beta-blockers is attributed to cardiac effects (decreased heart rate and cardiac output). However, long-term reductions in blood pressure appear greater in individuals with high renin forms of hypertension, suggesting that renal actions are important.

There is substantial evidence for outcome benefits with beta-blockers in the management of heart failure and in patients post-MI infarction.[1] Beta-blockers were included in the therapeutic regimens that established the benefits of treating hypertension.[6] However, recent data indicate that treatment based on beta-blockers is inferior to that based on other antihypertensives in cardiovascular protection likely related to blood pressure-lowering efficacy.[9]

This evidence, together with the increased risk of new-onset diabetes associated with these drugs, especially in combination with diuretics,[10] has resulted in the recommendation that beta-blockers are no longer appropriate first-line agents, except where there are compelling indications.

Beta-blockers may be considered as a first-line option in women of child-bearing potential because of concern about fetal renal maldevelopment with ACE inhibitors or ARBs.

Many beta-blockers have ancillary properties that influence choice in hypertensive individuals.

Selectivity Since the desired effects of beta-blockers are mediated by blockade of $beta_1$-receptors that predominate on the heart, 'cardioselective' agents with relative selectivity for this receptor are generally preferred. However, receptor selectivity is not absolute and is lost at high doses. Examples of 'cardioselective' beta-blockers include atenolol, bisoprolol and metoprolol. Beta-blockers with selectivity rates >50, *eg* bisoprolol and nebivolol (Nebilet), are least likely to lose selectivity at high doses.

Partial agonist activity (intrinsic sympathomimetic activity) This manifests as a beta-stimulant effect when background adrenergic activity is low, *eg* during sleep, but beta-blockade occurs when adrenergic activity is increased, *eg* during exercise. Beta-blockers with partial agonist activity include pindolol (Visken).

Other properties Some beta-blockers also block effects mediated at peripheral alpha receptors, *eg* carvedilol and labetalol, stimulate $beta_2$-receptors, *eg* celiprolol, or have direct vasodilator activity, *eg* nebivolol, but this does not necessarily confer greater blood pressure reduction in clinical practice.

Properties • vasodilatation • reduction of circulating aldosterone
Side-effects • hyperkalaemia due to potassium retention mediated by reduction of aldosterone: *rare except in renal impairment* • impairment of renal function: *caution if bilateral renal artery stenosis suspected* • dizziness and syncope: *rare but may be precipitated by volume depletion* • angioedema: *very rare*

Table 5. Properties and side-effects of ARBs

Side-effects (see Table 7)
Most adverse reactions and contraindications are predictable on the basis of blockade of beta-receptors. In most people, blockade of $beta_2$-receptors in the lungs is unimportant, but clearly matters in asthma. Likewise, beta-receptors cause peripheral arterial vasodilatation and, if blocked, vasoconstriction results. Worsening of peripheral vascular flow is usually only important in critical ischaemia.

Indications
Compelling indications are MI and angina, and beta-blockers are also useful in tachyarrhythmias, especially atrial fibrillation. Possible indications include stable heart failure, when initial low dose and careful titration are paramount. Caution is advised in unstable heart failure, peripheral vascular disease, diabetes (except those with CHD), with concomitant rate-limiting CCBs (diltiazem and verapamil) and with thiazide or thiazide-like diuretics (unless there is a compelling indication). Compelling contraindications are asthma, COPD with significant reversibility and heart block.

Other oral antihypertensive agents

Clonidine
This agent acts as an $alpha_2$-agonist in the brain stem to reduce sympathetic outflow. Clonidine can cause sedation and drowsiness, dry mouth and sexual dysfunction in men. In addition, this agent is associated with rapid rebound in hypertension on abrupt cessation of high doses, presumably as a consequence of receptor up-regulation.

Methyldopa
Methyldopa acts primarily via a metabolite as a relatively specific $alpha_2$-agonist. Methyldopa has a side-effect profile similar to that of clonidine, although rebound hypertension is not seen. However, methyldopa can give rise to immunological side-effects, including pyrexia, hepatitis and, rarely, haemolytic anaemia.

Moxonidine
Moxonidine binds preferentially to and acts as an agonist at imidazoline binding sites. It has better tolerability than that of earlier agents but dry mouth remains a significant problem. Moxonidine may have a role when diuretics, CCBs, ACE inhibitors (or ARBs), alpha-blockers and beta-blockers are not appropriate or have failed to control blood pressure.

Hydralazine
Hydralazine acts on vascular smooth muscle largely at arteriolar level. The vasodilatation causes a reflex tachycardia and therefore hydralazine is used with a rate-slowing agent such as a beta-blocker. Hydralazine can also cause fluid retention and a diuretic is often also required. The combination of hydralazine, beta-blocker and diuretic can be very effective. In higher doses used over the long term, hydralazine can cause a lupus-like syndrome. Doses over 100mg daily should be avoided.

Minoxidil (Loniten)
Minoxidil is a potent vasodilator that, like hydralazine, causes fluid retention and tachycardia. It is sometimes used as a last resort but not in females because excessive bodily hair growth can be extremely pronounced and unsightly. Large doses of furosemide are required to control oedema in some patients and a beta-blocker may be needed to control any unwanted tachycardia.

Properties • vasodilatation
Side-effects • first-dose postural hypotension associated with reflex cardioacceleration and palpitation: *less common with doxazosin* • vasovagal syncope after first dose if unable to mount a rapid heart rate response to hypotension: *caution, particularly with terazosin* • headache • oedema • stress incontinence in women • drowsiness with indoramin

Table 6. Properties and side-effects of alpha-blockers

Spironolactone

Although not licensed for the treatment of hypertension in the UK, spironolactone is recommended as a step 4 option in the BHS/NICE guidance (see Figure 1). Spironolactone is a direct aldosterone antagonist that can cause painful gynaecomastia and, at higher doses, particularly if used with an ACE inhibitor or ARB, hyperkalaemia. The current recommended dose is 25 or 50mg daily.

Furosemide

Loop diuretics such as furosemide have a limited role in the management of hypertension. Furosemide is useful as an alternative to a thiazide or thiazide-like diuretic if renal function is impaired (serum creatinine >160µmol per litre) and in refractory hypertension, where high doses (≥40mg twice daily) may be needed.

Aliskiren (Rasilez)

Aliskiren is the first of a new class of drugs, the direct renin inhibitors, to be licensed for the treatment of hypertension. This drug may be considered as an additional option for step 4 therapy. In patients receiving optimal diuretic therapy, or known to have high plasma renin, escalation of renin angiotensin system blockade is the logical next step.

Conclusions

Globally, elevated blood pressure is responsible for seven million deaths every year.[11] Control of blood pressure by rational use of antihypertensive drugs can eliminate much of the burden of cardiovascular morbidity and mortality.[1] With the availability of effective and well-tolerated antihypertensive agents the landscape has already changed. Deaths from hypertensive heart failure are now exceedingly rare, while improved blood pressure control has contributed importantly to the declining rates of stroke and MI in the UK. Yet more can be achieved by close adherence to the recommended treatment guidelines.

Properties • decreased heart rate and reduced contractility • reduction in renin release from kidneys
Side-effects • bronchospasm in susceptible individuals due to blockade of beta$_2$-receptors that mediate dilation in the bronchi: *asthma is an absolute contraindication for all beta-blockers* • bradycardia and impairment of myocardial contractility: *common but seldom symptomatic* • peripheral vasoconstriction due to reduced cardiac output and possibly blockade of beta$_2$-receptors that subserve vasodilatation in blood vessels supplying skeletal muscle beds, typically resulting in cold hands and feet and possibly exacerbation of Raynaud's phenomenon: *mainly nonselective agents* • CNS effects due to reduced sympathetic outflow, *eg* malaise, vivid dreams, nightmares and, rarely, hallucinations with highly lipid-soluble beta-blockers, which have greater penetration into the CNS • tiredness and fatigue due to reduced cardiac output exacerbated by blockade of beta$_2$-receptors in skeletal muscle associated with increased muscle activity: *mainly nonselective agents* • masking of hypoglycaemia in insulin-dependent diabetes because of blunting of sympathetic nervous activation: *mainly nonselective agents* • hyperglycaemia: *risk of new-onset diabetes*

Table 7. Properties and side-effects of beta-blockers

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Resources

Further reading

Better blood pressure control: how to combine drugs. Brown MJ, *et al. J Hum Hypertens* 2003;17: 81-6.

BMJ collected resources: http://bmjjournals.com/cgi/collection/hypertension. All articles published in the *BMJ* on hypertension since January 1998.

Hypertension: management of hypertension in adults in primary care. National Institute for Health and Clinical Excellence. Clinical guideline 34 (partial update of NICE clinical guideline 18). NICE, 2006.

Groups and organisations

Blood Pressure Association, 60 Cranmer Terrace, London SW17 0QS. Tel: 020 8772 4994, website: www.bpassoc.org.uk. Provides literature about hypertension for patients and GPs.

British Heart Foundation, Greater London House, 180 Hampstead Road, London NW1 7AW. Tel: 020 7554 0000, website: www.bhf.org.uk. Charity providing information on all aspects of heart disease, including hypertension, for patients and health professionals.

British Hypertension Society. BHS Information Service: Jackie Howarth, BHS Administrative Officer, Clinical Sciences Building, Level 5, Leicester Royal Infirmary, PO Box 65, Leicester LE2 7LX. Tel: 07717 467 973, website: www.bhsoc.org. Provides information on hypertension for health professionals. A scientific meeting is held annually.

High Blood Pressure Foundation, Department of Medical Sciences, Western General Hospital, Edinburgh EH4 2XU. Tel: 0131 332 9211, website: www.hbpf.org.uk. Charity dedicated to improving the basic understanding and public awareness of high blood pressure.