HYPERTENSION

Aetiology

In more than 95% of cases, a specific underlying cause of hypertension cannot be found. Such patients are said to have essential hypertension. There is very little evidence that 'stress' causes hypertension. Sphygmomanometry, particularly when performed by a doctor, can cause an unrepresentative surge in BP which has been termed 'white coat' hypertension, and as many as 20% of patients with apparent hypertension in the clinic may have a 'normal BP' when it is recorded by automated devices used in their own home.
Home or ambulatory BP measurements may be particularly helpful in patients with unusually labile blood pressure, those with refractory hypertension, those who may be experiencing symptomatic hypotension, and those in whom white coat hypertension is suspected.

In about 5% of unselected cases, hypertension can be shown to be a consequence of a specific disease or abnormality leading to sodium retention and/or peripheral vasoconstriction (secondary hypertension

CAUSES OF SECONDARY HYPERTENSION

Alcohol

Obesity

Pregnancy (pre-eclampsia)

Renal disease

- Renal vascular disease
- Parenchymal renal disease, particularly glomerulonephritis
- Polycystic kidney disease

Endocrine disease

- Phaeochromocytoma
- Cushing's syndrome
- Primary hyperaldosteronism (Conn's syndrome)
- Hyperparathyroidism
- Acromegaly
- Primary hypothyroidism
- Thyrotoxicosis
- Congenital adrenal hyperplasia due to 11-β-hydroxylase or 17-hydroxylase deficiency

Drugs

- e.g. Oral contraceptives containing oestrogens, anabolic steroids, corticosteroids, non-steroidal anti-inflammatory drugs

Coarctation of the aorta

MEASUREMENT OF BLOOD PRESSURE

- Use a machine that has been validated, well maintained and properly calibrated
- Measure sitting BP routinely, with additional standing BP in elderly and diabetic patients and those with possible postural hypotension
- Remove tight clothing from the arm
- Support the arm at the level of the heart
- Use a cuff of appropriate size (the bladder must encompass > two-thirds of the arm)
- Lower the mercury slowly (2 mm per second)
- Read the BP to the nearest 2 mmHg
- Use phase V (disappearance of sounds) to measure diastolic BP
- Take two measurements at each visit

DEFINITION OF HYPERTENSION

Category	**Systolic blood pressure (mmHg)**	**Diastolic blood pressure (mmHg)**
Blood pressure		
Optimal	< 120	< 80
Normal	< 130	< 85
High normal	130-139	85-89
Hypertension		
Grade 1 (mild)	140-159	90-99
Grade 2 (moderate)	160-179	100-109
Grade 3 (severe)	≥180	≥110
Isolated systolic hypertension		
Grade 1	140-159	< 90
Grade 2	≥160	< 90

History

Family history, lifestyle (exercise, salt intake, smoking habit) and other risk factors should be recorded. A careful history will also identify those patients with drug- or alcohol-induced hypertension and may elicit the symptoms of other causes of secondary hypertension such as phaeochromocytoma (paroxysmal headache, palpitation and sweating) or complications such as coronary artery disease (e.g. angina, breathlessness).

Radio-femoral delay (coarctation of the aorta, enlarged kidneys (polycystic kidney disease), abdominal bruits (renal artery stenosis) and the characteristic facies and habitus of Cushing's syndrome are all examples of physical signs that may help to identify one of the causes of secondary hypertension Examination may also reveal features of important risk factors such as central obesity and hyperlipidaemia (tendon xanthomas etc.). Nevertheless, the majority of abnormal signs are due to the complications of hypertension.

Non-specific findings may include left ventricular hypertrophy (apical heave), accentuation of the aortic component of the second heart sound, and a fourth heart sound. The optic fundi are often abnormal and there may be evidence of generalised atheroma or specific complications such as aortic aneurysm or peripheral vascular disease.

Target organ damage

The adverse effects of hypertension principally involve the blood vessels, central nervous system, retina, heart and kidneys, and can often be detected clinically.

Blood vessels:

In larger arteries (over 1 mm in diameter) the vessels dilate and become tortuous and their walls become less compliant. In smaller arteries (under 1 mm) hyaline arteriosclerosis occurs in the wall, the lumen narrows and aneurysms may develop. Widespread atheroma develops and may lead to coronary and/or cerebrovascular disease, particularly if other risk factors (e.g. smoking, hyperlipidaemia, diabetes) are present.

These structural changes in the vasculature often perpetuate and aggravate hypertension by increasing peripheral vascular resistance and reducing renal function.

Hypertension is also implicated in the pathogenesis of aortic aneurysm and aortic dissection.

Central nervous system :

Stroke is a common complication of hypertension and may be due to cerebral haemorrhage or cerebral infarction. Carotid atheroma and transient cerebral ischaemic attacks are more common in hypertensive patients. Subarachnoid haemorrhage is also associated with hypertension.

Hypertensive encephalopathy is a rare condition characterised by high blood pressure and neurological symptoms, including transient disturbances of speech or vision, paraesthesiae, disorientation, fits and loss of consciousness. Papilloedema is common, the neurological deficit is usually reversible if the hypertension is properly controlled.

Retina :

The optic fundi reveal a gradation of changes linked to the severity of hypertension; fundoscopy can, therefore, provide an indication of the arteriolar damage occurring elsewhere

'Cotton wool' exudates are associated with retinal ischaemia or infarction, and fade in a few weeks (. 'Hard' exudates (small, white, dense deposits of lipid) and microaneurysms ('dot' haemorrhages) are more characteristic of diabetic retinopathy).

Hypertension is also associated with central retinal vein thrombosis

Heart :

High blood pressure places a pressure load on the heart and may lead to left ventricular hypertrophy with a forceful apex beat and fourth heart sound. ECG or echocardiographic evidence of left ventricular hypertrophy is highly predictive of cardiovascular complications and therefore particularly useful in risk assessment.

Atrial fibrillation is common and may be due to diastolic dysfunction caused by left ventricular hypertrophy or the effects of coronary artery disease.

Severe hypertension can cause left ventricular failure in the absence of coronary artery disease, particularly when renal function, and therefore sodium excretion, is impaired.

Kidneys :

Long-standing hypertension may cause proteinuria and progressive renal failure by damaging the renal vasculature.

'Malignant' or 'accelerated' phase hypertension :

This rare condition may complicate hypertension of any aetiology and is characterised by accelerated microvascular damage with necrosis in the walls of small arteries and arterioles ('fibrinoid necrosis') and by intravascular thrombosis. The diagnosis is based on evidence of high blood pressure and rapidly progressive end organ damage such as retinopathy (grade 3 or 4), renal dysfunction (especially proteinuria) and/or hypertensive encephalopathy. Left ventricular failure may occur and, if this is untreated, death occurs within months.

Investigations

All hypertensive patients should undergo a limited number of investigations. Additional investigations are appropriate in selected patients

HYPERTENSION: INVESTIGATION OF ALL PATIENTS

- Urinalysis for blood, protein and glucose
- Blood urea, electrolytes and creatinine
    - **N.B.** Hypokalaemic alkalosis may indicate primary hyperaldosteronism but is usually due to diuretic therapy
- Blood glucose
- Serum total and high-density lipoprotein (HDL) cholesterol
- 12-lead ECG (left ventricular hypertrophy, coronary artery disease)

HYPERTENSION: INVESTIGATION OF SELECTED PATIENTS

- Chest X-ray: to detect cardiomegaly, heart failure, coarctation of the aorta
- Ambulatory BP recording: to assess borderline or 'white coat' hypertension
- Echocardiogram: to detect or quantify left ventricular hypertrophy
- Renal ultrasound: to detect possible renal disease
- Renal angiography: to detect or confirm presence of renal artery stenosis

The sole objective of antihypertensive therapy is to reduce the incidence of adverse cardiovascular events, particularly coronary heart disease, stroke and heart failure. The relative benefit of antihypertensive therapy (approximately 30% reduction in risk of stroke and 20% reduction in risk of coronary heart disease- is similar in all patient groups, so the absolute benefit (total number of events prevented) of treatment is greatest in those at highest risk. Systolic blood pressure and diastolic blood pressure are both powerful predictors of cardiovascular risk.

Patients with diabetes or cardiovascular disease are at particularly high risk and the threshold for initiating antihypertensive therapy is therefore lower (≥ 140/90) in these patient groups. the optimum blood pressure for reduction of major cardiovascular events was found to be 139/83 mmHg, and even lower in patients with diabetes; moreover, reducing blood pressure below this level caused no harm. The thresholds for treatment in the elderly are the same as for younger patients

Patients taking antihypertensive therapy require follow-up, typically at 3-month intervals, to monitor blood pressure, minimise side-effects and reinforce lifestyle advice.

Non-drug therapy :

Appropriate lifestyle measures may obviate the need for drug therapy in patients with borderline hypertension, reduce the dose and/or the number of drugs required in patients with established hypertension, and directly reduce cardiovascular risk.

Correcting obesity, reducing alcohol intake, restricting salt intake can all lower blood pressure.

Antihypertensive drugs

- *Thiazide :* More potent loop diuretics, such as furosemide 40 mg daily or bumetanide 1 mg daily, have few advantages over thiazides in the treatment of hypertension unless there is substantial renal impairment or they are used in conjunction with an ACE inhibitor.
- *Beta-adrenoceptor antagonists (β-blockers)*. Metoprolol (100-200 mg daily), atenolol (50-100 mg daily) and bisoprolol (5-10 mg daily) are cardioselective and therefore preferentially block the cardiac β_1-adrenoceptors
- *Carvedilol.* (6.25-25 mg 12-hourly) are combined β- and α-adrenoceptor antagonists which issometimes more effective than pure β-blockers.
- *Angiotensin-converting enzyme (ACE) inhibitors*. These drugs (e.g. enalapril 20 mg daily, ramipril 5-10 mg daily or lisinopril 10-40 mg daily) inhibit the conversion of angiotensin I to angiotensin II and are usually well tolerated. They should be used with particular care in patients with impaired renal function or renal artery stenosis because they can reduce the filtration pressure in the glomeruli and precipitate renal failure. Electrolytes and creatinine should be checked before and 1-2 weeks after commencing therapy. Side-effects include first-dose hypotension, cough, rash, hyperkalaemia and renal dysfunction.
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- *Angiotensin receptor blockers*. These drugs (e.g. losartan 50-100 mg daily, valsartan 40-160 mg daily) block the angiotensin II type I receptor and have similar effects to ACE inhibitors but do not

[1] In heart failure when used as monotherapy.
[2] ACE inhibitors or angiotensin II receptor blockers may be beneficial in chronic renal failure and those with renovascular disease but should only be used with caution, close supervision and specialist advice when there is established and significant renal impairment.
[4] In combination with a thiazide or thiazide-like diuretic.
[3] Caution with ACE inhibitors and angiotensin II receptor blockers in peripheral vascular disease because of association with renovascular disease.
[5] Beta-blockers are used increasingly to treat stable heart failure but may worsen acute heart failure.

The emergency treatment of accelerated phase or malignant hypertension :

In accelerated phase hypertension, it is unwise to lower blood pressure too quickly because this may compromise tissue perfusion (due to altered autoregulation) and can cause cerebral damage, including occipital blindness, and precipitate coronary or renal insufficiency. Even in the presence of cardiac failure or hypertensive encephalopathy, a controlled reduction, to a level of about 150/90 mmHg, over a period of 24-48 hours is ideal.

In most patients it is possible to avoid parenteral therapy and bring blood pressure under control with bed rest and oral drug therapy . Intravenous glyceryl trinitrate (0.6-1.2 mg/hour), intramuscular hydralazine (5 or 10 mg aliquots repeated at half-hourly intervals) are effective remedies .
Refractory hypertension:
The common causes of treatment failure in hypertension are non-adherence with drug therapy, inadequate therapy, and failure to recognise an underlying cause such as renal artery stenosis or phaeochromocytoma; of these, the first is by far the most prevalent. There is no easy solution to compliance problems, but simple treatment regimens, attempts to improve rapport with the patient and careful supervision may all help.