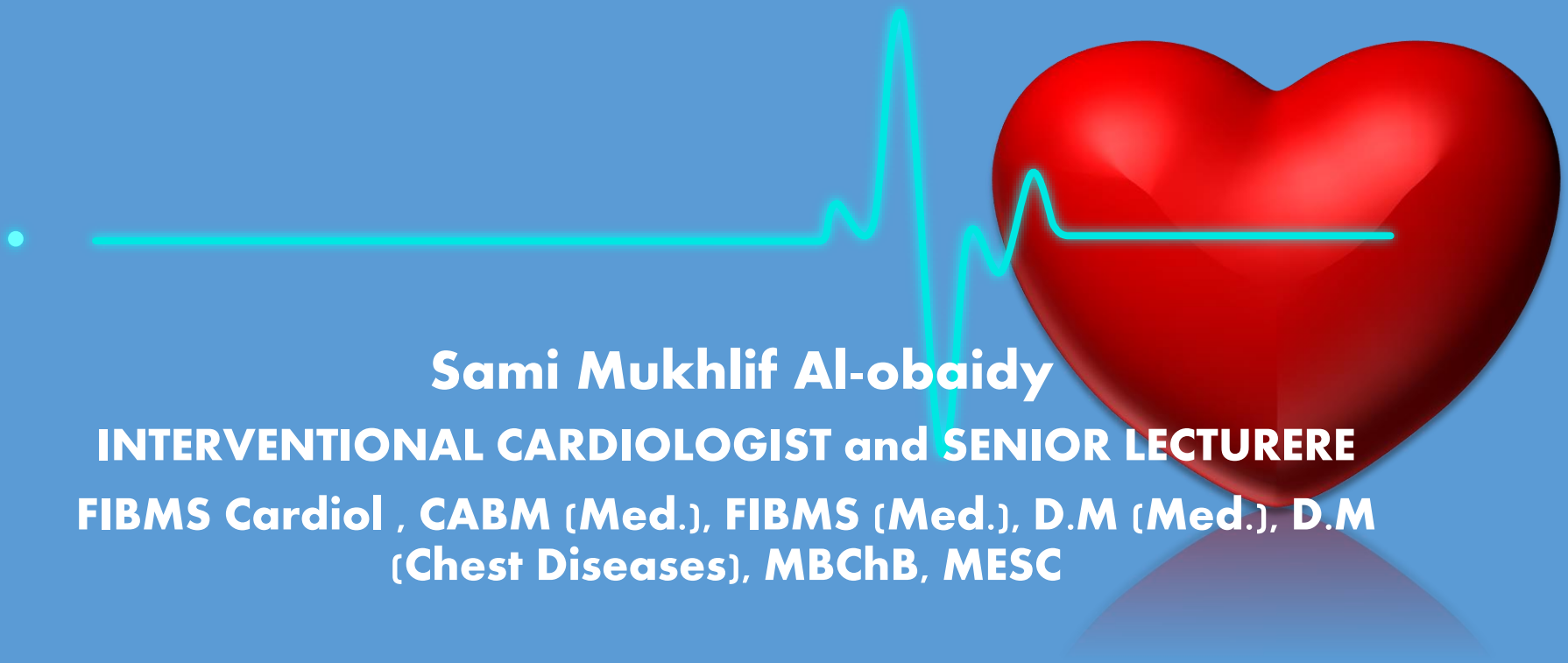
VALVULAR HEART DISEASES



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HYPERTENSION

The risk of cardiovascular diseases such as stroke and CAD is closely related to levels of BP.

The diagnosis of hypertension is therefore made when systolic and diastolic values rise above a specific threshold that corresponds to the level of BP at which the risk of cardiovascular complications and benefits of treatment outweigh the treatment costs and potential side effects of therapy.
The British Hypertension Society classification, provided defines mild hypertension as existing when the BP is above 140/90 mmHg. Similar thresholds have been published by the European Society of Hypertension and the WHO–International Society of Hypertension.

The cardiovascular risks associated with high BP depend on the combination of risk factors in an individual, such as age, gender, weight, physical activity, smoking, family history, serum cholesterol, diabetes mellitus and pre-existing vascular disease.

16.64 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

Pathogenesis

Many factors may contribute to the regulation of BP and the development of hypertension, including renal dysfunction, peripheral resistance, vessel tone, endothelial dysfunction, autonomic tone, insulin resistance and neurohumoral factors.

In more than 95% of cases, however, no specific underlying cause of hypertension can be found. Such patients are said to have essential hypertension. Hypertension is more common in some ethnic groups, particularly African Americans and Japanese, and approximately 40–60% is explained by genetic factors.

Age is a strong risk factor in all ethnic groups. Important environmental factors include a high salt intake, heavy consumption of alcohol, obesity and lack of exercise. Impaired intrauterine growth and low birth weight are associated with an increased risk of hypertension later in life. In about 5% of cases, hypertension is secondary to a specific disease.

16.65 Causes of secondary hypertension

Alcohol

Obesity

Pregnancy

Renal disease

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

Endocrine disease

- Phaeochromocytoma
- Cushing's syndrome
- Primary hyperaldosteronism (Conn's syndrome)
- Glucocorticoid-suppressible hyperaldosteronism
- Hyperparathyroidism
- Acromegaly
- Primary hypothyroidism
- Thyrotoxicosis
- Congenital adrenal hyperplasia due to 11β-hydroxylase or 17α-hydroxylase deficiency
- Liddle's syndrome (p. 361)
- 11β-hydroxysteroid dehydrogenase deficiency

Drugs

Coarctation of the aorta

Pathogenesis

Hypertension has a number of adverse effects on the cardiovascular system. In larger arteries (> 1 mm in diameter), the internal elastic lamina is thickened, smooth muscle is hypertrophied and fibrous tissue is deposited. The vessels dilate and become tortuous, and their walls become less compliant.

In smaller arteries (< 1 mm), hyaline arteriosclerosis occurs in the wall, the lumen narrows and aneurysms may develop. Widespread atheroma develops and may lead to coronary and cerebrovascular disease, particularly if other risk factors are present.

These structural changes in the vasculature often perpetuate and aggravate hypertension by increasing peripheral vascular resistance and reducing renal blood flow, thereby activating the renin–angiotensin–aldosterone axis.

CLINICAL FEATURES:

Hypertension is usually asymptomatic until the diagnosis is made at a routine physical examination or when a complication arises.

Reflecting this fact, a BP check is advisable every 5 years in adults over 40 years of age to pick up occult hypertension.

Can give hint of aetiology: Radio-femoral delay in patients with coarctation of the aorta , enlarged kidneys in patients with polycystic kidney

Disease, abdominal bruits that may suggest renal artery Stenosis, and the characteristic facies and habitus of Cushing's syndrome.

Examination may also reveal evidence of risk factors for hypertension, such as central obesity and hyperlipidaemia.

Other signs may be observed that are due to the complications of hypertension. These include signs of left ventricular hypertrophy, accentuation of the aortic component of the second heart sound, and a fourth heart sound.

AF is common and may be due to diastolic dysfunction caused by left ventricular hypertrophy or the effects of CAD.

Severe hypertension can cause left ventricular failure in the absence of CAD, particularly when there is an impairment of renal function. The optic fundi are often abnormal and there may be evidence of generalised atheroma or specific complications, such as aortic aneurysm, PAD or stroke.

Examination of the optic fundi reveals 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 .

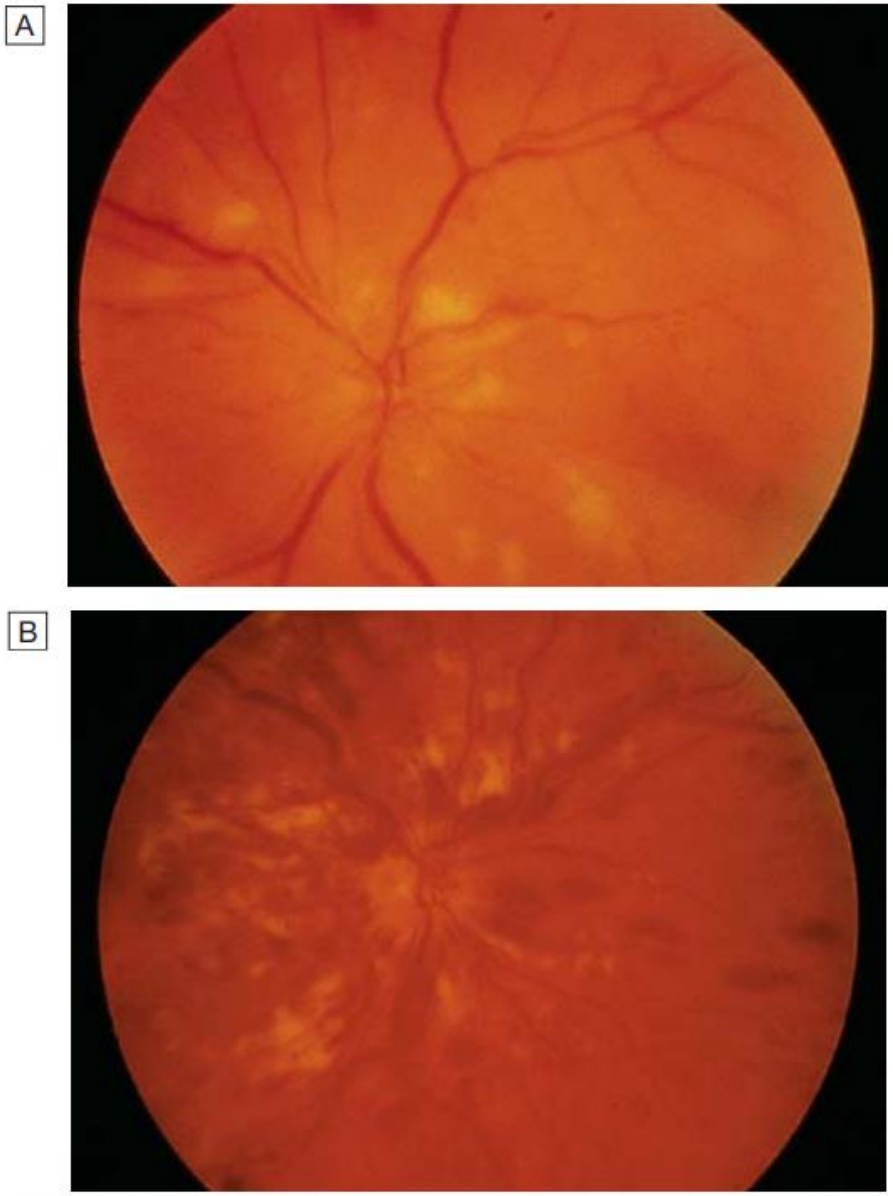


Fig. 16.76 Retinal changes in hypertension. A Grade 4 hypertensive retinopathy showing swollen optic disc, retinal haemorrhages and multiple cotton wool spots (infarcts). B Central retinal vein thrombosis showing swollen optic disc and widespread fundal haemorrhage, commonly associated with systemic hypertension. *A and B, Courtesy of Dr B. Cullen.*

16.66 Hypertensive retinopathy

Grade 1

- Arteriolar thickening, tortuosity and increased reflectiveness ('silver wiring')

Grade 2

- Grade 1 plus constriction of veins at arterial crossings ('arteriovenous nipping')

Grade 3

- Grade 2 plus evidence of retinal ischaemia (flame-shaped or blot haemorrhages and 'cotton wool' exudates)

Grade 4

- Grade 3 plus papilloedema

Investigations

A decision to embark on antihypertensive therapy effectively commits the patient to life-long treatment, so readings must be as accurate as possible. The objectives are to:

• confirm the diagnosis by obtaining accurate, representative BP measurements

• identify contributory factors and any underlying causes

• assess other risk factors and quantify cardiovascular risk

• detect any complications that are already present

• identify comorbidity that may influence the choice of antihypertensive therapy.

16.68 Investigation of hypertension

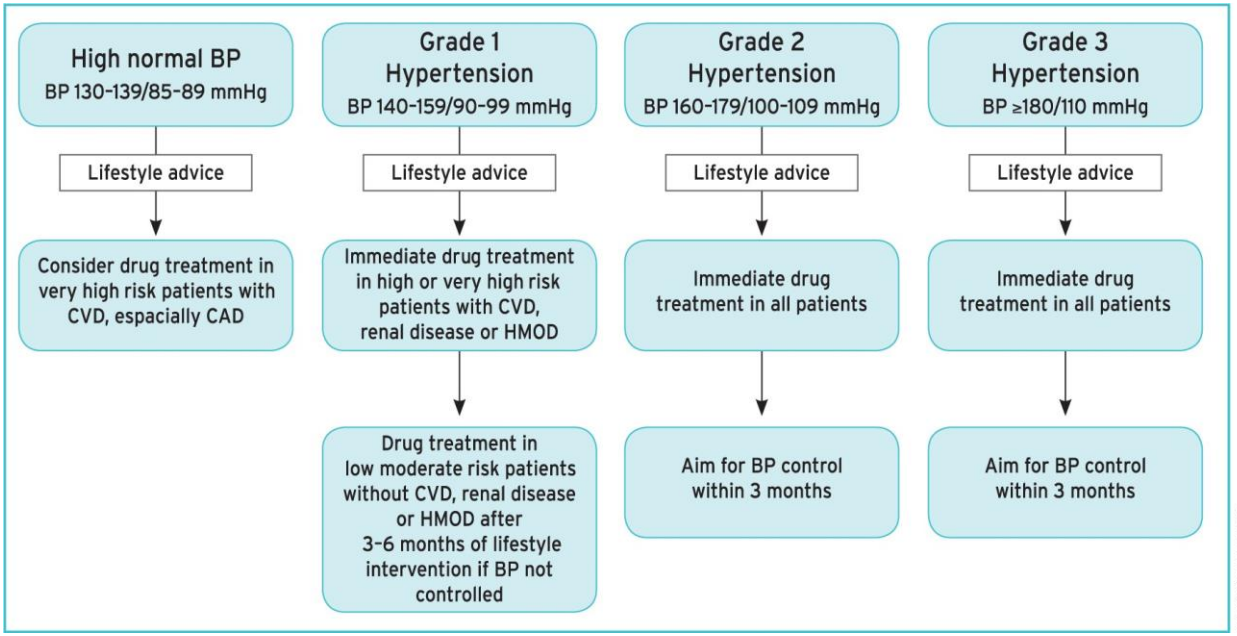
- Urinalysis for blood, protein and glucose
- Blood urea, electrolytes and creatinine
  Hypokalaemic alkalosis may indicate primary hyperaldosteronism but is usually due to diuretic therapy
- Blood glucose
- Serum total and HDL cholesterol
- Thyroid function tests
- 12-lead ECG (left ventricular hypertrophy,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

(HDL = high-density lipoprotein)

16.69 Specialised investigation of hypertension

- 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 the presence of renal artery stenosis
- Urinary catecholamines: to detect possible phaeochromocytoma (p. 675)
- Urinary cortisol and dexamethasone suppression test: to detect possible Cushing's syndrome (p. 666)
- Plasma renin activity and aldosterone: to detect possible primary aldosteronism (p. 674)

MANAGEMENT


High normal BP
BP 130-139/85-89 mmHg
Grade 1 Hypertension
BP 140-159/90-99 mmHg
Grade 2 Hypertension
BP 160-179/100-109 mmHg
Grade 3 Hypertension
BP ≥180/110 mmHg
Lifestyle advice
Lifestyle advice
Lifestyle advice
Lifestyle advice
Consider drug treatment in very high risk patients with CVD, espacially CAD
Immediate drug treatment in high or very high risk patients with CVD, renal disease or HMOD
Immediate drug treatment in all patients
Immediate drug treatment in all patients
Drug treatment in low moderate risk patients without CVD, renal disease or HMOD after 3-6 months of lifestyle intervention if BP not controlled
Aim for BP control within 3 months
Aim for BP control within 3 months
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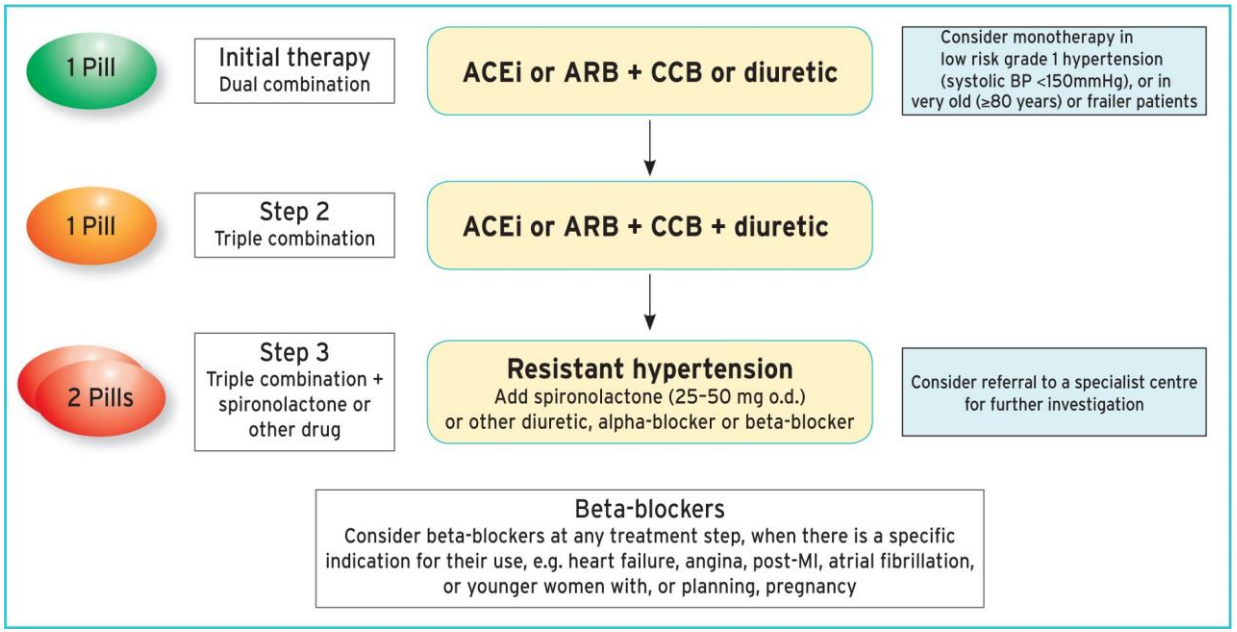
1 Pill
Initial therapy
Dual combination
ACEi or ARB + CCB or diuretic
Consider monotherapy in low risk grade 1 hypertension (systolic BP <150mmHg), or in very old (≥80 years) or frailer patients
1 Pill
Step 2
Triple combination
ACEi or ARB + CCB + diuretic
2 Pills
Step 3
Triple combination + spironolactone or other drug
Resistant hypertension
Add spironolactone (25-50 mg o.d.) or other diuretic, alpha-blocker or beta-blocker
Consider referral to a specialist centre for further investigation
Beta-blockers
Consider beta-blockers at any treatment step, when there is a specific indication for their use, e.g. heart failure, angina, post-MI, atrial fibrillation, or younger women with, or planning, pregnancy
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MANAGEMENT:

16.72 The influence of comorbidity on choice of antihypertensive drug therapy

Class of drug	Compelling indications	Possible indications	Caution	Compelling contraindications
α-blockers	Benign prostatic hypertrophy	–	Postural hypotension, heart failure[1]	Urinary incontinence
ACE inhibitors	Heart failure Left ventricular dysfunction, post-MI or established CAD Type 1 diabetic nephropathy Secondary stroke prevention[4]	Chronic renal disease[2] Type 2 diabetic nephropathy	Renal impairment[2] PAD[3]	Pregnancy Renovascular disease[2]
Angiotensin II receptor blockers	ACE inhibitor intolerance Type 2 diabetic nephropathy Hypertension with left ventricular hypertrophy Heart failure in ACE-intolerant patients, after MI	Left ventricular dysfunction after MI Intolerance of other antihypertensive drugs Proteinuric or chronic renal disease[2] Heart failure	Renal impairment[2] PAD[3]	Pregnancy
β-blockers	MI, angina Heart failure[5]	–	Heart failure[5] PAD Diabetes (except with CAD)	Asthma or chronic obstructive pulmonary disease Heart block
Calcium channel blockers (dihydropyridine)	Older patients, isolated systolic hypertension	Angina	–	–
Calcium channel blockers (rate-limiting)	Angina	Older patients	Combination with β-blockade	Atrioventricular block, heart failure
Thiazides or thiazide-like diuretics	Older patients, isolated systolic hypertension, heart failure, secondary stroke prevention	–	–	Gout[6]

[1]In heart failure when used as monotherapy. [2]ACE inhibitors or ARBs may be beneficial in chronic renal failure and renovascular disease but should be used with caution, close supervision and specialist advice when there is established and significant renal impairment. [3]Caution with ACE inhibitors and ARBs in PAD because of association with renovascular disease. [4]In combination with a thiazide or thiazide-like diuretic. [5]β-blockers are used increasingly to treat stable heart failure but may worsen acute heart failure. [6]Thiazides or thiazide-like diuretics may sometimes be necessary to control BP in people with a history of gout, ideally used in combination with allopurinol.
(ACE = angiotensin-converting enzyme; ARBs = angiotensin II receptor blockers; CAD = coronary artery disease; MI = myocardial infarction; PAD = peripheral arterial disease)

Refractory hypertension

It is defined as BP>140/90 mmHg in a patient treated with >=3 different antihypertensive drugs including a diuretic.

Refractory hypertension

It is defined as BP>140/90 mmHg in a patient treated with >=5 different antihypertensive drugs including diuretic and MRA .

Accelerated hypertension

Accelerated or **malignant hypertension** is a rare condition that can complicate hypertension of any aetiology. It 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 BP 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. Management In accelerated phase hypertension, lowering BP too quickly 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 BP under control with bed rest and oral drug therapy.

Intravenous or intramuscular labetalol (2 mg/min to a maximum of 200 mg), intravenous GTN (0.6–1.2 mg/hr), intramuscular hydralazine (5 or 10 mg aliquots repeated at half-hourly intervals) and intravenous sodium nitroprusside (0.3–1.0 μg/kg body weight/min) are all effective but require careful supervision, preferably in a high-dependency unit.