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BHS GUIDELINES

Guidelines for management of hypertension: report of the third working party of the British Hypertension Society

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for the British Hypertension Society

- **Use non-pharmacological measures in all hypertensive and borderline hypertensive people.**
- **Initiate antihypertensive drug therapy in people with sustained systolic blood pressures (BP) ⩾160 mm Hg or sustained diastolic BP ⩾100 mm Hg.**
- **Decide on treatment in people with sustained systolic BP between 140 and 159 mm Hg or sustained diastolic BP between 90 and 99 mm Hg according to the presence or absence of target organ damage, cardiovascular disease or a 10-year coronary heart disease (CHD) risk of ⩾15% according to the Joint British Societies CHD risk assessment programme/risk chart.**
- **In people with diabetes mellitus, initiate antihypertensive drug therapy if systolic BP is sustained ⩾140 mm Hg or diastolic BP is sustained ⩾90 mm Hg.**
- **In non-diabetic hypertensive people, optimal BP treatment targets are: systolic BP <140 mm Hg and diastolic BP <85 mm Hg. The minimum acceptable level of control (Audit Standard) recommended is <150/<90 mm Hg. Despite best practice, these levels will be difficult to achieve in some hypertensive people.**
- **In diabetic hypertensive people, optimal BP targets are; systolic BP <140 mm Hg and diastolic BP <80 mm Hg. The minimum acceptable level of control (Audit Standard) recommended is <140/<90 mm Hg. Despite best practice, these levels will be difficult to achieve in some people with diabetes and hypertension.**
- **In the absence of contraindications or compelling indications for other antihypertensive agents, low dose thiazide diuretics or beta-blockers are preferred as first-line therapy for the majority of hypertensive people. In the absence of compelling indications for beta-blockade, diuretics or long acting dihydropyridine calcium antagonists are preferred to beta-blockers in older subjects. Compelling indications and contraindications for all antihypertensive drug classes are specified.**
- **For most hypertensives, a combination of antihypertensive drugs will be required to achieve the recommended targets for blood pressure control.**
- **Other drugs that reduce cardiovascular risk must also be considered. These include aspirin for secondary prevention of cardiovascular disease, and primary prevention in treated hypertensive subjects over the age of 50 years who have a 10-year CHD risk ⩾15% and in whom blood pressure is controlled to the audit standard. In accordance with existing British recommendations, statin therapy is recommended for hypertensive people with a total cholesterol ⩾5 mmol/L and established vascular disease, or 10-year CHD risk ⩾30% estimated from the Joint British Societies CHD risk chart. Glycaemic control should also be optimised in diabetic subjects.**
- **Specific advice is given on the management of hypertension in specific patient groups, ie, the elderly, ethnic subgroups, diabetes mellitus, chronic renal disease and in women (pregnancy, oral contraceptive use and hormone replacement therapy).**
- **Suggestions for the implementation and audit of these guidelines in primary care are provided.**

Keywords: BHS; management of hypertension

Introduction

These guidelines update previous reports by working parties of the British Hypertension Society in 1989[1] and 1993.[2] Since the 1993 guidelines much new evidence has emerged, notably on optimal blood pressure targets during antihypertensive treatment[3]; management of hypertension in diabetic patients[3–7]; treatment of isolated systolic hypertension in the elderly[8]; comparison of the antihypertensive efficacy and tolerability of different classes of drug[9–11]; and the role of non-pharmacological measures in the prevention[12–15] and treatment[16] of hypertension. There has been a vigorous debate about the safety of dihydropyridine calcium antagonists,[17,18] but also new evidence from randomised controlled trials for their efficacy and safety, particularly in isolated systolic hypertension in the elderly.[8] These important additions to an already formidable body of evidence are very welcome and provide the basis for these new recommendations from the British Hypertension Society.

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On the whole, physicians report that they adhered to previous recommendations by the British Hypertension Society,[19] but with some important exceptions. They are less aware of, or less inclined to implement, recommendations to treat mild hypertension and isolated systolic hypertension in the elderly.[19,20] National and international surveys continue to reveal that there is under-diagnosis of hypertension, that those diagnosed as hypertensive often do not continue on treatment, and that those treated are often not controlled satisfactorily.[21–23] The situation has improved in recent years but in general the management of hypertension in the United Kingdom remains suboptimal.[23]

Also disturbing is further evidence that conventional management of hypertension leaves patients at an unacceptably high risk of cardiovascular complications and death, particularly from coronary heart disease (CHD) but also from stroke.[24–29] In part this is a consequence of suboptimal blood pressure control,[30] but other factors are also important. In a recent study, the persistent excess of CHD events in treated hypertensive subjects was predicted by three factors; (i) evidence of target organ damage before treatment, (ii) a history of cigarette smoking before treatment, and (iii) the serum cholesterol values before and during treatment.[29] These observations support the concept that effective management of hypertension requires the identification of those at highest cardiovascular risk and the adoption of multifactorial intervention, targeting not only blood pressure levels, but also associated cardiovascular risk factors. These new guidelines embrace this concept and provide detailed guidance on the management of hypertension and associated cardiovascular risk factors.

The recent trials of statins[31–34] and aspirin[3,35] for the prevention of CHD are important steps forward for hypertension management since the previous guidelines. A significant proportion of hypertensive patients will benefit from aspirin and statin treatment, even if these treatments are only targeted at those with a high level of CHD risk.[36,37] Formal estimation of CHD risk has been proposed as an aid to treatment decisions in hypertension,[38–45] and debated.[46–49] This estimation ideally entails counting and weighting major cardiovascular risk factors in addition to blood pressure itself.[50] Mindful of the strong relationship between blood pressure and the risk of stroke, the Society acknowledges that targeting cardiovascular disease risk (CVD) rather than CHD risk is preferable. However, in order to be consistent with three existing National guideline recommendations,[50–52] we recommend formal estimation of 10-year CHD risk using the computer programme 'Cardiac Risk Assessor' or the CHD risk chart issued by the Joint British Societies in their recommendation for the prevention of CHD.[50] It is reasonable to make this pragmatic recommendation because CHD risk is a good predictor of CVD risk which can be estimated by multiplying the estimated 10-year CHD risk level by 4/3 (eg, 30% CHD risk ≈40% CVD risk) [see Appendix 1]. Moreover, individualised estimates of 10-year stroke risk as well as CHD risk are provided by the Joint British Societies 'Cardiac Risk Assessor' computer programme. Consequently, whilst acknowledging that CVD prevention is the proper focus of hypertension management, the levels of CHD risk quoted in these guidelines, appropriately precipitate intervention for those at higher CVD risk.

BOX 1. Categories of Strength Used in Statements
(based on North of England evidence based guidelines, *BMJ* 1998)[53]

Strength of evidence
la-Evidence from meta-analysis of randomised controlled trials
Ib-Evidence from at least one randomised controlled trial
IIa-Evidence from at least one controlled study without randomisation
IIb-Evidence from at least one other type of quasi-experimental study
III-Evidence from descriptive studies, such as comparative studies, correlation studies, and case-controlled studies
IV-Evidence from expert committee reports or opinions or clinical experience of respected authorities, or both

Strength of recommendation
A-Directly based on category I evidence
B-Directly based on category II evidence or extrapolated recommendation from category I evidence
C-Directly based on category III evidence or extrapolated recommendation from category I or II evidence
D-Directly based on category IV evidence or extrapolated recommendation from category I, II or III evidence

These guidelines are intended for general practitioners, practice nurses, and generalists in hospital practice, and aim to present as clearly as possible, the best currently available evidence on hypertension management. The evidence supporting the recommendations contained in these new British Hypertension Society guidelines is graded using the North of England Group Criteria[53] (Box 1). The guidelines should be applied with due regard to local circumstances and policies, and with appropriate clinical judgement as regards the needs of individual patients (Box 2).

Blood pressure measurement (Box 3)

All adults should have blood pressure measured routinely at least every 5 years until the age of 80 years. Those with high-normal values (135–139/85–89 mm Hg) and those who have had high readings at any time previously should have blood pressure re-measured annually. The British Hypertension Society recommendations for measuring blood pressure,[54] now available on CD-ROM (see Appendix 2), should be followed (Table 1). Seated blood pressure recordings are generally sufficient, but standing blood pressure should be measured in elderly or diabetic patients to exclude orthostatic hypotension. When assessing CHD/CVD risk the average of several measurements at separate visits is more accurate than measurements taken at a single visit.[55] In uncomplicated mild hypertension the average of two readings per visit at monthly intervals over 4–6 months should be used to guide the decision to treat. In more severe hypertension prolonged

BOX 2. Guidelines

- These guidelines, which are an update of previous reports in 1989 and 1993 (**B**), are based on collective expert interpretation of current clinical evidence as assessed by members of the British Hypertension Society.
- Important new inclusions are optimal blood pressure targets (**A**), management of hypertension in diabetic patients (**A**), treatment of isolated systolic hypertension in the elderly (**A**), comparison of different classes of drug therapy (**A**) and the role of non-pharmacological treatment (**A**).
- In view of clear evidence for other measures to reduce cardiovascular risk; lowering cholesterol (**A**), use of aspirin (**A**), treatment of diabetes (**A**) and discontinuation of smoking (**B**), formal estimation of risk and risk thresholds for treatment have been included (**B**) see Appendix 1.
- There is evidence that current guidelines are not being implemented and the detection and management of hypertension remains sub-optimal (**B**). There is a need to improve the quality of care by better screening, protocols to support clinical decision making and increased education of doctors, nurses and patients.

BOX 3. Blood Pressure Measurement

- Use the British Hypertension Society recommendations (**C**).
- Use a device with validated accuracy that is properly maintained and calibrated.
- Patient should be seated with the arm at the level of the heart. The bladder size should be adjusted for the arm circumference, the cuff deflated at 2 mm/sec and the blood pressure measured to the nearest 2 mm Hg. Diastolic pressure is recorded as disappearance of the sounds (phase V).
- At least two measurements should be made at each visit and four visits to determine blood pressure thresholds. Possible indications for 'home' or ambulatory blood pressure monitoring include the diagnosis of 'white coat hypertension', suspected hypotension, excessive blood pressure variability and resistance to drug therapy (**C**).

Table 1 Blood pressure measurement

- Follow BHS guidelines on technique.[55]
- Use device with validated accuracy, that is properly maintained and calibrated.
- Measure sitting BP routinely: standing BP in elderly or diabetic patients.
- Remove tight clothing, support arm at heart level, ensure hand relaxed.
- Use cuff of appropriate size.
- Lower mercury slowly, by 2 mm per second.
- Read BP to the nearest 2 mm Hg.
- Measure diastolic as disappearance of sounds (phase 5).
- Take two measurements at each visit.
- Use the average for several visits when estimating cardiovascular risk in mild hypertension.

observation before treatment is not necessary or warranted. The average blood pressure is only one factor determining cardiovascular risk in uncomplicated mild hypertension. Formal estimation of CHD/CVD risk needs consideration of age, sex, smoking habit, diabetes, total:HDL cholesterol ratio, and family history in addition to blood pressure,[50] as described later.

Systolic or diastolic blood pressure?

There has been much debate on the relative importance of systolic and diastolic blood pressure, but in practice systolic blood pressure should be regarded as the more important. In general, systolic and diastolic blood pressure correlate highly, and in epidemiological studies both are important risk factors for cardiovascular disease.[56] Outcome trials of antihypertensive treatment based on thresholds of diastolic[57,58] or systolic blood pressure[8,59] have shown similar reductions in cardiovascular events. This has important implications with regard to blood pressure *thresholds* for the treatment of hypertension and blood pressure *targets* during treatment. When treatment is recommended at a blood pressure threshold of 140/90 mm Hg, this means 140 mm Hg systolic or 90 mm Hg diastolic. An optimal blood pressure target of $<$140/85 mm Hg means $<$140 mm Hg systolic and $<$85 mm Hg diastolic.

Ambulatory blood pressure (ABPM)

All outcome trials in hypertension have been based on surgery or clinic blood pressure, not ABPM, and it is therefore difficult to provide firm guidance based on evidence for use of this technique. Nevertheless, ABPM is widely used and may be valuable in special circumstances. ABPM provides numerous measurements over a short time, and so reduces variability when compared to the average of a limited number of surgery or clinic readings.[60,61] Blood pressure by ABPM correlates more closely with evidence of target organ damage,[60–63] presumably in part because of reduced variability and measurement error.[60,61] In one controlled trial[64] treatment based on ABPM rather than surgery or clinic readings resulted in less drug treatment, but at the expense of slightly but significantly higher ABPM and clinic blood pressures. There was no difference in well-being or cost-effectiveness. Incorporation of ABPM measurements in formal CHD/CVD risk estimation has little effect on risk, because blood pressure is only one of several risk factors for cardiovascular complications.[65]

ABPM may be indicated in the following circumstances:

- when blood pressure shows unusual variability;
- in hypertension resistant to drug therapy, defined as blood pressure $>$150/90 mm Hg on a regimen of three or more antihypertensive drugs;
- when symptoms suggest the possibility of hypotension;
- to diagnose white coat hypertension.[66] The term 'white coat hypertension' is widely used to

describe consistent hypertension in the clinic with consistent normotension by ABPM. There is a systematic clinic-ABPM difference in the population that is related to the level of clinic blood pressure,[66,67] and white coat hypertension is considered to be present only when the clinic-ABPM difference exceeds the population average difference.

It is not necessary or feasible to perform ABPM to exclude white coat hypertension in *all* hypertensive patients. It is *not* indicated in patients who are at high CHD/CVD risk. This includes patients who already have target organ damage or cardiovascular complications (Table 2), and those who have an estimated 10-year CHD risk of 15% or higher. In these patients treatment decisions should be based on surgery or clinic pressures rather than ABPM, as was the case in outcome trials of hypertension treatment. ABPM is also unnecessary in patients with mild hypertension (140–159/90–99 mm Hg) with no target organ damage, no cardiovascular complications, and an estimated 10-year CHD risk is <15%.[65] These patients may be left untreated without using ABPM but must be followed up.

ABPM may alter management when the average clinic blood pressure is ⩾160/100 mm Hg, there is no target organ damage or cardiovascular complications, *and* the estimated 10-year CHD risk is <15%. Here elevated blood pressure is the *only* indication of high CHD/CVD risk, and for antihypertensive treatment, and normal blood pressure values by ABPM may alter the treatment decision. However a decision to withhold treatment in such patients should be based on appropriately-adjusted normal values for ABPM (see below), and should be confirmed by a second ABPM record because of within-patient variability and limited reproducibility.[67] Furthermore patients left untreated on the basis of ABPM will need to be followed up, with reassessment of blood pressure and cardiovascular risk at least once a year. The annual reassessment may require repeated ABPM measurement.

Some important points in interpreting the results of ABPM records need emphasis. The average daytime blood pressure should be used for treatment decisions, not the average *24-h* blood pressure. Blood pressure measured by ABPM is *systematically* lower than surgery or clinic measurements in hypertensive and normotensive people.[67,68] Because of this, treatment thresholds and targets must be adjusted downwards when making decisions based on ABPM data. Precise adjustment is complex, but the average difference between clinic and daytime mean pressures determined by ABPM is approximately 12/7 mm Hg.[65,67,68] Thus an ABPM average daytime blood pressure of 148/83 mm Hg is approximately equivalent to a surgery blood pressure of 160/90 mm Hg, and this may require treatment in some patients. Recommended targets for ABPM measurements are given in Table 3. These are extrapolated from clinic or surgery thresholds and targets derived from controlled trials using conventional blood pressure measurements.

Table 2 Initial evaluation of the hypertensive patient

- **Causes of hypertension:**
  - –drugs (NSAID's, oral contraceptive, steroids, liquorice, sympathomimetics, ie. some cold cures).
  - –renal disease (present, past or family history: palpable kidney(s) – polycystic, hydronephrosis or neoplasm).
  - –renovascular disease (abdominal or loin bruit).
  - –phaeochromocytoma (paroxysmal symptoms).
  - –Conn's syndrome (tetany, muscle weakness, polyuria).
  - –coarctation (delayed or weak femoral pulses).
  - –Cushings (general appearance).
- **Contributory factors:**
  - –overweight
  - –excess alcohol (>3 units/day)
  - –salt intake
  - –lack of exercise
- **Complications of hypertension/target organ damage:**
  - –stroke, TIA, dementia
  - –LVH, heart failure
  - –myocardial infarct, angina, CABG or angioplasty
  - –peripheral vascular disease
  - –fundal hemorrhages or exudates
  - –proteinuria
  - –renal impairment
- **Cardiovascular risk factors:**
  - –smoking
  - –diabetes
  - –total – cholesterol:HDL-cholesterol ratio
  - –family history
  - –age
  - –sex
- **Contraindications to drugs:**
  See Table 5

Blood pressure measurement at home

This technique is less expensive and more convenient for patients than ABPM. Evidence on the role of self-measurement of blood pressure is less extensive than for ABPM, but many of the same considerations apply. In particular, measurements made at home need to be 'adjusted' upwards by approximately 12/7 mm Hg for equivalence to surgery or clinic measurements when making treatment decisions.[69] Information on the validity of different home blood pressure monitoring devices can be obtained from the British Hypertension Society Information Service (see Appendix 2).

Table 3 Suggested target blood pressures during antihypertensive treatment. Systolic and diastolic should *both* be attained, eg <140/85 mm Hg means less than 140 systolic *and* less than 85 diastolic

	Clinic BP (mm Hg)		*Mean day-time ABPM or home BP*	
	No diabetes	*Diabetes*	*No diabetes*	*Diabetes*
Optimal BP:	<140/85	<140/80	<130/80	<130/75
Audit Standard:[a]	<150/90	<140/85	<140/85	<140/80

The Audit Standard[a] reflects the minimum recommended levels of blood pressure control.
Despite best practice, the Audit Standard will not be achievable in all treated hypertensives.

Accuracy of sphygmomanometers

A properly maintained mercury sphygmomanometer is the traditional accurate and robust gold standard for routine clinical practice, but for health and safety reasons the use of mercury devices is declining and will eventually cease completely. There will be increasing use of alternative methods such as aneroid, semi-automated and automated devices. There is evidence that many alternatives to the mercury sphygmomanometer are unacceptably inaccurate when they are subjected to formal validation.[70] Those purchasing or using alternatives to the mercury sphygmomanometer should insist on evidence from the manufacturer that the device has been validated formally, and proved accurate, according to the standards of the British Hypertension Society protocol[71] or American National Standards.[72,73] Information on the validity of different devices can be obtained from the British Hypertension Society Information Service (see Appendix 2).

Non-pharmacological measures (Box 4)

Primary prevention of hypertension

The current strategy for preventing cardiovascular complications associated with hypertension is unsatisfactory in that it requires the detection and lifelong drug therapy of a large proportion of the adult population including about one half of all elderly people. Moreover, such management as currently practised does not reduce the cardiovascular risk of hypertensive patients to that of normotensive subjects, as discussed earlier. There is increasing evidence that a population strategy could prevent the rise in blood pressure with age, reduce the prevalence of hypertension and need for drug therapy, and reduce overall cardiovascular risk.[74–77] The requirements for this strategy have been summarised by Stamler[77] as a diet high in fruit and vegetables; high in legumes and whole grains; high in fat-free and low-fat dairy, poultry, fish, shellfish, and meat products; high in all essential nutrients; reduced in salt; reduced in total fat, saturated fat and cholesterol; with no more than 2–3 units of alcohol per day; and controlled in calories to prevent or correct obesity.

BOX 4. Non-Pharmacological Measures

- Non-pharmacological measures; weight reduction (**A**), reduced salt intake (**A**), reduced fat intake (**A**), limited alcohol consumption (**A**), dynamic exercise (**A**) and increased fruit and vegetable consumption (**A**) are effective in lowering blood pressure.
- Alone or in combination these interventions can reduce the need for drug therapy and enhance the effect of antihypertensive agents (**A**). A favourable effect on cardiovascular outcome is assumed but not proven.
- To reduce overall cardiovascular risk, patients should stop smoking (**B**), reduce their saturated fat intake and increase consumption of poly-unsaturated, mono-unsaturated fats and oily fish (**B**).

Established hypertension

Recent controlled trials[12,13,15,16,78–83] have confirmed that changes in diet and lifestyle do lower blood pressure and may also reduce cardiovascular risk. Clear verbal and written advice on the measures below should be provided for all hypertensive patients and also for those with high-normal blood pressure or a strong family history. They may lower blood pressure as much as drug monotherapy; reduce the need for drug therapy[12,16]; enhance the antihypertensive effect of drugs; reduce the need for multiple drug regimens; and favourably influence overall cardiovascular risk. Conversely, failure to adopt these measures may attenuate the response to antihypertensive drugs.[84]

(1) Measures that lower blood pressure:
- weight reduction
- reduced salt intake
- limitation of alcohol consumption
- physical exercise
- increased fruit and vegetable consumption
- reduced total fat and saturated fat intake.

(2) Measures to reduce cardiovascular risk:
- stop smoking;
- replace saturated fat with polyunsaturated and monounsaturated fats;
- increase oily fish consumption.
- reduce total fat intake.

In patients with mild hypertension, but no cardiovascular complications or target organ damage, the response to these measures should be observed during the initial 4–6 month period of evaluation. When drug therapy has to be introduced more quickly, for example in patients with severe hypertension, non-pharmacological measures should be instituted in parallel with drug treatment.

Weight reduction by calorie restriction is appropriate for the majority of hypertensive patients because most are overweight, and results in blood pressure reduction[12,16] of about 2.5/1.5 mm Hg for each kilogram lost.

Salt reduction from an average of 10 to 5 grams (5 grams ≈1 teaspoon) daily lowers blood pressure by about 5/3 mm Hg,[16,85,86] with larger blood pressure falls in the elderly and those with higher initial blood pressure levels.[86] All hypertensive patients should have clear verbal and written advice to reduce salt intake to 5 grams per day. Many will have already discontinued adding salt at the table and even when cooking, but few are aware of the large amounts of salt in processed foods, such as bread (one slice contains 0.5 grams of salt), some breakfast cereals, and flavour enhancers such as stock cubes or manufactured sauces. Patients, and those who cook for patients, should be provided with specific written advice (see Appendix 2).

Alcohol intake above 21 units per week is associated with blood pressure elevation that is reversible by reducing the intake.[87] Binge drinking is associated with an increased risk of stroke.[88] Hypertensive patients should be advised to limit their alcohol intake to 21 units per week for men, and 14 units

per week for women. However in a recent study this had a limited effect as a single intervention, because alcohol intake was little reduced.[14] Consumption of smaller amounts of alcohol, up to the recommended limit, may protect against CHD[89] and should not be discouraged.

Exercise should be regular; dynamic (eg, brisk walking) rather than isometric (eg, weight training); and tailored to the individual patient.[13] For example, three vigorous training sessions per week may be appropriate for fit younger patients[82] or brisk walking for 20 min per day for older patients.[81,90]

Increased fruit and vegetable consumption is now supported by controlled-trial evidence showing that an increase from two to seven portions daily lowered blood pressure significantly in hypertensive patients, by 7/3 mm Hg.[12] This effect of fruit and vegetables on blood pressure may be a consequence of increased potassium intake.[78,91] Hypertensive patients should have clear advice on increasing fruit and vegetable intake.[92] When this is combined with an increase in low-fat dairy products and reduction of saturated and total fat, blood pressure falls may be larger, averaging 11/6 mm Hg in hypertensive patients and 4/2 mm Hg in those with high-normal blood pressure.[12]

Cigarette smoking increases cardiovascular risk more than mild hypertension, and smoking was one factor related to the persistent increase in coronary mortality in men with treated hypertension.[29] Hypertensive patients who smoke should be given advice and help to stop smoking. The use of nicotine replacement therapies approximately doubles smoking cessation rates.[93]

Serum cholesterol before and during the treatment of hypertension is also an important predictor of cardiovascular disease.[29] All patients should be advised to reduce saturated fat and cholesterol intake, and to substitute polyunsaturated and monounsaturated fats. These diet changes will reduce serum cholesterol by an average of 6%,[94] but it is important to recognise the difficulties in implementing and sustaining these measures.[95] Many hypertensive patients are at very high risk of CHD[36,37] and will need aspirin and statin treatment *in addition* to non-pharmacological measures if they are to have adequate coronary prevention.

Effective implementation of these non-pharmacological measures requires enthusiasm, knowledge, patience, and considerable time spent with patients and other family members. It is best undertaken by well-trained health professionals, eg, practice or clinic nurse, and should be backed up by simple clear written information (see Appendix 2).

Evaluation of hypertensive patients

All hypertensive patients should have a thorough history and physical examination, but need only a limited number of routine investigations. It is beyond the scope of these guidelines to discuss every detail of the clinical evaluation, but it may be useful to summarise the aims, which are to elicit and document:

- causes of hypertension, eg, renal disease, endocrine causes;
- contributory factors, eg, obesity, salt intake, excess alcohol intake;
- complications of hypertension, eg, previous stroke, left ventricular hypertrophy;
- cardiovascular risk factors, eg, smoking, family history;
- contraindications to specific drugs, eg, asthma (beta-blockers), gout (thiazides).

These aspects are summarised in more detail in Table 2.

Routine investigation should be limited to:

- urine strip test for protein and blood;
- serum creatinine and electrolytes;
- blood glucose;
- serum total: HDL cholesterol;
- ECG.

Note that chest X-ray, urine microscopy and culture, and echocardiography are not required routinely. An echocardiogram is valuable to confirm or refute the presence of left ventricular hypertrophy when the ECG shows 'high' left ventricular voltage without T-wave abnormalities, as is often the case in young patients. When the clinical evaluation or results of these simple investigations suggests a need for further investigation it is usually best to refer for specialist advice, because the additional investigations needed are often difficult to arrange from general practice. Indications for referral for specialist advice or treatment are suggested in Table 4.

Table 4 Suggested indications for specialist referral

- Urgent treatment needed:
  –accelerated (malignant) hypertension
  –severe hypertension (eg, >220/120 mm Hg)
  –impending complications (eg, TIA, left ventricular failure)
- **Possible underlying cause:**
  –any clue in history or examination of a secondary cause
  –hypokalaemia/increased plasma sodium (Conn's syndrome?)
  –elevated serum creatinine
  –proteinuria or haematuria
  –recent onset or worsening of hypertension
  –resistant to a three drug regimen
  –young age (any hypertension <20 years; needing treatment <30 years)
- **Therapeutic problems:**
  –treatment resistance
  –multiple drug intolerance
  –multiple drug contraindications
  –persistent non-compliance
  –treatment declined (the reluctant hypertensive)
- **Special situations**
  –unusual BP variability
  –possible isolated clinic hypertension
  –hypertension in pregnancy

Thresholds for antihypertensive drug therapy (Box 5)

Absolute cardiovascular risk

The importance of absolute cardiovascular risk was recognised in the previous British Hypertension Society guidelines,[2] which advised early drug treatment of patients with more severe hypertension (≥200/110 mm Hg), treatment of sustained blood pressure ≥160/100 mm Hg, and treatment of patients with diastolic pressure 90–99 mm Hg who had particularly high cardiovascular risk. Patients with cardiovascular complications (eg, previous stroke or coronary disease) or target organ damage (eg, left ventricular hypertrophy) were recognised as having cardiovascular risk sufficiently high to warrant treatment of even mild hypertension, for example 140/90 mm Hg. These recommendations remain sound, and are not altered.

A difficulty acknowledged in the previous guidelines concerned the treatment decision for patients with 'mild' hypertension, averaging 140–159/90–99 mm Hg, who were at variable risk depending on other risk factors. Advice to treat, or to leave untreated and observe, was based on the presence of additional cardiovascular risk factors including age, male sex, smoking, serum lipids, and family history. However no formal method to estimate cardiovascular risk using these risk factors was provided. Intuitive estimates of absolute risk are very inaccurate.[96,97] Risk estimation is improved when additional risk factors are simply counted,[98] but is significantly more accurate when all major risk factors are counted and *weighted* using risk functions derived from epidemiological studies, most commonly the Framingham risk function. The Joint British Societies recently issued recommendations on preventing CHD and included a computer programme; the 'Cardiac Risk assessor' and a CHD risk chart both of which are based on the Framingham risk function.[50] The BHS recommends the use of either of these methods to estimate 10-year CHD risk and thereby help to rationalise treatment decisions for hypertensive people. The Joint British Societies computer programme 'Cardiac Risk Assessor' is the preferred method for estimating 10-year CHD risk and can also be used to calculate stroke risk over the same period. The use of a computer to estimate CHD risk may not be practical in all clinical settings. In such circumstances, we recommend the use of the 'coronary risk chart' issued by the Joint British Societies (Figure 1). This chart is also based on the Framingham risk function and uses colour-coded bands to specify three levels of 10-year CHD risk; ≥30%, ≥15% and ≤15%. The practicalities of how to measure CHD risk using the Joint British Societies computer programme or risk chart are detailed in Appendix 1 and figure 2.

Targeting antihypertensive treatment at absolute CHD/CVD risk is underpinned by evidence from meta-analyses of outcome trials which show that the relative risk reduction by antihypertensive treatment is approximately constant, with a 38% reduction in stroke and 16% reduction in coronary events.[57,58] Benefit from treatment can be expressed as the number needed to treat (NNT) over 5 years.[99] In patients with mild hypertension treatment reduces cardiovascular complications by approximately 25%,[44,55] and treatment of patients at a 10-year CHD risk of ≥15% (CVD risk of ≥20%) corresponds to a NNT for 5 years of 40. This means treatment of 40 patients for 5 years to prevent one cardiovascular complication. Formal estimation of CHD/CVD risk also informs decisions on the treatment of hypertensive people with aspirin or statins, as discussed below.

It is recommended that all patients with average blood pressure 140–159 *or* 90–99 mm Hg should be offered antihypertensive drug treatment if: (i) there is any complication of hypertension or target organ damage, or diabetes (Table 2) and/or (ii) The 10-year

BOX 5. Thresholds and Treatment Targets for Antihypertensive Drug Therapy

- Drug therapy should be started in all patients with sustained systolic blood pressures ≥160 mm Hg or sustained diastolic blood pressures ≥100 mm Hg despite non-pharmacological measures (**A**).
- Drug treatment is also indicated in patients with sustained systolic blood pressures 140–159 mm Hg or diastolic blood pressures 90–99 mm Hg if target organ damage is present, or there is evidence of established cardiovascular disease, or diabetes, or the 10-year CHD risk is ≥15% (**B**).
- For most patients a target of ≤140 mm Hg systolic and ≤85 mm Hg is recommended (**A**). For patients with diabetes a lower target of ≤140/80 mm Hg is recommended (**A**).

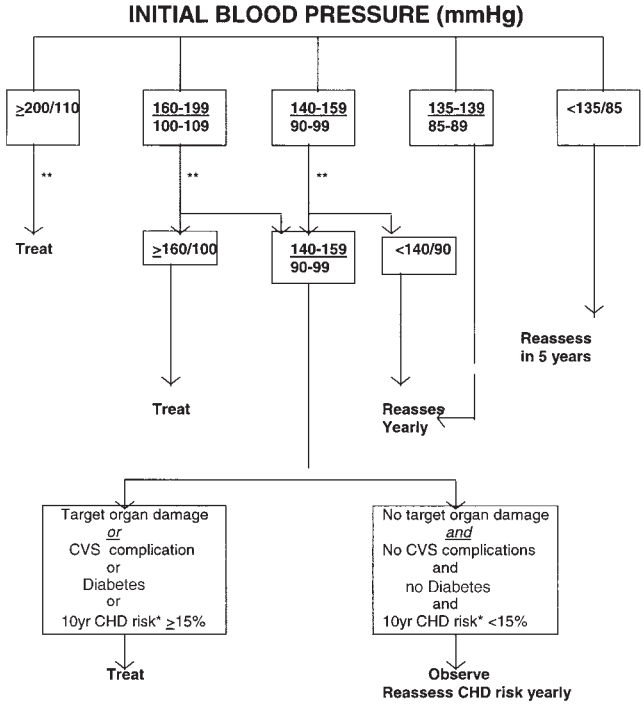


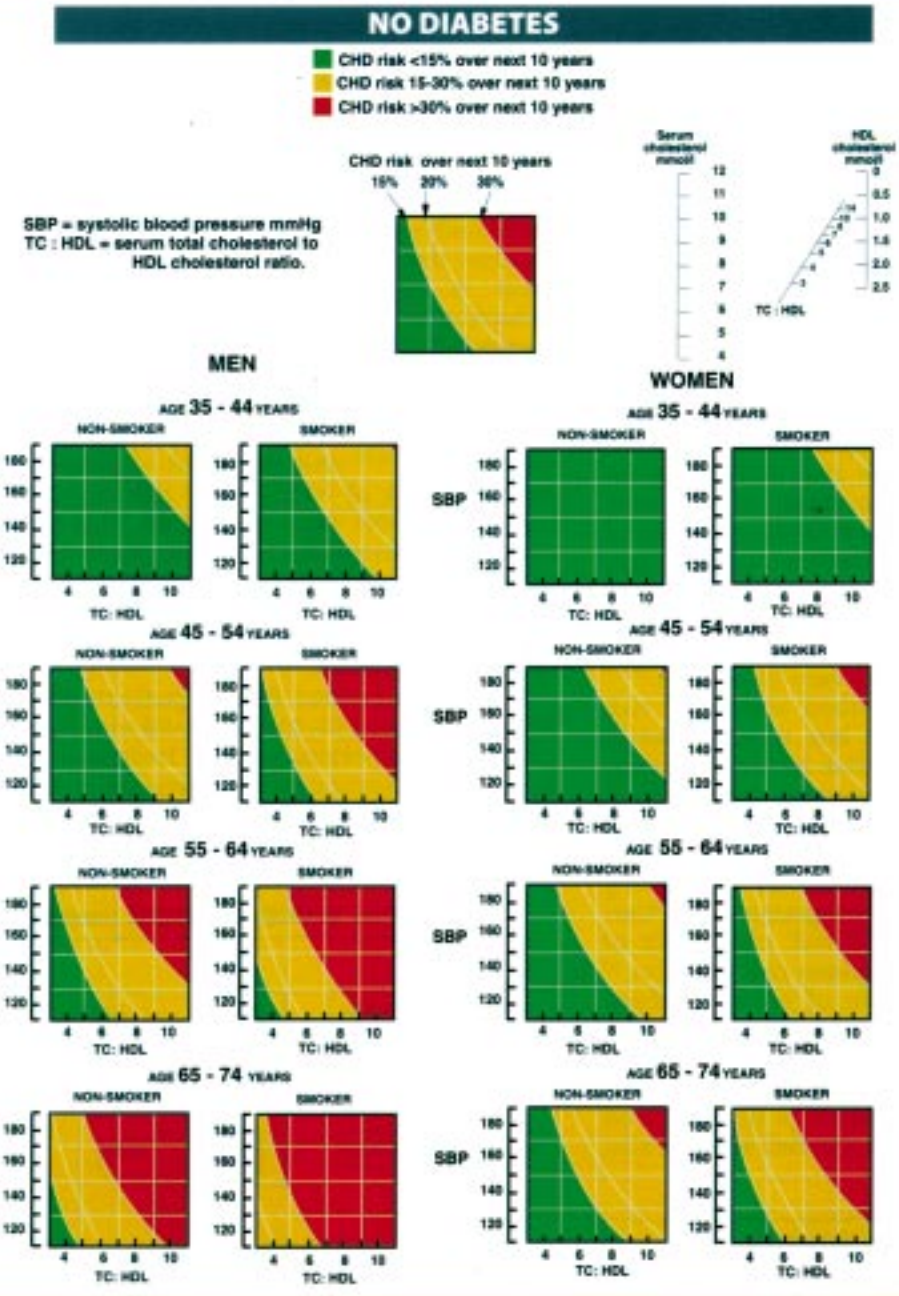
Figure 1 *Estimated by the Joint British Societies 'Cardiac Risk Assessor' computer programme or CHD risk chart.[51] **Repeated measurements (see text).

British Cardiac Society

British Hypertension Society

Joint British Societies

Joint British Recommendations on Prevention



How to use the Coronary Risk Prediction Chart for Primary Prevention

These charts are for estimating coronary heart disease (CHD) risk (non fatal MI and coronary death) for individuals who have not developed symptomatic CHD or other major atherosclerotic disease.

The use of these charts is not appropriate for patients who have existing disease which already puts them at high risk. Such diseases are:

- CHD or other major atherosclerotic disease
- Familial hypercholesterolaemia or other inherited dyslipidaemia
- Established hypertension (systolic BP > 160 mmHg and/or diastolic BP > 100 mmHg) or associated target organ damage
- Diabetes mellitus with associated target organ damage
- Renal dysfunction

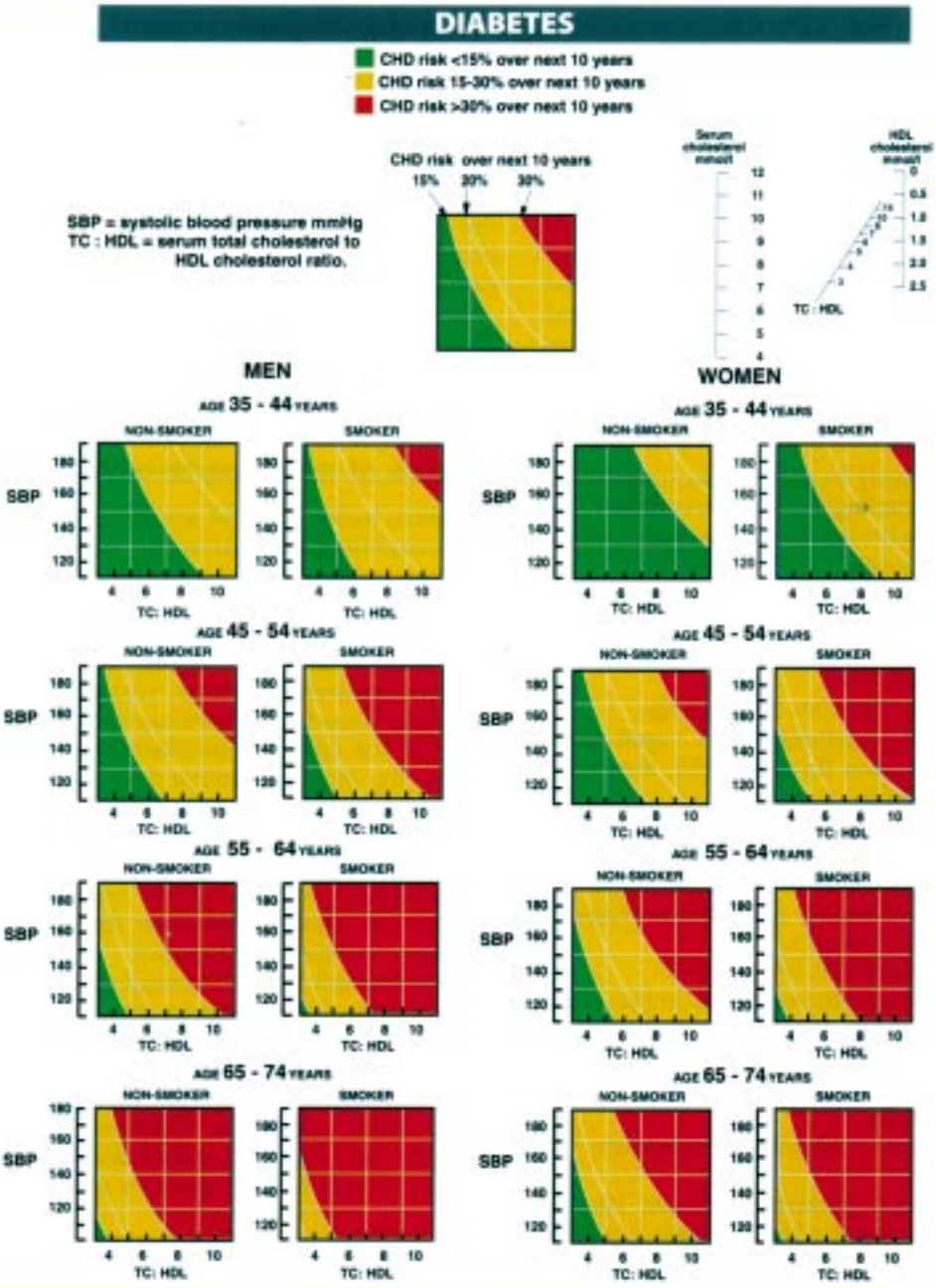
- To estimate an individuals absolute 10 year risk of developing CHD find the table for their gender, diabetes (yes/no), smoking status (smoker/non smoker) and age. Within this square define the level of risk according to systolic blood pressure and the ratio of total cholesterol to HDL cholesterol. If there is no HDL cholesterol result then assume this is 1.0mmol/1and then the lipid scale can be used for total cholesterol alone.
- High risk individuals are defined as those whose 10 year CHD risk exceeds 15% (equivalent to a *cardiovascular* risk of 20% over the same period). As a minimum those at highest risk (≥ 30% red) should be targeted and treated now, and as resources allow others with a risk of > 15% (orange) should be progressively targeted.

Coronary Risk Prediction Chart

of Coronary Heart Disease in Clinical Practice

British Hyperlipidaemia Association

British Diabetic Association



- Smoking status should reflect lifetime exposure to tobacco and not simply tobacco use at the time of risk assessment.
- The initial blood pressure and the first random (non fasting) total cholesterol and HDL cholesterol can be used to estimate an individuals risk. However, the decision on using drug therapy should be based on repeat risk factor measurements over a period of time. The chart should not be used to estimate risk after treatment of hyperlipidaemia or blood pressure has been initiated.
- CHD risk is higher than indicated in the charts for
  - Those with a family history of premature CHD (men <55 years and women <65 years) which increases the risk by a factor of approximately 1.5.
  - Those with raised triglyceride levels
  - Those who are not diabetic but have impaired glucose tolerance.
  - Women with premature menopause.
  - As the person approaches the next age category. As risk increases exponentially with age the risk will be closer to the higher decennium for the last four years of each decade.
- In ethnic minorities the risk chart should be used with caution as it has not been validated in these populations.
- The estimates of CHD risk from the chart are based on groups of people and in managing an *individual* the physician also has to use clinical judgement in deciding how intensively to intervene on lifestyle and whether or not to use drug therapies.
- An individual can be shown on the chart the direction in which the risk of CHD can be reduced by changing smoking status, blood pressure or cholesterol.

CHD risk is ⩾15% (≈20% CVD risk) despite advice on non-pharmacological measures.

Decisions on treatment at lower levels of CHD/CVD risk will be influenced by the patient's attitude to treatment, and the benefit anticipated from treatment. The benefit expected for an individual patient can be calculated using the absolute risk of a cardiovascular event and the relative risk reduction by treatment (25%).

When a decision is reached not to treat any patient with mild hypertension, it is essential to continue observation and monitoring of blood pressure, at least once per year. Blood pressure will rise within 5 years to levels clearly requiring treatment in about 10–15% of patients.[100] In addition CHD/CVD risk will increase with age, and risk should be reassessed at yearly intervals. These patients should all be encouraged to continue with non-pharmacological measures to lower blood pressure and cardiovascular risk.

Thresholds for intervention are summarised below and in Figure 1.

- Accelerated (malignant) hypertension [papilloedema, fundal hemorrhages and exudates] *or* impending cardiovascular complications; admit for immediate treatment.
- Blood pressure ⩾220/120 mm Hg; treat immediately.
- Blood pressure 200–219/110–119 mm Hg: confirm over 1–2 weeks, then treat.
- Blood pressure 160–199/100–109 mm Hg:
  –cardiovascular complications/target organ damage (Table 2) or diabetes (type I or II), *present*—confirm over 3–4 weeks, then treat.
  –cardiovascular complications/target organ damage or diabetes (type I or II) *absent*:
  –non-pharmacological advice, re-measure weekly initially, and treat if blood pressure persists at these levels over 4–12 weeks.
- Blood pressure 140–159/90–99 mm Hg:
  –cardiovascular complications/target organ damage (Table 2) or diabetes (type I or II) *present*—confirm within weeks and treat;
  –cardiovascular complications/target organ damage or diabetes *absent*:
  –non-pharmacological advice, re-measure at monthly intervals.
  –If mild hypertension persists, estimate 10-year CHD risk formally using the Joint British Societies 'Cardiac Risk Assessor' computer programme or the Joint British Societies CHD risk chart[50] (Figure 2). Treat if the estimated 10-year CHD ⩾15% (≈20% CVD risk).

Treatment goals (Box 5)

The HOT trial[3] has provided the best evidence to date on optimal blood pressure targets during antihypertensive treatment. In patients with diastolic pressures of 100–115 mm Hg, the optimal blood pressure based on an on-treatment analysis for reduction of cardiovascular events was reported to be 139/83 mm Hg. However, hypertensive patients were apparently disadvantaged little provided blood pressure was below 150/90 mm Hg. Reduction of blood pressure below the optimal level caused no harm. An important practical point is that the optimal blood pressure was attained, by titrating treatment in stepped-care fashion aiming for diastolic blood pressures of ⩽90, ⩽85 and ⩽80 mm Hg. With this systematic method of treatment the final diastolic blood pressure was above 90 mm Hg in only 7% of patients. Using an intention-to-treat analysis, in hypertensive patients with diabetes there appeared to be a significant advantage in lowering blood pressure to below the optimal level for non-diabetic patients. Titration of treatment in diabetics aiming for a diastolic pressure ⩽80 mm Hg halved the incidence of major cardiovascular events when compared to treatment aiming for diastolic blood pressure ⩽90 mm Hg.

Considering prospective observational data and the findings of the HOT trial,[3] recommendations for target blood pressures during treatment are shown in Table 3.

Choice of antihypertensive drug (Box 6)

For each major class of antihypertensive drug there are compelling indications for use in specific patient groups, and also compelling contraindications. There are also indications, contraindications, and cautions that are less clear-cut, and which are given different weight by different doctors. These indications, contraindications, and cautions for each of the drug classes are summarised in Table 5. When none of the special considerations listed in Table 5 apply, the least expensive drug, with the most supportive trial evidence; a low dose of a thiazide diuretic, should be preferred.

Since the previous guidelines[2] three long-term double-blind studies have compared the major classes of antihypertensive drug (thiazide, beta-blocker, calcium antagonist, ACE-inhibitor, and alpha-blockers,[9–11] and overall showed no consistent or important differences as regards antihypertensive efficacy, side effects, or quality of life. There were, however, differences in average response between drug classes related to age and ethnic group.[10] Few trials have compared different classes of drugs directly as regards reduction in cardiovascular events,[101] and none is entirely satisfactory, but they

BOX 6. Choice of Antihypertensive Drug Treatment

- Use a low dose of thiazide as first-line treatment unless there is a contraindication or a compelling indication for another drug class (**A**).
- Long acting dihydropyridine calcium antagonists are a suitable alternative for isolated systolic hypertension in the elderly when low-dose thiazide is not tolerated or contraindicated (**A**).
- Choice of drug will depend on relative indications and contra-indications in the individual patient (Table 5).
- Less than half of all hypertensives will be controlled on monotherapy and one-third will require three or more drugs (**A**).

Table 5 Compelling and possible indications, contraindications and cautions for the major classes of antihypertensive drug

Class of drug	*Compelling indications*	*Possible indications*	*Possible contraindications*	*Compelling contraindications*
Alpha-blockers	Prostatism	Dyslipidaemia	Postural hypotension	Urinary incontinence
ACE-inhibitors	Heart failure LV dysfunction Type 1 diabetic Nepropathy	Chronic renal disease[a] Type 2 diabetic Nephropathy	Renal impairment[a] PVD[b]	Pregnancy Renovascular disease
All-antagonists	ACE inhibitor-induced Cough	Heart failure Intolerance of other antihypertensive drugs	PVD[b]	Pregnancy Renovascular disease
Beta-blockers	Myocardial infarction Angina	Heart failure[c]	Heart failure[c] Dyslipidaemia PVD	Asthma/COPD Heart block
Calcium antagonists (dihydropyridine)	Elderly ISH	Elderly Angina	–	–
Calcium antagonists (rate-limiting)	Angina	Myocardial infarction	Combination with Beta-blockade	Heart block Heart failure
Thiazides	Elderly		Dyslipidaemia	Gout

[a] ACE-inhibitors 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.
[b] Caution with ACE-inhibitors and AII-antagonists in peripheral vascular disease because of association with renovascular disease.
[c] Beta-blockers may worsen heart failure, but in specialist hands may be used to treat heart failure.
COPD, chronic obstructive pulmonary disease; ISH, Isolated systolic hypertension; PVD, peripheral vascular disease.

have shown no consistent differences between regimens based on different drug classes.[55] With the exception of Syst-Eur,[8] Syst-China[102] and the CAPPP study,[103] most evidence from outcome trials is for treatment based on thiazide and/or beta-blockers.[58,59,104] Indirect comparison between SHEP,[59] based on diuretic treatment, and Syst-Eur based on a dihydropyridine calcium antagonist,[8] suggests that the outcome with these regimens was similar qualitatively and quantitatively.

Overall these outcome trials have shown significant reductions in stroke, by 38%, in coronary events, by 16%, and in cardiovascular mortality, by 21%.[57,58] The absolute benefit from treatment is smaller in women than men, but this is compatible with their lower cardiovascular risk.[58] The reduction in coronary events observed in all trials was less than the 20–25% reduction predicted from epidemiological observations.[24,105] The reduction in coronary events in trials based on low-dose thiazides has been significantly larger, at 28%, than those in trials of regimens based on high-dose thiazide or beta-blocker.[8,59,105] Low-dose thiazide based regimens also reduced cardiovascular and all-cause mortality significantly.[8,59,105] The larger benefit on coronary events observed in these trials with low-dose thiazides is not necessarily related to the dose of thiazide *per se*. It may be related to differences in age,[102] to more effective potassium conservation in these trials,[106] or to the play of chance. However higher doses of thiazide diuretics (bendrofluazide >2.5 mg or hydrochlorthiazide >25 mg daily) are unnecessary and should no longer be used.

Hypertensive patients controlled adequately on thiazides and beta-blockers have a persistent excess risk of coronary and stroke death,[24–30] as discussed before. These drug classes cause metabolic disturbances, including changes in lipids and glucose tolerance, that are dose-dependent.[104] Beta-blockers may also promote weight gain[6] and in the CAPPP study,[103] treatment based on beta-blockers and thiazides resulted in significantly more patients (+21%) developing diabetes over 5 years when compared to treatment based on ACE-inhibition. Nevertheless in these studies, body weight and metabolic changes did not adversely influence the efficacy of antihypertensive therapy at reducing cardiovascular morbidity and mortality. The question as to whether metabolically neutral classes of antihypertensive therapy will improve the outcome with regard to coronary and stroke reduction will only be answered by the results of large comparative trials now in progress, such as ALLHAT[107] and ASCOT.[108]

There have been suggestions that dihydropyridine calcium antagonists may increase the risk of coronary events, cancer, bleeding, depression, suicide, and other adverse events.[17,18] These signals of possible harm have emerged from case-control studies, or from subgroup interim analyses or secondary end-point analyses in randomised controlled trials. Evidence of this type cannot be ignored, but it is invariably open to bias and confounding and is therefore less robust when evaluating cause and effect. There is little biological plausibility for some of the adverse effects proposed. Controlled trials of dihydropyridine calcium antagonists such as PRAISE,[109] STONE,[110] and particularly Syst-Eur[8] have not supported these concerns about safety. Any remaining doubts may be resolved by large comparative trials in progress such as ASCOT and ALLHAT.

Nifedipine in capsule form should no longer be prescribed,[111] but otherwise the evidence available now strongly suggests that the benefits of dihydropyridine calcium antagonist treatment clearly exceed any risks, when they are prescribed for appropriate indications.

Dosage

The drug or formulation used should ideally be effective when taken as a single daily dose. An interval of at least 4 weeks should be allowed to observe the full response, unless it is necessary to lower blood pressure more urgently. The dose of thiazide diuretic should not be titrated up,[104] whereas other drug classes should be titrated according to the manufacturers' instructions. When the first drug is well tolerated but the response is insufficient, as is the case in about one half of all hypertensive patients, the options are to substitute an alternative drug or to add a second drug. Substitution of an alternative drug is appropriate when hypertension is mild and uncomplicated and the response to the initial drug was small. In more severe or complicated hypertension it is safer to add drugs stepwise until blood pressure control is attained. Treatment can be stepped down later if the blood pressure falls substantially below the optimal level.

In the HOT study, less than one-third of hypertensive patients were controlled by monotherapy and more than one-third required three or more drugs in combination to achieve optimal blood pressure control.[3] Similar conclusions were reported from the UKPDS study of hypertension in type 2 diabetes.[6] Thus, the majority of hypertensive people will require combinations of antihypertensive therapy to achieve optimal blood pressure control. The major classes of drug generally have additive effects on blood pressure when they are prescribed together.[112] Submaximal doses of two drugs result in larger blood pressure responses and fewer side effects than maximal doses of a single drug. Rational drug combinations combine drugs with different modes of action that are additive. Such combinations include; a diuretic with beta-blocker, diuretic with ACE inhibitor, beta-blocker with calcium antagonist, calcium antagonist with ACE inhibitor. For the third-line drug therapy commonly used combinations are diuretic, ACE inhibitor and calcium antagonist or diuretic, beta-blocker and calcium antagonist. Fixed-dose combinations are not widely used in Britain, but are convenient for patients and acceptable provided they are used as second-line treatment when monotherapy is ineffective; the individual drug components are appropriate; and there are no major cost implications. Certain drugs should not be co-prescribed for the treatment of hypertension, for example beta-blocker with verapamil or diltiazem; ACE-inhibitor with angiotensin II antagonist; or potassium-sparing diuretic with ACE-inhibitor.

Aspirin (Box 7)

In the HOT trial[3] aspirin 75 mg daily in treated hypertensive patients aged 50 years or more reduced cardiovascular events by 15% and myocardial infarction by 36%, but had no effect on fatal events. In the Thrombosis Prevention Trial of aspirin 75 mg daily for primary prevention,[35] 26% of those studied had treated hypertension. The outcome was similar to that of the HOT trial; a 16% reduction in all cardiovascular events, 20% reduction in myocardial infarction, and no effect on fatal events. In both trials the number of clinically-significant bleeding episodes caused by aspirin was similar to the number of cardiovascular events prevented by aspirin, suggesting that the margin between benefit and harm was narrow. The average cardiovascular event risk in the two trials was 1.0–1.5% per year, and the threshold for aspirin treatment for primary prevention in hypertension should therefore probably be set slightly higher than this. The HOT trial studied well-controlled hypertensives, and in the Thrombosis Prevention Trial aspirin was withheld when blood pressure was above 170/100 mm Hg. Furthermore, those who developed cerebral haemorrhage in the Thrombosis Prevention Trial had significantly higher systolic blood pressures before the adverse event (158 mm Hg *vs* 135 mm Hg in those with no stroke). Hypertension must therefore be controlled satisfactorily (to the Audit standard, <150/90 mm Hg) before starting aspirin treatment for *primary prevention* of cardiovascular disease in hypertensive subjects.

Given these considerations, aspirin 75 mg daily is recommended for hypertensive patients who have:

- no contraindication to aspirin
  and
- *Secondary Prevention:* Cardiovascular complications (myocardial infarction, angina, non-haemorrhagic cerebrovascular disease, peripheral vascular disease or atherosclerotic renovascular disease)

or

- *Primary Prevention:*
  blood pressure controlled to <150/90 mm Hg

BOX 7. Other Measures to Reduce Cardiovascular Risk

- Patients with established cardiovascular disease or at high risk according to the Joint British Societies 'Cardiac Risk Assessor' computer programme or CHD risk chart should be considered for aspirin (**A**) and statin therapy (**A**) as follows:
- *For primary prevention*, 75 mg aspirin is recommended for hypertensive patients aged 50 years or more who have satisfactory control of their blood pressure and either target organ damage or diabetes.
- *For primary prevention*, statin therapy is indicated when the serum total cholesterol is ⩾5.0 mmol/l and the 10 year CHD risk is ⩾30%.
- *For secondary prevention*, statin therapy is indicated when the total serum cholesterol is ⩾5.0 mmol/L and there is evidence of cardiovascular disease ie. angina/myocardial infarction etc. Aspirin is also indicated when there is evidence of cardiovascular disease.

and
Age ⩾50 years
and
target organ damage (eg, LVH, renal impairment, or proteinuria)
or
A 10-year CHD risk ⩾15% when estimated formally by Joint British Societies computer programme or risk chart.
or
Type II diabetes mellitus.

Patients with an estimated 10-year CHD risk of ⩾15% will have their cardiovascular risk reduced by 25% using antihypertensive treatment (see above). The addition of aspirin further reduces major cardiovascular events by 15%,[3,35] giving a NNT for 5 years of about 90 for one cardiovascular complication and 60 for one myocardial infarction prevented by aspirin. This benefit is acceptable given that aspirin treatment is simple and inexpensive.

HMG CoA reductase inhibitors (statins) (Box 7)

In the last 4 years the results of several controlled outcome trials have shown that statin treatment for secondary[31,33] and primary[32,34] prevention reduces major coronary events by 30%, reduces all-cause mortality significantly, and is safe, simple and well tolerated. Statin treatment also reduces the risk of stroke substantially in patients who have coronary heart disease.[31,33] In subgroup analyses the benefits were similar in hypertensive patients.[33,34] Given the persistent high risk of coronary and stroke death in treated hypertensive patients,[24–30] and the relation of this risk to serum cholesterol before and during treatment,[29] the implications of these trials for management of hypertension are large. There is general acceptance that statin treatment should be targeted at a specified threshold of coronary risk, and *not* at thresholds of lipid values.[113] Statin treatment could now be justified at a 10-year CHD risk of 6%,[34] but this would entail treating approximately half of all adults in Britain, and an even higher proportion of hypertensive patients. The main constraints on statin treatment at present are the workload for general practitioners and the enormous potential cost.

Our recommendations for statin therapy are designed to be consistent with the Joint Recommendations of the British Hypertension Society, British Cardiac Society, British Diabetic Association and British Hyperlipidaemia Association,[50] and with guidance from a national Standing Medical Advisory Committee (SMAC)[51] and those from the Scottish Intercollegiate Guideline Network (SIGN).[52] We emphasise that these are very conservative recommendations and represent minimum acceptable levels of treatment on the basis of existing evidence. We advise that statin treatment is prioritised to those at highest cardiovascular risk, as follows:

- Serum total cholesterol ⩾5.0 mmol/L; and
- *Secondary prevention:* myocardial infarction, angina, CABG or angioplasty, non-haemorrhagic cerebrovascular disease, peripheral vascular disease, or atherosclerotic renovascular disease.
- *Primary prevention:* 10-year CHD risk ⩾30% (≈10 year CVD risk of ⩾40%) estimated formally by the Joint British Societies cardiac risk assessor programme or risk chart (Figure 2).
- *Familial hypercholesterolaemia.*

Based on current evidence, the upper age limits for starting statin treatment suggested are 75 years for secondary prevention, and 70 years for primary prevention.

Follow-up

The frequency of follow-up for treated patients after adequate blood pressure control is attained depends upon factors such as the severity of the hypertension, variability of blood pressure, complexity of the treatment regimen, patient compliance, and the need for non-pharmacological advice. Review every 3 months is sufficient when treatment and blood pressure are stable, and the interval should not generally exceed 6 months. Those who have been hypertensive in the past, or who have untreated mild hypertension and a low estimated 10-year CHD/CVD risk, should have their blood pressure measured and their 10-year CHD/CVD risk estimated annually. The routine for follow-up visits should be simple; measure blood pressure and weight; enquire about general health, side effects and treatment problems; reinforce advice on non-pharmacological measures; and test urine for proteinuria annually. In general practice and hospital clinics trained nurses have an important role in the accurate measurement of blood pressure, and can advise and educate patients on aspects such as non-pharmacological measures and possible side effects from drugs. A large proportion of hypertensive patients disappear from regular follow-up for a variety of reasons. This may be reduced by thorough education of the patient about hypertension and its treatment, and provision of written information (see Appendix 2). A formal system of recall for those who miss routine appointments, using the practice computer, is desirable.

Special patient groups

Elderly hypertensives (Box 8)

Hypertensive people over the age of 60 years deserve special consideration for several reasons. Systolic blood pressure rises steadily with increasing age, and the prevalence of hypertension including isolated systolic hypertension (⩾160/<90 mm Hg) is more than 50% in those over 60 years.[23] These people have a high risk of cardiovascular complications when compared to younger hypertensives,[114] and antihypertensive treatment of diastolic[105] and isolated systolic[8,59] hypertension reduces this risk. Recent evidence also shows that antihypertensive therapy reduces the incidence of heart failure by 50%[115] and possibly dementia,[116] important complications of hypertension in this age group.[117] The

BOX 8. Elderly

- Absolute benefit from treatment is greater in the elderly (at least up to 80 years) than younger age groups (**A**).
- Optimum BP levels on treatment should be similar to those of younger patients if possible (**A**).
- Older patients tolerate antihypertensive treatment as well as younger age groups (**A**).
- Low-dose thiazides are the drug of choice for elderly hypertensive people.
- Dihydropyridine calcium antagonists are a suitable alternative when thiazides are contraindicated or poorly tolerated (**A**).

absolute benefit from treatment in the elderly is much larger than that for younger hypertensives[114] because of their higher absolute risk. Physicians may not be fully aware of this evidence,[19,20] and hence treatment is not implemented fully. This is not surprising, because before these outcome trials it was a widely and incorrectly held view that a rise in blood pressure with age was inevitable and harmless, and that isolated systolic hypertension was of no consequence. There was also concern that elderly people might tolerate antihypertensive drugs poorly, a concern that has been dispelled by detailed analyses of adverse effects experienced in the outcome trials.[114] Evidence for benefit from antihypertensive treatment extends until at least the age of 80 years, and regular blood pressure screening should continue until this age.

Once started, antihypertensive treatment should be continued after patients reach the age of 80. When hypertension is first diagnosed beyond the age of 80 there is no firm evidence to guide policy, but decisions should probably be based on biological rather than chronological age. Patients with newly-diagnosed hypertension after the age of 80 should be considered for treatment provided they are generally fit and have reasonable life expectancy, particularly if they have hypertensive complications or target organ damage.

Elderly hypertensives respond to non-pharmacological measures to lower blood pressure at least as well as younger patients.[13,78–81,86] Antihypertensive therapy is indicated and clearly beneficial in people aged 60 years or more, when blood pressure averages are >160 mm Hg systolic and >90 mm Hg diastolic.[114]

There is no firm evidence to guide policy for systolic blood pressure 140–159 mm Hg and diastolic blood pressures <90 mm Hg (borderline isolated systolic hypertension). Treatment is advised when there are cardiovascular complications or evidence of target organ damage. Management of those with borderline isolated systolic hypertension who do not have these complications represents a difficult dilemma. Most will have an estimated 10-year CHD risk in excess of 15% because of the profound effect of age on cardiovascular risk. A large absolute benefit from antihypertensive therapy would be anticipated on this basis. The treatment of choice, a low dose thiazide diuretic, is safe and effective in the elderly. On the other hand, direct evidence of benefit from randomised controlled trials is lacking for elderly people with blood pressures 140–159/<90 mm Hg and the high prevalence would mandate treatment of a large proportion of the elderly population. A firm recommendation is not appropriate in the circumstances. Doctors should consider the benefit anticipated and the resource implications in the context of other health and quality of life issues when reaching a treatment decision in individual elderly patients with borderline isolated systolic hypertension.

Low-dose thiazides are the accepted first-line treatment for the elderly.[114] Beta-blockers were second-line agents in many of the outcome trials, but are less effective than thiazides as first-line treatment and meta-analyses suggest that beta-blockers decrease stroke but no other cardiovascular events in this age group.[118] In Syst-Eur[8] treatment of isolated systolic hypertension in elderly patients was based on the dihydropyridine nitrendipine, and the outcome was similar to that with diuretic-based treatment in SHEP.[59] Hence a dihydropyridine calcium antagonist is a suitable alternative for elderly hypertensive patients when thiazides are ineffective, contraindicated, or not tolerated.

Hypertension in diabetes (Box 9)

Hypertension is common in diabetes and plays a major role in the development of diabetic macrovascular and microvascular complications.[6,119] The prevalence of hypertension differs in type I and type II diabetes. In type I diabetes, in the absence of nephropathy (microalbuminuria or proteinuria) the prevalence of hypertension is similar to that in the non-diabetic population.[120] In type 2 diabetes, hypertension (>140/90 mm Hg) is present in over 70% of patients.[121,122]

Type 1 diabetes without nephropathy: The threshold for intervention with antihypertensive therapy in patients without evidence of nephropathy is a systolic BP of ⩾140/90 mm Hg and the optimal blood pressure target is <140/80 mm Hg. The prin-

BOX 9. Diabetes

Type 1 Diabetes
- Threshold for starting antihypertensive treatment is ⩾140/90 mm Hg (**B**).
- Target blood pressure <140/80 mm Hg or lower if proteinuria present (**A**).
- BP reduction and ACE inhibitors reduce the rate of decline in renal function (**A**).

Type II Diabetes
- Threshold for starting antihypertensive treatment is ⩾140/90 mm Hg (**B**).
- Target blood pressure <140/80 mm Hg (**A**).
- Optimal first-line therapy is not yet established but trial evidence supports the use of: ACE inhibitors, beta-blockers, dihydropyridine CCBs, alpha-blockers, and low-dose thiazide diuretics (**B**).

ciples guiding the management of these patients is similar to that recommended for type 2 diabetes (see below).

Type 1 diabetes and diabetic nephropathy: Hypertension in type 1 diabetes often indicates the presence of diabetic nephropathy.[123] Blood pressure reduction and ACE-inhibitor treatment slow the rate of decline of renal function in overt diabetic nephropathy[4] and delay progression from the microalbuminuric phase to overt nephropathy.[123–125] ACE-inhibitors may have a specific renoprotective action in patients with incipient or overt nephropathy and are recommended as first-line therapy. If ACE-inhibitor treatment has to be discontinued because of persistent cough an angiotensin II receptor antagonist may be considered, although evidence for specific renoprotection by this drug class is awaited from ongoing clinical trials. The ACE-inhibitor should be titrated to the maximum dose recommended and tolerated. For renoprotection blood pressure control is most important,[126] and combinations of antihypertensive drugs are invariably required to achieve recommended blood pressure targets. Thiazide diuretics, calcium antagonists, cardioselective beta-blockers, and alpha-blockers are all suitable. The threshold for antihypertensive treatment in type 1 diabetes with nephropathy is ⩾140/90 mm Hg. The target blood pressure is <130/80 mm Hg,[3,123] or lower (<125/75 mm Hg) when there is proteinuria ⩾1 g/24 h.[126] Type 1 diabetic subjects with persistent microalbuminuria or proteinuria and 'normal' blood pressures, may also benefit from ACE-inhibition titrated to the recommended maximum dose.[4,123,127] It remains unclear whether this benefit accrues from ACE-inhibition *per se*, or the associated blood pressure reduction.[128] Type 1 diabetic subject with evidence of nephropathy are at very high risk of cardiovascular disease and may be considered for statin therapy if their total cholesterol is ⩾5 mmol/L and aspirin therapy if they if they satisfy the criteria set out above.

Hypertension in type 2 diabetes: Hypertension is very common in type 2 diabetes. It is strongly related to but not fully accounted for by obesity[121,129] and is highly predictive of cardiovascular and microvascular complications.[119,129] In the UKPDS study, antihypertensive therapy was more effective than tight glycaemic control[6] in protecting against microvascular and macrovascular disease, and was the only intervention that improved survival of patients with type 2 diabetes. Dramatic survival benefits were also observed in the elderly diabetic cohort treated with antihypertensive therapy in the SystEur trial.[7] Data from the UKPDS demonstrated that many type 2 diabetic subjects have established cardiovascular or microvascular disease at diagnosis and overall, have a 10-year cardiovascular event rate (predominantly coronary events) of >30%.[6,130] Since the presence of target organ damage or a 10-year CHD risk of ⩾15% per year would mandate treatment of a blood pressure of ⩾140/90 mm Hg, we recommend the threshold for intervention with antihypertensive therapy as ⩾140/90 mm Hg in type 2 diabetes.

In the diabetic cohort of the HOT study, blood pressure lowering improved cardiovascular outcome and importantly there were 50% fewer cardiovascular end-points when diastolic blood pressure was titrated beyond <90 mm Hg to <80 mm Hg.[3] Thus, for optimal blood pressure control, diastolic blood pressure should be targeted to below 80 mm Hg. There is less evidence to guide optimal systolic BP targets; thus we recommend systolic blood pressure should be maintained at the levels observed in those randomised to ⩽80 mm Hg in the HOT trial, ie, systolic blood pressure ⩽140 mm Hg.

The aforementioned studies have highlighted that at least two antihypertensive drugs are usually required to achieve optimal levels of blood pressure control in type 2 diabetes.[6] As regards the choice of drug therapy for hypertension in type 2 diabetes, one study suggested that regimens based on ACE-inhibition (captopril) and beta-blockade (atenolol) were equally effective at reducing macrovascular complications but the study was too small to exclude a difference.[131] Subgroup analysis of outcome trials have shown that other classes of antihypertensive drugs, ie, diuretics and dihydropyridine calcium antagonists, also improve the prognosis of diabetics with hypertension.[3,6,7,132] Thus, the optimal first-line drug is yet to be established but there is evidence from sub-group analyses of outcome trials in diabetic people for the safety and efficacy of ACE-inhibitors, dihydropyridine calcium antagonists, low dose thiazide diuretics and beta-blockers. The choice among these drug classes should be determined using the criteria set out earlier for non-diabetic patients.

Type 2 diabetes and diabetic nephropathy: There is much less evidence to guide practice in type 2 diabetic subjects with nephropathy. Hypertension accelerates the decline of renal function in type 2 diabetic patients with established nephropathy.[123,133] Moreover, antihypertensive therapy slows the progression of nephropathy in patients with type 2 diabetes.[123] ACE-inhibitors have an antiproteinuric action and delay progression from microalbuminuria to overt nephropathy,[123,125,134] but it is less clear whether they have a specific renoprotective action beyond blood pressure reduction in overt nephropathy complicating type 2 diabetes.

The high cardiovascular risk of type 2 diabetic subjects and the fact that many have established cardiovascular disease at diagnosis, means that therapeutic strategies other than blood pressure lowering are important. Aspirin should be offered to all patients with diabetes and hypertension using the criteria outlined above. Many type 2 diabetic people with hypertension will also require statin therapy for primary prevention because their estimated 10-year CHD risk is ⩾30%. These risk levels can be estimated from the Joint British Societies computer programme which adjusts for the higher CHD risk of diabetic subjects, or the risk chart which contains boxes dedicated to CHD risk estimation in diabetic subjects.[50] This multifactorial approach should be

complemented by efforts to optimise glycaemic control and continued non-pharmacological advice because many diabetic patients, particularly those with type 2 diabetes are overweight and have many other cardiovascular risk factors.[121,122,129,135]

Renal disease and hypertension

Renovascular disease (renal artery stenosis) is relatively uncommon, but is the most frequent curable cause of hypertension. Routine investigation of all hypertensive patients is not justifiable, but doctors should be aware of important clues suggesting renovascular disease. These are:

- onset of hypertension before the age of 30;
- documented sudden onset of hypertension or recent worsening of hypertension in middle age;
- accelerated (malignant) hypertension;
- resistant hypertension (to a three drug regimen);
- renal impairment of unknown cause;
- elevation of serum creatinine by ACE inhibitor or angiotensin II antagonist treatment;
- peripheral vascular disease or severe generalised atherosclerotic disease;
- recurrent pulmonary oedema or heart failure with no obvious cause.

Patients with any of these features should be referred for specialist advice because the investigations required to confirm or exclude renovascular disease are complex.

Hypertensive patients with elevated serum creatinine or proteinuria may have parenchymal or obstructive renal disease, and should be referred for specialist evaluation. Accelerated (malignant) hypertension requires immediate hospital treatment because it causes rapid loss of renal function that can be irreversible if untreated. Otherwise hypertension *per se* does not cause renal failure,[136] and the corollary is that renal impairment in the absence of previous accelerated phase hypertension suggests primary renal disease or renovascular disease.[136] In patients with chronic renal impairment, hypertension accelerates the rate of loss of renal function, and good blood pressure control is essential to retard this process.[137] Whether ACE inhibitors have a specific renoprotective effect in non-diabetic renal failure, over and above their antihypertensive action, remains uncertain.[137,138] Meta-analysis of all controlled trials showed a 30% reduction in incidence of end-stage renal failure with ACE-inhibitors,[138] but this may not all be explained by additional blood pressure reduction.[138] ACE-inhibitors reduce proteinuria, and are probably renoprotective in patients with proteinuria $\geqslant$3 g/day or who have rapidly-progressing renal failure[139,140] but may not be renoprotective in those with polycystic kidney disease.[138] ACE-inhibitors may cause or worsen renal impairment in patients with critical renovascular disease, thus they should be used with caution, preferably with specialist supervision, in patients with advanced chronic renal impairment. Blood pressure is particularly salt sensitive in patients with impaired renal function, and dietary salt reduction is important. Thiazide diuretics may be ineffective in patients with renal impairment, and loop diuretics (ie, frusemide), often in high dose, are frequently required. The dose of renally-excreted antihypertensive drugs may need to be adjusted.

The threshold for antihypertensive treatment is $\geqslant$140 mm Hg systolic, or $\geqslant$90 mm Hg diastolic for patients with persistent proteinuria or renal impairment. Optimal blood pressure control is $<$130/85 mm Hg[126,141] and reducing blood pressure to $<$125/75 mm Hg may produce additional benefit in patients with chronic renal disease of any aetiology and proteinuria of $\geqslant$1 g per 24 h.[126,141] Patients with renal failure have a very high risk of cardiovascular complications,[142] and may need aspirin or statin treatment in addition to non-pharmacological measures to reduce their cardiovascular risk burden risk.

Hypertension in pregnancy

This has been reviewed elsewhere.[143,144] It occurs in up to 10% of pregnancies, and may be the first sign of impending pre-eclampsia, a potentially more serious condition of the second half of pregnancy and the puerperium. Diastolic blood pressure should be measured at the disappearance of sounds (phase V) and not at muffling (phase IV) as recommended in the past.[145,146]

Idiopathic hypertension in pregnancy

Care must be taken to distinguish between chronic hypertension (defined as a blood pressure of $\geqslant$140/90 mm Hg before 20 weeks' gestation) and pre-eclampsia. Elevated blood pressure before 20 weeks' gestation usually means that hypertension preceded pregnancy. This will commonly be idiopathic but clinical evaluation is needed (Table 2) to exclude other causes such as renal disease, Conn's syndrome or co-arctation. Phaeochromocytoma is rare but may cause sudden death in pregnancy and should be excluded by measuring urinary catecholamines if there are suggestive features. An apparent onset of hypertension after 20 weeks' gestation may reflect hypertension that was undetected prior to pregnancy, and disguised by the blood pressure fall of early-mid pregnancy. All women with hypertension in pregnancy should have their blood pressure checked post-natally.

Meta-analysis of trials of antihypertensive drugs in pregnancy shows reduction in the risk of progression to severe hypertension and fewer hospital admissions.[147] Firm evidence is not available on the optimal threshold for treatment. There is consensus that treatment is essential at $\geqslant$170/110 mm Hg but many treat blood pressure at levels $\geqslant$140/90 mm Hg. Women with essential hypertension are at increased risk of pre-eclampsia and intra-uterine growth restriction (IUGR). Management should therefore include frequent blood pressure checks, preferably once a week, urinalysis and an assessment of foetal growth. Hospital referral should be made if there is poorly controlled hypertension, new onset proteinuria or suspicion of IUGR.

Pre-eclampsia

Criteria for the diagnosis of pre-eclampsia include: a rise in blood pressure of >15 mm Hg diastolic or >30 mm Hg systolic from early pregnancy, or diastolic blood pressure of >90 mm Hg on two occasions 4 h apart or >110 mm Hg on one occasion and proteinuria (1+ is a indication for referral and >300 mg/24-h is the criterion for diagnosis). It is emphasised that 30% of eclamptic convulsions occur in the absence of either raised blood pressure or proteinuria. Risk factors for pre-eclampsia are: first pregnancy, change of partner, previous pre-eclampsia, family history of pre-eclampsia, idiopathic hypertension, chronic renal disease, diabetes, SLE, multiple pregnancy and obesity. Women with pre-eclampsia generally have no symptoms and can only be detected by routine screening. When present, the most frequent symptoms are headache, visual disturbance (often 'flashing lights'), vomiting, epigastric pain, oedema (particularly facial oedema). These symptoms in conjunction with raised blood pressure require urgent referral and treatment. Women rarely present with a convulsion, but a first fit in the second half of pregnancy with no other known cause is highly suggestive of eclampsia.

Choice of antihypertensive therapy in pregnancy

Evidence underpinning the choice of antihypertensive therapy in pregnancy is inadequate to make firm recommendations. Methyldopa remains the antihypertensive drug of choice for idiopathic hypertension or pre-eclampsia because of its long and extensive use without reports of serious adverse effects on the foetus.[148] Calcium antagonists (especially nifedipine) and the vasodilator hydralazine are commonly used as second-line drugs. Labetolol (alpha and beta-blocker) is also widely used as a second-line agent, particularly for resistant hypertension in the third trimester.[149] Other beta-blockers are used less often, particularly before 28 weeks' gestation because of concerns that they may inhibit foetal growth.[150] Meta-analysis of controlled trials of diuretics has shown a reduced incidence of pre-eclampsia,[151] although no benefit was shown on foetal outcome. In practice, diuretics are used little for the management of hypertension on theoretical grounds that they have the potential to further reduce the already decreased circulatory blood volume in women with pre-eclampsia.[152] However, there is nothing to suggest that low-dose thiazide diuretics in women with pre-existing hypertension are harmful and they may be continued through pregnancy. ACE inhibitors should be avoided because they may cause oligohydramnios, renal failure, hypotension and intrauterine death in the foetus.[153] Hypertensive women who plan pregnancy, or who become pregnant whilst on antihypertensive treatment, should be advised to change their therapy to one of the drugs recommended for the treatment of hypertension in pregnancy. It is usual to switch from such agents back to the previous antihypertensive regimen after delivery.

Oral contraceptives and hypertension

Limited trial data[154] and other observational studies[155,156] suggest that combined oral contraceptives (OCs) have a small adverse effect on blood pressure with average increases of 5/3 mm Hg.[157] In a small proportion of women (≈1%) severe hypertension may be induced.[158] The increase in blood pressure appears to be idiosyncratic in that it may occur many months or years after first using a combined OC. No particular subgroups of OC users have been clearly identified as being susceptible to blood pressure increases and the cause for the blood pressure has not been established.[157] Moreover, the dose-related effects of oestrogens and progestogens on blood pressure have not been clearly established. As the blood pressure response to any combined OC preparation is unpredictable, and there is a small increase in cardiovascular risk associated with OC use[159] blood pressure should be measured before starting OC use and then 6 monthly thereafter.

Observational data suggest that oral progestogen-only contraceptive pills (POPs) do not on average induce an increase in blood pressure.[156,160] Although, clinical trial evidence in support of this conclusion is very limited, hitherto, the POPs have been recommended for use in women with a previous history of combined OC-induced hypertension, or those women with hypertension wishing to use an oral contraceptive.

Pending further evidence, the use of the combined OC is not absolutely contraindicated for women who are already hypertensive or those who develop hypertension during use of combined OCs. We recommend, however, that other non-hormonal forms of contraception should be sought, particularly if other risk factors for cardiovascular disease (eg, smoking or migraine) coexist. In those women for whom other methods of contraception are unacceptable, changing to a POP with careful monitoring of blood pressure is recommended. It should be recognised that data on the impact of POPs on cardiovascular risk is limited and that POPs are less effective contraceptives than combined OCs. This is most relevant for younger women (<35 years) who are more fertile and in whom the risk of pregnancy associated adverse cardiovascular events outweighs that related to the use of the combined OC. Advice to modify coexisting risk factors (eg, smoking, excess alcohol consumption) should be supplied. If blood pressure does not fall below 160/100 mm Hg, antihypertensive medication should be initiated.

Hormone replacement therapy (HRT) and hypertension

Observational data[161] and a recent clinical trial examining various HRT formulations,[162] have suggested that in general, the use of HRT is not associated with an increase in blood pressure. HRT is not contraindicated for women with hypertension and women with hypertension should not be denied access to HRT as long as BP levels can be controlled by antihypertensive medication. Given the uncertainties regarding cardiovascular protection associa-

ted with 'opposed HRT' (containing oestrogen and progestogen[163] it is prudent to monitor blood pressure after the initiation of HRT, 2–3 times in the first 6 months, then 6 monthly. Rare idiosyncratic rises in blood pressure in patients receiving HRT have been reported. Consequently, HRT should be temporarily discontinued in women with resistant hypertension to assess the contribution of HRT to the blood pressure.

Hypertension in ethnic groups

Hypertension is particularly common in African-Caribbean blacks, with a prevalence as high as 50% over the age of 40 years.[164] Among this group hypertension tends to be more severe and associated with a higher risk of complications, particularly stroke, renal failure, and left ventricular hypertrophy. The hypertension is often sensitive to dietary salt restriction and in those without evidence of target organ damage, a low salt diet may occasionally be sufficient to control blood pressure. Black subjects are more responsive to diuretic and calcium antagonist treatment.[10] ACE-inhibitors and beta-blockers may be ineffective in monotherapy in black subjects because the renin angiotensin system is frequently suppressed.[10] However, these patients may respond to ACE-inhibition or beta-blockade when given in combination with drugs that activate the renin-angiotensin system, ie, diuretics, calcium channel blockers or alpha-blockers. In resistant hypertension a combination of diuretic, calcium antagonist, ACE-inhibitor and/or alpha-blocker is particularly effective.

British South Asians (from the Indian subcontinent) also have a high prevalence of hypertension, are commonly insulin resistant, and have a high prevalence of type 2 diabetes.[165,166] They are at increased risk of stroke and particularly high risk of coronary heart disease.[167] The limited evidence available suggests that the response to drug treatment of hypertension in South Asian patients is similar to that in white Europeans. Important points in management are particular emphasis on glucose tolerance, lipids, and the increased coronary risk.[165,166] Clear advice is needed to reduce fat and refined sugar intake and to increase exercise. Good control of blood pressure is particularly important in those with diabetes, and aspirin and/or statin treatment may be indicated for those at high risk of coronary heart disease.

Implementation and audit

Challenges for the future

The British Hypertension Society believes that the following objectives should have high priority:

- To promote the primary prevention of hypertension and cardiovascular disease by changes in the diet and lifestyle of the whole population.
- To increase the detection and treatment of undiagnosed hypertension by routine screening and increase awareness of hypertension among the public.
- To increase the proportion of patients on antihypertensive treatment who are controlled to optimal blood pressure levels.
- To reduce the cardiovascular risk of treated hypertensive patients by non-pharmacological measures, and by appropriate use of aspirin and statin treatment.
- To increase identification and treatment of patients with mild hypertension who are at high cardiovascular risk, for example;
  - elderly patients
  - in those with isolated systolic hypertension
  - diabetic people
  - those with target organ damage or multiple risk factors
- To promote continuation of drug treatment, and compliance with treatment, by optimising the choice and use of drugs, minimising side effects, and increasing information and choice for patients.

Implementation

Realisation of these objectives will depend largely on the efforts of doctors and nurses in general practice. Surveys revealing incomplete detection, treatment and control of hypertension[21–23] indicate a serious failure to implement the knowledge we have, although there has been some improvement in recent years.[23] Current methods of delivering medical care to hypertensive people are evidently unsatisfactory, a problem that is not unique to Britain. A WHO-WHL audit project[168] in several European countries showed that no more than 29% of all treated hypertensive patients had attained normal blood pressure. The same study revealed a failure to implement non-pharmacological measures to reduce blood pressure and cardiovascular risk, and inadequacy of information and education for patients. A study from Australia[169] showed that hypertensive patients were no more knowledgeable than normotensive controls about hypertension, and 70% of patients expressed a desire for more information about their condition. The quality of care of hypertensive patients might be enhanced[170] by improved screening or case-finding; use of computer-based systems and protocols to support clinical decision making; workshops on hypertension for general practitioners and practice nurses; clinical audit; improved doctor-patient or nurse-patient communication; and increased patient education.

It is suggested that all practices or primary care groups should develop a protocol for hypertension management that covers the screening policy; initial evaluation and investigation; implementation of non-pharmacological measures; formal estimation of cardiovascular risk; treatment policy for antihypertensive drugs, aspirin and statins; treatment targets; policy for follow-up; and methods for identifying and recalling patients who drop out of follow-up. Written information should be available for patients about hypertension and its treatment, and on non-pharmacological measures to reduce blood pressure and cardiovascular risk (see Appendix 2). The practice policy should detail those aspects of manage-

ment that are in the province of the practice nurse and of the doctor, and the indications and procedure for passing management from nurse to doctor or vice versa. It is recommended that implementation of the practice policy should be audited periodically.

Audit points

Several aspects of hypertension management lend themselves readily to audit procedures that are objective and reasonably simple. For example:

- the proportion of all adults in the practice who have had a blood pressure measurement in the last 5 years;
- the proportion of all hypertensives given non-pharmacological advice;
- the proportion of all hypertensives given antihypertensive therapy;
- the proportion of hypertensives receiving antihypertensive therapy who have suboptimal control, ie, blood pressure levels ⩾150 mm Hg systolic and ⩾90 mm Hg diastolic BP;
- the proportion of patients lost from follow-up; or of treated patients who have not been reviewed within the last 6 months;
- the use of aspirin and statins by those who require secondary prevention; or their use when indicated for primary prevention, ie, when the estimated 10-year CHD risk is ⩾15% (aspirin) or ⩾30% per year (statins).

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Appendix 1

How to measure 10-year coronary heart disease risk

The British hypertension society recommends that the computer programme 'Cardiac Risk Assessor' based on the Framingham risk function and issued by the Joint British Societies should be used wherever appropriate to estimate 10 year CHD risk. This programme will also estimate 10-year CHD and stroke risk simultaneously.

The computer programme or risk chart should not be used in patients with: (i) established CHD or atherosclerotic disease, or (ii) familial hypercholesterolaemia, because these patients are already at sufficiently high CHD/CVD risk to justify intervention.

The following information is needed:

(1) Name
(2) Sex
(3) Age
(4) Systolic BP (mm Hg)
(5) Diastolic BP (mm Hg)
(6) Smoking status
(7) Serum cholesterol (any units)
(8) HDL cholesterol (same units as serum cholesterol)
(9) Diabetic or non-diabetic
(10) Presence of LVH on the ECG

The 10-year % probability of developing CHD (fatal or non-fatal myocardial infarction) and stroke is estimated by the cardiac risk assessor computer programme. When the CHD risk and stroke risk are added, this equates to 10-year CVD risk.

Family history: The estimated 10-year CHD risk should be adjusted upwards by a factor of 1.5 for patients who have a first degree male relative developing CHD or other atherosclerotic disease before the age of 55 years, or a female first degree relative with a similar history before the age of 65 years.

Ethnic minorities: The Framingham risk equation has not been validated in these populations.

Joint British Societies CHD Risk Chart: When the use of the computer programme is impractical, the Joint British Societies CHD risk chart provides a useful alternative for the estimation of 10-year CHD % risk. CVD risk can easily be derived from CHD risk, eg, a 10-year CHD risk of $\geqslant$15% is equivalent to a 10-year CVD risk of $\geqslant$20%, or a 10-year CHD risk of $\geqslant$30% is equivalent to a 10-year CVD risk of $\geqslant$40%.

Appendix 2

Available from the *British Hypertension Society Information Service*

Blood Pressure Unit
St George's Hospital Medical School
Cranmer Terrace
London SW17 0RE
Tel: 0181 725 3412
Fax: 0181 725 2959
E-mail: bhsis@sghms.ac.uk
Website: www.hyp.ac.uk/bhsinfo and
www.hyp.ac.uk/bhs

- Patient information booklet:
  –Understanding High Blood Pressure.
- Patient fact sheets:
  –Self-help Measures.
  –Antihypertensive Drugs
  –Blood Pressure Measurement
  –Reducing Dietary Salt
  –Blood Pressure and Kidney Disease
- Diet sheet:
  –Healthy Eating Diet Sheet

Available from *BMJ Publications*

- Blood Pressure Measurement – Recommendations of the British Hypertension Society. 3rd Edition. O'Brien E *et al* (ed) (£4.95).
- BHS/BMJ CD-ROM – Recommendations for Blood Pressure Measurement.

The Joint British Societies 'Cardiac Risk Assessor' Computer programme and copies of the Joint British Societies CHD risk assessment chart (Figure 2) can be downloaded from the BHS web site.