Recommendations for prevention, diagnosis and management of hypertension and cardiovascular risk factors in sub-Saharan Africa

Daniel Lemogoum, Yackoob Kassim Seedat, Abdul Fattah Biola Mabadeje, Shanti Mendis, Pascal Bovet, Basden Onwubere, Kathleen Ngu Blackett, Claude Lenfant, Jean René M'buyamba Kabangu, Pierre Block, Mohamed Belhocine and Jean Paul Degaute, on behalf of the International Forum for Hypertension control and prevention in Africa (IFHA)

Journal of Hypertension 2003, **21**:1993–2000

Keywords: recommendations, prevention, diagnosis, management and control of hypertension, sub-Saharan Africa

Correspondence and requests for reprints to Professor Y.K. Seedat, Nelson R. Mandela School of Medicine, Faculty of Health Sciences, University of Natal, Durban, South Africa.

Tel: +27 31 260 4222; fax: +27 31 205 2984; e-mail: seedaty1@nu.ac.za

Sponsorship: The three expert consultative meetings that lead to the elaboration of these recommendations on hypertension in SSA and the final meeting held in Brussels on 10–12 May 2003 were supported and sponsored by European Union–Research Directorate Branch. WHO regional office for Africa co-sponsored the first African experts meeting held in Libreville in November 2001.

Introduction

The need for a common set of recommendations for sub-Saharan Africa

The available data from a few countries in the sub-Saharan Africa (SSA) have highlighted the increasing importance of non-communicable diseases (NCD) in this region, and these countries have taken steps to develop relevant policies and programs to address this issue [1,2]. It is likely that cardiovascular diseases (CVD) are particularly poorly detected and treated in the primary health care setting in SSA. Countries in SSA should therefore be encouraged to establish country-specific recommendations for the prevention and management of NCD [as already recommended by the World Health Assembly and the World Health Organization (WHO) Regional Committee for Africa]. Blacks in SSA develop complications of stroke, heart failure, renal failure and peripheral vascular disease from hypertension [3]. At present, coronary heart disease is relatively uncommon, probably because blacks in SSA have lower serum cholesterol levels and higher high-density lipoprotein cholesterol levels compared to the Caucasian population group in the Western World and African-Americans [3]. Although there are good studies on the response and tolerability of antihypertensive drugs in SSA, there are no long-term morbidity and mortality data available. The ALLHAT Study, which included a large subset sample of African-Americans, and the AASK study assessed long-term morbidity and mortality [4,5].

In the face of the emerging CVD epidemic faced by African countries, African heads of state and government adopted the Durban Decision in 2001 (AHG/Decision 179) that emphasized the need to address arterial hypertension and CVD risk control in Africa. The Erasmus Hospital–Free Brussels University (Belgium), with the assistance of the European Commission, took leadership to support this program, and subsequent support was gathered from the World Health Organization and several other partners, such as the World Hypertension League, World Heart Federation, US Center for Diseases Control, Global Forum for Health Research in Developing countries and African Union. A Conference on Hypertension and Cardiovascular Disease in the African region was held at the Erasmus Hospital–Free Brussels University on 10–12 May 2003 and was attended by 70 prominent African and international experts on hypertension and CVD. Current knowledge related to hypertension and CVD in Africa was reviewed and participants identified recommendations on hypertension for the SSA region as a priority. A draft of recommendations for the diagnosis and management of hypertension in SSA was prepared during the conference on the basis of conclusions of three previous experts consultative meetings held in Africa in 2001 and 2002. The main principles of such recommendations were discussed and agreed upon in a plenary session, and the document was finalized by an open group of these experts through electronic correspondence (Chairman: Y.K. Seedat).

The recommendations follow the main lines stated in the 2003 WHO/ISH Statement on Management of Hypertension [6], the 2002 WHO Cardiovascular Risk Management Package in Low- and Medium-Resource

0263-6352

DOI: 10.1097/01.hjh.0000098140.70956.1a

Settings [7], the American JNC 7 guidelines [8], the European guidelines [9] and a consensus statement of the Hypertension in African-Americans working group of the International Society on Hypertension in Blacks [10]. Although it is important to consider the science of medicine for the treatment of hypertension, particular consideration should be given to cost-effectiveness and affordability because many countries in SSA have severe resource constraints. In some of them, the health budget per capita does not exceed US$10 per year and this is severely insufficient to address the needs posed by the double burden of non-communicable diseases and infectious diseases, including AIDS.

Blood pressure measurement and clinical evaluation

Blood pressure detection and confirmation

This is a vital clinical sign but poorly understood by health care categories. The European Society of Hypertension recently published detailed recommendations which the IFHA endorses [11]. It should be emphasized that the diagnosis, management, treatment, epidemiology and research of hypertension is dependent on accurate measurement of blood pressure and that, if blood pressure measurement is inaccurate, it follows that incorrect decisions will be made [11]. Health care professionals are strongly advised to measure blood pressure at each encounter with the patient or other health care seeker. Blood pressure should be measured in subjects comfortably seated for at least 10 min in a quiet setting. Because of the variability of measurements of casual blood pressure, decisions based on single measurements will result in erroneous diagnosis and inappropriate management. Reliability of measurement is improved if repeated measurements are made. At least two measurements at 1-min intervals should be taken at each visit with a repeat measurement if there is uncertainty, if blood pressure differs by > 5 mmHg between two readings or there is distraction. It is better to perform a few carefully taken measurements rather than carry out a number of hurried measurements [11]. Self-measurement of blood pressure (or in pharmacy) is a simple way to both monitor and assess the 'white coat effect', which has considerable clinical and economic importance because wrongly labelled hypertensive patients are likely to be given unneeded drugs for years.

Hypertension is thus defined as a persistently elevated systolic and/or diastolic arterial blood pressure of 140/90 mmHg or more in subjects aged 15 years and above (Table 1). The relationship between blood pressure and risk of CVD events is continuous and consistent across the entire blood pressure range starting from 115/75 mmHg [6,8]. However, it is acknowledged that individuals with blood pressure readings in the range 120–139/80–89 mmHg have a non-optimal pressure (called pre-hypertension in the American guidelines) and are at increased risk for progression of hypertension. In the context of SSA, such patients should be advised to have their blood pressure checked once per year and advised to follow non-pharmacological measures in the interval.

Table 1 JNC 7 classification of blood pressure for adults aged 18 years and older [8]

Blood pressure classification	Systolic blood pressure (mmHg)[a]	Diastolic blood pressure (mmHg)
Normal	< 120	And < 80
Prehypertension	120–139	Or 80–89
Stage 1 hypertension	140–159	Or 90–99
Grade 2 hypertension	$\geqslant 160$	Or $\geqslant 100$

[a]When a patient's systolic and diastolic blood pressures fall into different categories, the higher category should apply. In SSA, the age under consideration is 15 years and above.

Recommended devices

When it is not feasible to use automated devices validated by international standardized protocols, good quality mercury devices are generally recommended. Automated devices should be used if independently validated devices are available at affordable prices. When arriving at a decision to use automated devices, consideration must be given to the cost of batteries, annual servicing charges and durability in addition to the purchase price. In certain settings, anaeroid devices may have to be used as they are least expensive. However, they can become inaccurate without the user being aware of it and require calibration every 6 months [11]. An up-to-date list of validated devices is available on the World Wide Web at http://www.dableducational.com) [12].

Self-measurement/ambulatory monitoring

Twenty-four-hour ambulatory blood pressure monitoring should not be part of routine blood pressure evaluation but would be valuable in subjects with refractory hypertension, borderline hypertension, episodic hypertension and white-coat hypertension. Due to the unavailability of such equipment in most parts of SSA, repeated self-measurement using electronic devices may be an alternative. In general, it has been shown that office values of 140/90 mmHg approximately correspond to 24-h average values of 125/80 mmHg [9].

Clinical evaluation

Evaluation: history, clinical examinations, routine laboratory investigations

As emphasized by the WHO and other guidelines [7,9], blood pressure should not be assessed in isolation, but be part of a more comprehensive assessment that includes other risk factors of CVD. This will allow assessing a patient's total CVD risk and clinical management (pharmacological and non-pharmacological)

can be better adapted. In all cases, age, sex, smoking and alcohol consumption, physical activity, weight – height (body mass index, BMI) and the personal and family history of premature CVD and other risk factors (particularly diabetes) should be recorded.

The clinical and laboratory evaluation of the hypertensive patient should be conducted with four aims:

(i) To confirm a persistent elevation of blood pressure and determine the level.
(ii) To determine the presence of target organ damage and quantify its extent.
(iii) To search for other cardiovascular risk factors and clinical conditions that may influence the prognosis and treatment.
(iv) To identify or exclude secondary causes of hypertension.

History and physical examination

- History should inquire into age, gender, tobacco habits, physical activity, previous or current antihypertensive medication, use of drugs or substances likely to raise blood pressure and daily intake of salt and fruits and vegetables in the diet.
- Personal and family history should include enquiry about CVD (stroke, heart failure, angina, heart attack) kidney disease and risk factors for diabetes, and possibly hypercholesterolemia, obesity, premature CVD among parents and siblings.
- Full physical examination should be performed including murmurs at heart level and bruit in main arteries (carotids, abdominal aorta, femoral), artery pulses (e.g. feet), and evidence of left or right heart failure.
- Weight should be obtained at each visit. The current normal weight of patient should be determined using BMI (< 25 kg/m^2) and health workers should inform patients on their normal weight. It is advisable to also measure waist–hip circumference to assess central obesity [13].
- Routine laboratory investigations
- Standard routine laboratory investigations will depend on available facilities and resources [3,7]. If feasible, hypertensive patients should undergo at least the following:
  (i) Urinalysis for blood, protein and glucose. Repeat tests and consider further analyses if any of these tests are positive.
  (ii) Microscopic examination of urine.
  (iii) Blood chemistry for potassium, creatinine, fasting and/or random glucose. Always repeat fasting blood glucose if the test is abnormal and the patient is unaware of previous diabetes.
  (iv) Fundoscopy, electrocardiograph and serum lipids (facilities for these investigations may not be available in many settings in SSA). Serum lipids are not an important investigation in Blacks of SSA because of their low levels [3].
- Further investigations to exclude secondary hypertension and target organ damage should be guided from the history, examination, routine investigations and patient resources.

Risk factor identification and stratification

Rationale for cardiovascular risk evaluation

A comprehensive risk factor evaluation is central to the management of hypertension. (Table 2). A patient with hypertension and no risk factors will not be managed clinically in the same way as a patient with the same level of hypertension together with several other risk factors. Factors that affect blood pressure management are divided into: (i) risk factors for CVD; (ii) target organ damage (Table 3) and (iii) associated clinical conditions.

Management of high blood pressure and cardiovascular risk factors

Trexatment goals

- To achieve blood pressure < 140 mmHg and < 90 mmHg in those with uncomplicated hypertension.
- To achieve target blood pressure of < 130 mmHg and < 80 mmHg in those with established CVD (coronary heart disease or cerebrovascular disease) or diabetes or chronic renal disease.
- To manage identified risk factors, target organ damage and associated clinical conditions.
- To institute appropriate patient education.

Table 2 Risk stratification (ESH/ESC guidelines) [9]

	Blood pressure (mmHg)		
Other risk factors and disease history	Grade 1 (mild hypertension) SBP 140–159 or DBP 90–99 mmHg	Grade 2 (moderate hypertension) SBP 160–179 or DBP 100–109 mmHg	Grade 3 (severe hypertension) SBP $\geq$ 180 or DBP $\geq$ 110 mmHg
I No other risk factors	Low risk	Medium risk	High risk
II One or two risk factors	Medium risk	Medium risk	Very high risk
III Three or more risk factors of target organ damage or diabetes	High risk	High risk	Very high risk
IV Associated clinical conditions	Very high risk	Very high risk	Very high risk

SBP, Systolic blood pressure; DBP, diastolic blood pressure.

Table 3 Cardiovascular risk factors and target organ damage [8,9]

Factor affecting management
Major risk factors
Hypertension
Smoking
Obesity (BMI ⩾ 25 kg/m^2), especially central obesity
(abdominal circumference: men, ⩾ 102 cm; women, ⩾ 88 cm)
Physical inactivity
Diabetes mellitus
Dyslipidemia
Proteinuria, microalbuminuria
Age (> 55 years for men; > 65 years for women)
Family history of premature CVD (men < 55 years or women < 65 years)
New emerging risk factor
Metabolic syndrome
Target organ damage
Heart
• Left ventricular hypertrophy
• Heart failure
• Angina or prior myocardial infarction
• Previous coronary revascularization
Brain
• Stroke or transient ischemic attack
Chronic kidney disease
Peripheral artery disease
Retinopathy

BMI, body mass index; CVD, cardiovascular disease.

Lifestyle modification

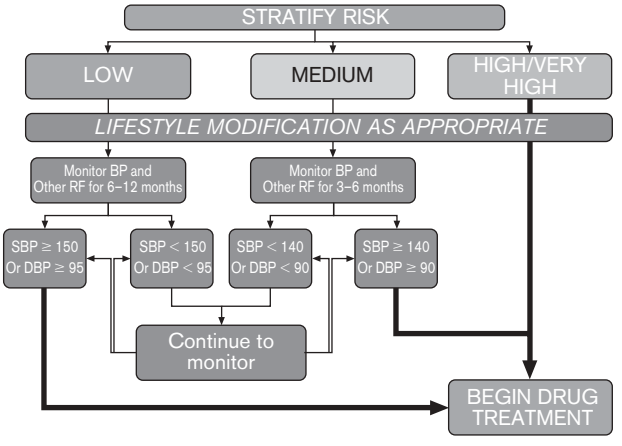
The WHO-CVD Risk Management Package for Low- and Medium- and High-Resource Settings [7] provides guidance for applying lifestyle management in health care settings with different resource levels (Fig. 1).

Lifestyle modification is required for patients with prehypertension and should be a key component of management of all hypertensive patients.

When high blood pressure has been confirmed, the following lifestyle changes should be started where appropriate:

- Weight reduction if BMI is ⩾ 25 kg/m^2.
- Limit salt intake to no more than one teaspoon (5 g) per day.
- Limit alcohol intake to not more than two standard drinks per day for men and not more than one standard drink for women.
- Abstain from tobacco use.
- Promote a 'balanced' diet. This includes a diet:
  (i) low in saturated fats (animal fats and some vegetables oils) and sugar. Oils consumed for cooking should be low in saturated fats (hence avoid coconut oil, butter and lard and limit palm oil);
  (ii) high in fibres, unrefined carbohydrates, fruits and vegetables; avoid preserved foods because of their high salt intake;
  (iii) including low-fat dairy products; and
  (iv) including fish.
- Take regular aerobic exercise for 30–60 min on most days of the week (e.g. brisk walking).

Fig. 1



WHO-CVD Risk Management for Low and Medium Resource Settings. Overall management strategy. BP, Blood pressure; RF, risk factor; SBP, systolic blood pressure; DBP, diastolic blood pressure. WHO Lifestyle Management Package for low and medium resource settings [7].

Pharmacological treatment

Principles of drug management

- Because the main benefits of antihypertensive therapy are due to lowering blood pressure, in the absence of compelling indications, low-dose thiazide diuretics (e.g. hydrochlorothiazide 12.5 mg daily) should be used to initiate therapy in almost all categories of patients (including diabetics) as they have shown to constitute the basis of treatment in most outcome trials in hypertensive patients [3,4].
- The choice of drug *vis-à-vis* compelling and possible indications is shown in Table 4. This is based on new outcome trials of antihypertensive treatment [14].
- If a low dose of a single drug is well tolerated but the blood pressure is not controlled, it is reasonable to increase the dose of the same drug (in the absence of side-effects) or to add a small dose of a second drug from a different class [**using by preference generic preparation for all drugs**, thiazide-like diuretic, long-acting calcium channel blocker (CCB), β-blocker, angiotensin-converting enzyme inhibitor (ACEI) or reserpine] Most patients require two or more antihypertensive drugs to achieve treatment goals and diuretics should generally be one of them [6,8,9,15–17].
- Centrally acting drugs such as methyldopa, clonidine and α-blockers such as prazosin and doxazosin, or hydralazine should be reserved for resistant hypertension.
- In the absence of any published mega-trial directly comparing ACEI and angiotensin II receptor an-

Table 4 Choice of drug: compelling and possible indications

Compelling indications	Drugs
Diabetes mellitus (types 1 and 2) with proteinuria	ACEI (ARB)
Elderly with isolated systolic hypertension	Thiazide diuretic – long-acting CCB
Angina	β-Blocker or CCB (heart rate limiting)
Post myocardial infarct or coronary arterial disease	β-Blocker, ACEI (ARB)
Left ventricular hypertrophy	Thiazide diuretic or indapamide and ACEI (ARB), CCB
Congestive heart failure	ACEI (ARB), β-blocker, αβ-blocker, spironolactone, thiazide diuretic and/or loop diuretic for volume overload
Stroke	Thiazide diuretic, ACEI, CCB
Pre-existing stroke	Indapamide or thiazide diuretic, ACEI
Pregnancy	Methyldopa, labetalol, CCB
Prostatism	α-Blocker (Not used as monotherapy for hypertension
Chronic renal disease	ACEI (ARB) combined with thiazide diuretic, loop diuretic and/or metolazone instead of thiazide diuretic (serum creatinine $>$ 116 μg/l, GFR $<$ 30 ml/min), NDHP CCB

ACEI, Angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; CCB, calcium channel blockers (long acting); GFR, glomerular filtration rate; NDHP CCB, non-dihydropyridine calcium channel blocker. The blood pressure lowering effect of ACEI and ARB is greatly increased by the addition of a thiazide diuretic. ARBs are used only if ACEIs are not tolerated. Do not combine ACEI with ARB and β-blocker together in heart failure [24].

tagonist (ARA), generic ACEI should be prescribed in preference to ARA. The exception is poor tolerability to ACEI such as cough or angioneurotic oedema.

- The use of long-acting drugs providing 24-h efficacy on a once daily basis is highly recommended to improve compliance with therapy and minimize blood pressure variability as a consequence of smoother, more consistent blood pressure control. This may also provide greater protection against the risk of major cardiovascular events and the development of target organ damage.
- The use of loop diuretic (furosemide) and diazepam is frequent in SSA. Diazepam is ineffective and the long-term use of loop diuretics is possibly dangerous. Furosemide should only be used for selected indications (including congestive heart failure, advanced renal disease or refractory hypertension).
- Responses of black hypertensives to β-blockers, ACE inhibitors or the ARA are poor unless these agents are combined with a thiazide diuretic. Black hypertensive patients respond best to thiazide-like diuretics, CCBs or vasodilators [3,10].
- A disadvantage of initiating with two drugs, even at a low dose, is that of potentially exposing the patient to an unnecessary agent, but the advantages are important: (i) effectiveness can be increased due to different mechanisms of action; (ii) tolerance can be increased due to less side-effects; (iii) adherence can be increased due to the use of fixed low-dose combinations within a single tablet.
- The following two-drug combinations have been found to be effective and well tolerated: thiazide diuretic and β-blocker; thiazide diuretic and ACEI or ARA; CCB (dihydropyridine) and β-blocker; CCB and ACEI or ARA; CCB and thiazide diuretic; α-blocker and β-blocker; other combinations (e.g. with central agents, including α_2-adrenoreceptor agonists and imidazoline I_1 receptor modulators, or between ACEI and ARA) can be used if necessary [9].
- Follow-up of therapy (monitoring response to therapy).
- During the stabilization period of treatment, patients need to be seen at regular intervals (e.g once per 1–2 months) until blood pressure levels are satisfactorily controlled. The main task of doctors is to ensure that target systolic and diastolic blood pressure is maintained and other risk factors are controlled. Gradual and careful lowering of blood pressure (versus abrupt lowering) will minimize side-effects and complications, and will improve compliance. After stabilization of blood pressure, follow-up visits at 3–6-month intervals may be adequate. As a rule, antihypertensive therapy should be maintained indefinitely.

Special situations

Hypertension emergencies and urgencies

Hypertensive emergencies are situations that require urgent management of blood pressure to prevent or limit target organ damage. These patients may be treated on an outpatient basis but preferably require hospitalization for supervised and gradual reduction of blood pressure.

In most situations, high blood pressure should be managed very carefully and slowly in the hospital. The aim is generally to achieve a progressive blood pressure lowering to a diastolic blood pressure of 100 mmHg over 48–72 h. Indeed, brain arteries adapt only slowly

to reduced blood pressure (within days or weeks) and too rapid blood pressure decrease can precipitate stroke or other complications. Rapid decrease in blood pressure is unwarranted in the case of stroke and heart attack [18,19]. High blood pressure is common after ischaemic stroke and primary intracerebral haemorrhage, and is associated with poor functional outcome. However, the optimal management of blood pressure in acute stroke remains unknown [18,19].

The following drugs are often used.

(i) Long-acting calcium channel blockers. Never use sublingual or other forms of short-acting CCBs.
(ii) ACE inhibitors, using initial low doses. Check serum electrolytes for possible severe hyponatraemia or hyperkalaemia before drug use.
(iii) β-blockers
(iv) Diuretics. This may also potentiate the effects of the other classes mentioned above.

Hypertensive emergencies are life threatening. Common situations include:

- Hypertensive encephalopathy.
- Cerebrovascular accident (CVA, stroke); CVA and stroke require a more prudent approach for the first 10 days. After stabilization, the blood pressure should be progressively decreased.
- Hypertensive heart failure presenting with acute left ventricular failure with severe pulmonary oedema.
- Malignant hypertension.
- Dissecting aneurysm of the aorta.
- Eclampsia and pre-eclampsia.
- Unstable angina/myocardial infarction.

Refractory hypertension

Hypertension may be termed refractory when a therapeutic plan including lifestyle measures and more than three drugs (which include thiazide diuretics) of different classes in adequate doses has failed to lower blood pressure below 140/90 mmHg in patients with classical essential hypertension, or below 140 mmHg systolic in patients with isolated systolic hypertension. In these situations, referral to a specialist should be considered.

Causes of refractory hypertension include poor adherence to therapeutic plan, excess sodium intake, excess alcohol intake, intake of drugs that increase blood pressure (e.g. NSAIDs), illicit drugs (e.g. cocaine, amphetamines), oral contraceptives, obesity, sleep apnoea syndrome and suspected secondary causes (e.g. renal or endocrine).

Hypertension in children and adolescents

There is evidence of increasing prevalence of hypertension in children and adolescents. The main cause of hypertension in children relates to the epidemic of obesity worldwide [13].

Patient education

Patient education strategy

This is aimed at empowering the patient to actively participate in and take responsibility for good quality hypertensive care.

- Patients must be educated about the entire concept of hypertension, especially with regard to the significance of symptoms in a majority of cases, and the possible complications if they are not appropriately treated.
- Patients should be encouraged to keep records of their own blood pressure readings performed on them in the hospital or from home checks (self or otherwise). They should not panic in the case of markedly elevated readings but should report them to their doctor for action.
- Emphasis should be placed on lifestyle modifications and the need for life-long treatment.
- Drug information should be provided to the patient in reasonable detail.

Prevention of hypertension

There is evidence that prevalence of hypertension and CVD is increasing rapidly in SSA [20]. Two recent surveys indicated that, in Tanzania, just under 20% of hypertensive subjects were aware of their diagnosis, approximately 10% reported receiving treatment and less than 1% were controlled (blood pressure $< 140/90$ mmHg) [21]. The treatment status for South African Black males showed that 20% were aware of their hypertension, 14% were on treatment and only 7% were controlled (blood pressure $< 140/90$ mmHg) and, for females, 47% were aware of their hypertension, 29% were on treatment and only 15% were controlled ($< 140/90$ mmHg) [22].

Primordial prevention

The main objective is to avoid or decrease the social, economic and cultural determinants that contribute to development of hypertension. Primordial prevention relies on health policies that create a congenial environment and promote healthy behaviours and population-wide education programs. They depend, in turn, on many factors, including political commitment, advocacy by health professionals and involvement of community leaders and the mass media [23].

Primary prevention of hypertension

The objective is to reduce or modify the risk factors of hypertension (e.g. salt intake and obesity) through appropriate policies and educational programs and to prevent or delay the development of hypertension. The

resultant changes in behaviour at the population level (e.g. low salt intake or increased physical activity) can produce benefits across the whole spectrum of blood pressure distribution. Because a substantial number of adults have blood pressure above the optimal level, even a small reduction in blood pressure level can bring about a significant decrease in cardiovascular risk.

At the individual level, primary prevention of hypertension consists of adopting healthy lifestyles at an early age [23].

Screening for hypertension

Screening activities are an important component of any prevention and control program. They allow detection of previously unaware hypertensive individuals and provide them with early treatment to prevent CVD. Screening is particularly important in population subgroups at high risk for developing CVD (e.g. older individuals, some ethnic groups), and those with limited access to medical care (e.g. some minorities, migrants). However, screening has to be linked with proper diagnosis (e.g. repeated blood pressure measurements), assessment of cardiovascular risk and therapeutic follow-up.

To be sustainable, a typical screening program needs to be supported by (i) health education programs that suit local conditions and socio-cultural realities; (ii) awareness-raising programs that target patients and the general population through media and other local communication channels; (iii) dissemination of context specific recommendations for management and assessment of high blood pressure and CVD risk factors; and (iv) inter-regional and global CVD information exchange networks.

References

1 SA Hypertension Society Executive Committee. South African Hypertension Clinical guideline 2002. *S Afr Med J* 2002; **92**:171–172.
2 Mabadeje AF. 1999 WHO-ISH Guidelines for management of hypertension: implementation in Africa – the Nigerian experience. Nigerian Guidelines. *Clin Exp Hypertens* 1999; **21**:671–681.
3 Seedat YK. Hypertension in black South Africans. *J Hum Hypertens* 1999; **13**:97–103.
4 The ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker versus diuretic: the Antihypertensive and Lipid-Lowering treatment to prevent Heart Attack Trial (ALLHAT). *JAMA* 2002; **288**: 2981–2997.
5 Wright JT, Bakris G, Greene T, Agodoa LY, Appel LJ, Charleston J, *et al.* Effect of blood pressure lowering and antihypertensive drug class on progression of hypertensive kidney disease: results from the AASK trial. *JAMA* 2002; **288**:2421–2431.
6 WHO/ISH. 2003 World Health Organization/International Society of Hypertension Statement on Management of Hypertension. *J Hypertens* 2003; **21**:1983–1992.
7 WHO. *CVD-Risk Management Package for Low and Medium Resource Settings*. Geneva: WHO; 2002.
8 Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, *et al*. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003; **289**:2560–2571.
9 ESH/ESC. 2003 European Society of Hypertension–European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* 2003; **21**:1011–1053.
10 Douglas JG, Bakris GL, Epstein M, Ferdinand KC, Ferrario C, Flack JM, *et al*. Management of high blood pressure in African Americans: consensus statement of the Hypertension in African Americans Working Group of the International Society on Hypertension in Blacks. *Arch Intern Med* 2003; **163**:525–541.
11 O'Brien E, Asmar R, Beilin L, Imai Y, Mallion JM, Mancia G, *et al*., on behalf of the European Working Group on Blood Pressure Monitoring. European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J Hypertens* 2003; **21**:821–848.
12 O'Brien E, Atkins N. A website for blood pressure monitoring devices. *Blood Press Monit* 2003; **8**:177–179.
13 The problem of overweight and obesity. *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation.* WHO Technical Report Series 894. Geneva: WHO; 2000, p. 537.
14 Oparil S, Wang JG, Bakir S, Staessen JA. New Outcome Trials of Antihypertensive Treatment. In: Schachter M, Kaplan N (editors): *New frontiers in hypertension*. London: Lippincott Williams and Wilkins; 2002, pp. 61–99.
15 Staessen JA, Wang JG and Birkenhager WH. Outcome beyond blood pressure control? *Eur Heart J* 2003; **24**:504–514.
16 Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, *et al*. Primary prevention of hypertension: Clinical and public health advisory from the National High Blood Pressure Education Program Coordinating Committee. *JAMA* 2002; **288**:1882–1888.
17 Neal B, MacMahon S, Chapman N. Effects of ACE inhibitors, calcium antagonists and other blood pressure lowering drugs: results of prospectively designed overviews of randomized overviews of randomized trials. Blood Pressure Treatment Trialists' Colloboration. *Lancet* 2000; **356**:1955–1964.
18 Seedat YK. Malignant accelerated hypertension In: Mancia G, Chalmers J, Julius S, Saruta T, Weber M, Ferrari A, Wilkinson I (editors): *Manual of hypertension*. London: Churchill Livingstone Harcourt Publishers Limited; 2002, pp. 623–634.
19 International Society of Hypertension Writing Group. International Society of Hypertension (ISH): statement on the management of blood pressure in acute stroke. *J Hypertens* 2003; **21**:666–672.
20 Nissinen A, Bothig S, Granroth H, Lopez AD. Hypertension in developing countries. *World Health Stat Q* 1988; **41**:141–154.
21 Edwards R, Unwin N, Mugusi F, Whiting D, Rashid S, Kissima J, Aspray TJ, *et al*. Hypertension prevalence and care in an urban and rural area of Tanzania. *J Hypertens* 2000; **18**:145–152.
22 Steyn K, Gaziano TA, Bradshaw D, Laubscher R, Fourie J. South African Demographic and Health Coordinating Team. Hypertension in South African adults: results from the Demographic and Health Survey, 1998. *J Hypertens* 2001; **19**:1717–1725.
23 Seedat YK. Hypertension in developing nations in sub-Saharan Africa. *J Hum Hypertension* 2000; **14**:739–747.
24 Cohn JN, Tognoni GA, on behalf of the VALHEFT Trial Investigators. Randomised trial of the angiotensin-receptor blocker valsartan in chronic heart failure. *N Engl J Med* 2001; **345**:1667–1675.

Appendix

The following experts participated actively in the development of the present recommendations. None of the authors have any conflict with any pharmaceutical company with respect to the owning of shares, educational or pharmaceutical support in the preparation of this paper. Algeria – M. Issad; Angola – M. Vidigal; Belgium – P. Block, H. Boudrez, Y. Carpentie, J.P. Degaute, S. Degré, M. Kornitzer, M. Leeman; M. Meysman, A. Nemery P. Van de Borne; Benin – H. Agboton, L. Wetchoko; Cameroon – A.R. Dongmo, D. Lemogoum, J. Meli, J. Ndjebet, P. Ndobo, K. Ngu Blackett; Canada – A. Chockalingam, P. Hamet; Cape Verde – J. Dos reis; DR-Congo – Longo Mbenza, J.R. M'Buyamba-Kabangu, D. Ngoy; France – R. Asmar, A. Fournier, F. Gueyffier, J.J. Pik, P. Sosner, J.V.

Tchienehom; Gabon – A.M. Antchouey, P.A. Kombila, D. Mavoungou; Gambia – O. Nyan, M. Van der Sande; Ghana – A. Amoah; Guinea – G. Sultan; Kenya – E.N. Ogola; Mali – B. Diarra, K. Toure, M. Traoré; Mauritius – E. Bissessur; Mozambique – A. Damasceno; Nigeria – O.O. Akinkugbe, A.F.B. Mabadeje, B. Onwubere; Rwanda – E. Gasakure, N. Karekesi; Senegal – M. Thiam; Seychelles – P. Bovet; South Africa –Y.K. Seedat, K. Steyn; Sudan – I. Hamed; Switzerland – J.P. Gervasoni; Uganda – E. Rutangirwa; United Kingdom – J. Atkinson, A. Heines; Zambia – E. Nyemdwa; Zimbabwe – J. Chifamba. Representatives of the following organizations were present: African Union – V. Djomatchoua-Toko; Centers for Diseases Control, USA – K. Greenlund, G. Mensah; European Union – S. Nair-Debouell; Global Forum for Health Research in Developing countries – T. Nchindat; Initiative for Cardiovascular Health–Global Forum for Health Research in developing countries, India – S. Reddy; Panafrican Society of Cardiology – W. Muna; World Heart Federtion – L. Ryden; World Hypertension League, USA – C. Lenfant, P. Mulrow; WHO/Africa Region – M. Belhocine; WHO/Geneva – S. Mendis.