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- South African Hypertension Guideline 2006

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CLINICAL GUIDELINE

South African Hypertension Guideline 2006

Joint National Hypertension Guideline Working Group 2006: Y K Seedat[1], M A Croasdale[2], F J Milne[1], L H Opie[1], V J Pinkney-Atkinson[1], B L Rayner[1], Y Veriava[1]

Outcomes. Extensive data from many randomised controlled trials have shown the benefit of treating hypertension. The target blood pressure (BP) for antihypertensive management should be systolic BP < 140 mmHg, diastolic BP < 90 mmHg, with minimal or no drug side-effects. However, a lesser reduction will elicit benefit although this is not optimal. The reduction of BP in the elderly should generally be achieved gradually over 6 months. Stricter BP control is required for patients with end-organ damage, co-existing risk factors and co-morbidity, e.g. diabetes mellitus. Co-existent risk factors should also be controlled.

Benefits. Reduction in risk of stroke, cardiac failure, renal insufficiency and coronary artery disease. The major precautions and contraindications to each antihypertensive drug recommended are listed.

Recommendations. Correct BP measurement procedure is described. Evaluation of cardiovascular risk factors and recommendations for antihypertensive therapy are stipulated. The total cardiovascular disease risk profile should be determined for all patients and this should inform management strategies. Lifestyle modification and patient education are cornerstones in the management of every patient. Drug therapy for the patient with uncomplicated hypertension should be as follows: first line – low-dose thiazide or thiazide-like diuretics; second line – add either an angiotensin-converting enzyme inhibitor (ACE-I) or a calcium channel blocker (CCB); third line – add another second-line drug not already used. In resistant hypertension where a fourth drug is needed, use either a centrally acting drug, vasodilator, alpha-blocker, or beta-blocker. The order of drug choice may change in those with compelling indications for a particular drug class. The guideline includes management of specific situations including hypertensive emergency and urgency, severe hypertension with target-organ damage and hypertension in diabetes mellitus, etc.

Validity. The guideline was developed by a joint Southern African Hypertension Society and National Department of Health Directorate: Chronic Diseases, Disabilities and Geriatrics working group. Input was also obtained from representatives of the various related professional societies.

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1. Introduction

This is the third hypertension guideline published by the Southern African Hypertension Society (SAHS)[1-3] and the second by the National Department of Health (DOH).[4] It is the first joint DOH and SAHS hypertension guideline, to ensure that treatment in the two sectors is seamless and does not conflict on matters of fact or principle. It represents an important step towards the implementation of a national standard of care that will translate into quality care for persons living with hypertension. It reflects realistic objectives that can be applied widely and aims to diminish the impact of hypertension and related cardiovascular disease (CVD) risk in this country. The methodology and participants for the 2006 revision are given in Annexure A.

[1]**Southern African Hypertension Society Guideline Committee**
[2]**Directorate: Chronic Diseases, Disabilities and Geriatrics, National Department of Health**

Correspondence to: **Dr V J Pinkney-Atkinson, PO Box 122, River Club, South Africa, 2149. Fax: 011-706-4915, tel. 011-706-4196, e-mail: sahs@hypertension.org.za, website: www.hypertension.org.za**

The last two versions of the SAHS guideline have emphasised improved diagnosis and treatment, tighter control and risk factor stratification.[2,3] Other guidelines support the same trends[5-9] as well as the movement to evidence-based guidelines.[10,11] The SAHS has a policy of ongoing review of its guideline given the changing nature of the evidence. This is highlighted by the decision of the British Hypertension Society[12] to revise its guideline[11] in the light of the results of the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT).[13]

The importance of the alliance between the DOH and the SAHS is highlighted by past differing definitions of hypertension as in the 1998 *South Africa Demographic and Health Survey*[14] where hypertension was defined as either a blood pressure (BP) of ≥ 160/95 mmHg or the taking of antihypertensive medication in persons aged 15 years or older. If the internationally accepted definition of hypertension is used as in this guideline (BP ≥ 140/90 mmHg), a further 3 million people will be added to the hypertensive population.[15,16] CVD has been shown to be the second highest cause of mortality in South Africa, with 4 of the top 20 causes of death accounting for 7.4% of all deaths.[17] All of this has major cost implications for

a developing country and requires a national strategy for all levels of prevention and management.

Hypertension is a global health burden affecting developed and developing countries, and South Africa is no exception.[18] The high prevalence of hypertension worldwide has contributed to the present and expected pandemic of CVD and this is of particular concern in developing countries.[19] The control of hypertension in conjunction with other major risk factors such as cigarette smoking, dyslipidaemia and diabetes mellitus constitutes the ideal approach to the primary prevention of atherosclerotic disease and remains a major challenge for the community at large. The trend towards comprehensive cardiovascular risk factor management is now the internationally accepted model of care.[20]

Hypertension is a costly and major contributor to CVD. In 1991, CVD accounted for R4 - 5 billion in direct and indirect costs.[21] This expenditure constituted 7.5% of the direct health care spending in South Africa.[22] This guideline had adopted a more evidence-based approach to the estimation of CVD risk which will allow for treatment of those South Africans at highest risk and those who can gain maximally from lifestyle and drug interventions at the lowest cost, given South Africa's limited national resources.[23]

2. Objective

The objective of this guideline is to promote evidence-based, accessible, and comprehensive management of hypertension by health care professionals in the public and private sectors. Further, it should act as a resource document for patients with hypertension to inform them of the national approach to hypertension care in South Africa.

3. Abbreviations

ABPM = ambulatory BP monitoring; ABP = ambulatory BP; ACE-I = angiotensin-converting enzyme inhibitor; ARB = angiotensin II receptor blocker; BP = blood pressure; CCB = calcium channel blocker; DBP = diastolic blood pressure; SBP = systolic blood pressure.

4. BP measurement

BP measurement is a vital clinical skill but it is poorly performed by all health care professional categories. The European Hypertension Society[24] and the American Heart Association[25] published detailed recommendations on the measurement of BP which the Joint Hypertension Working Group fully endorses.

4.1. Generic measurement principles

These recommendations are generic and apply equally to all validated devices, especially clinic and self-measurement of BP, e.g. arm position, posture of the patient, cuff size and the number of readings that should be taken.

BP is recorded using an approved device with the patient in a sitting position (with the back supported, arm bared and resting on a surface at heart level) for at least 5 minutes before measurement. Patients should not have smoked, ingested caffeine-containing beverages or had food in the previous 30 minutes. In persons aged over 60 years, those with diabetes mellitus and others at risk (see Table III), the BP should also be recorded after standing for 1 minute to document postural hypotension.

An appropriate size cuff should be used – a standard cuff (12 cm) for a normal arm, and a larger cuff (15 cm) for an arm with a mid-upper circumference > 33 cm (the bladder within the cuff should encircle 80% of the arm). If an undersized cuff is used, the BP can be overestimated (undercuffing), and if the cuff and bladder are too large the BP can be underestimated (overcuffing).

Both systolic BP (SBP) and diastolic BP (DBP) should be recorded. At the initial consultation BP should be measured in both arms, and if there is any discrepancy it should be measured thereafter in the arm with the higher BP. The SBP should be first estimated by palpation to avoid missing the auscultatory gap. SBP is measured at the first appearance of sound (phase I) and DBP is measured at the disappearance of the sounds (phase V). Phase V is also recommended in pregnancy. In cardiac arrhythmias (e.g. atrial fibrillation), the highest phase I and lowest phase V are recorded as the SBP and DBP, respectively. There are circumstances when both phase IV (muffling) and phase V should be recorded, e.g. aortic regurgitation, pregnancy, and severe anaemia.

The BP recorded should be the average of 2 readings taken 1 minute apart. If the first 2 readings differ by > 5 mmHg, additional readings should be taken. Repeat measurements should be performed on 3 separate occasions when either the initial SBP is between 140 and 160 mmHg or the DBP is between 90 and 100 mmHg. This should occur within 2 months to determine if the patient should be diagnosed as hypertensive. All measurements should preferably be taken at the same time of the day and using the same arm.

The elderly may present special problems with BP measurement because there may be considerable BP variability, with periods of hypotension as well as hypertension. This occurs particularly in hot weather. The most common form of hypertension in the elderly is isolated systolic hypertension, due to the stiffening of the large arteries that occurs with ageing.

It may be advisable to check the standing BP in hot weather, particularly in diabetics, the elderly, those who have symptoms of postural hypotension such as dizziness and those who appear dehydrated.[26]

The BP measurement device and its attachments (tubing, cuff, valve) need to be serviced and calibrated at least every 2 years.

4.2 Mercury sphygmomanometer

There are increasing criticisms of the use of mercury sphygmomanometers. Mercury is inert and does not degrade. Although it is not toxic to patients or operators when the device is intact, mercury becomes a major environmental hazard when it is discarded.[27] There are international moves to replace mercury sphygmomanometers with battery-operated digital devices. However, in South Africa and other developing countries, there is concern about the availability of accurate devices and the safe disposal of lead-containing batteries, but if a mercury sphygmomanometer needs replacement, a validated oscillometric device should be considered. An inexpensive device for developing countries is becoming available.[28]

4.3 Self-measurement of BP

Self-measurement of BP is recommended in selected circumstances and for selected target groups:[29] (*i*) suspected white-coat hypertension; (*ii*) to guide antihypertensive medication; (*iii*) the elderly; (*iv*) pregnant women; (*v*) those with diabetes mellitus; (*vi*) those with refractory hypertension; (*vii*) to improve adherence to treatment; (*viii*) to predict outcomes; and (*ix*) to empower the patient.

The recommendation for the purchase of a device should come from the patient's medical practitioner. The practitioner is responsible for educating the patient on user procedure and the types of validated device that are available. Many of the devices currently on the market have not been validated according to stringent international standards. An up-to-date list of validated devices can be found on the independent websites **www.dableducational.com**[30] or **http://afssaps.sante.fr**.[28]

A list of devices can be found on the SAHS website **www.hypertension.org.za** or by contacting the SAHS (**sahs@hypertension.org.za**).

Only upper-arm devices are recommended, but even these are unsuitable in patients with sustained arrhythmias. They should not be used for BP measurement during exercise and they are not as specific as ambulatory BP monitoring (ABPM) for the diagnosis of white-coat hypertension.

Ideally, the patient should take 2 early-morning and 2 late-afternoon readings over 5 days and calculate the average of all the readings to establish a reliable reading. Patients must discuss any proposed change in drug medication with their health care professional.

4.4 Twenty-four-hour ABPM

ABPM is not part of routine BP evaluation but is increasingly used worldwide.[15] The indications for ABPM are very similar to those for self-BP monitoring but are now given in clinical terms: (*i*) when there is a large difference between office BP and BP measured elsewhere (pharmacy, self-BP measurement) – this is suspected white-coat hypertension and is commonly found in the elderly, in diabetics and in pregnancy; (*ii*) refractory hypertension (see section 9) with little clinical target-organ damage; (*iii*) when there is clinical target-organ damage, but the office BP is normal or borderline, suggesting nocturnal hypertension or masked hypertension (not uncommon in diabetics); and (*iv*) suspected postural hypotension (due to medication or postprandial).

The ABPM measuring device must be validated rigorously according to acceptable international standards before purchase and must, together with its attachments (tubing, cuff, etc.), be serviced on a regular basis. In selected situations where ABPM is not available, self-BP monitoring may be used as a substitute.[5]

An up-to-date list of validated devices can be found on independent websites **www.dableducational.com**[30] and **http://afssaps.sante.fr**.[28] The list can be found on the SAHS website **www.hypertension.org.za** or by contacting the SAHS at **sahs@hypertension.org.za**.

4.5 Comparison of different methods of BP measurement

Table I shows the comparative value of the three different methods of BP measurement.

Table I. Different methods of BP measurement[25]

	Clinic	Home	Ambulatory
Predicts outcome	Yes	Yes	Strongly
Initial diagnosis	Yes	Yes	Yes
Cut-off BP levels (mmHg)	140/90*	135/85	120/70 (mean night) 135/85 (mean day) 130/80 (24-hour)*
Evaluation of treatment	Yes	Yes	Limited but valuable
Assess diurnal rhythm	No	No	Yes

*The small difference between the clinic BP and the ambulatory BP (ABP) shown in this table does not clearly reflect that there is an increasing difference between the two measurements as the clinic BP rises. As the clinic BP rises, the ABP rises much less.[31] Thus a clinic BP of 180/110 mmHg corresponds to an ABP of 150/95 mmHg. This may result in a large difference of 30/15 mmHg between the higher clinic BP levels and the ABP.

5. Cardiovascular disease risk stratification

5.1 Rationale for cardiovascular risk assessment

The DOH and the SAHS remain committed to the format of the CVD risk assessment as outlined in Tables II and III until there is wide national consensus on a different CVD risk model by all stakeholders (professionals, providers, government and health care funders) and supported by adequate local data. This model, developed by the European Hypertension Society and the European Society of Cardiology,[7] reflects absolute risk and the continuous risk associated with BP as used in many other guidelines.[6,9] It is possible to use the tool as a pragmatic risk assessment model which is adaptable for use in multiple settings including those low-resource settings where some of the measures are unavailable and beyond the resources of budgets.[32]

Consensus has been reached on the necessity of immediate drug treatment for those with known associated clinical conditions and/or target-organ damage and/or a SBP ≥ 180 mmHg or DBP ≥ 110 mmHg (Fig. 1).[23] In the absence of associated clinical conditions and/or target-organ damage or very high levels of BP, the exact level of BP at which to initiate drug treatment has changed over time and remains the subject of debate.

5.2 Risk factors, target-organ damage and associated clinical conditions

Table II lists the major risk factors, target-organ damage and associated clinical conditions.[7] Risk factors that are modifiable (e.g. smoking and dyslipidaemia) should be the target of lifestyle intervention and other treatment as appropriate. In addition to controlling hypertension, target-organ damage and associated clinical conditions must be managed appropriately and referred if necessary to a higher level of care.

There is still lack of consensus on the importance of some of the newer risk factors such as obesity and the metabolic syndrome in treatment decisions.[6] For example, obesity may be measured using the body mass index (BMI), abdominal circumference or waist-to-hip ratio.[33] The metabolic syndrome has not been listed as a risk factor as in the JNC 7 report[6] despite increasing evidence of its link to cardiovascular risk and the future risk of diabetes. The metabolic syndrome represents a combination of underlying and major risk factors[34] but there is debate on which criteria to adopt[35-37] and on whether the clustering of risk factors represents a greater cardiovascular risk over and above the individual components as used in the Framingham risk calculation. However, it must be remembered that where the metabolic syndrome is present, a SBP ≥ 130 mmHg and/or DBP ≥ 85 mmHg requires treatment (weight loss and exercise followed by drugs after an appropriate trial of lifestyle modification).

Table II. Major risk factors, target-organ damage and associated clinical conditions*

Major risk factors	Target-organ damage	Associated clinical conditions
Levels of systolic and diastolic BP	Left ventricular hypertrophy: based on ECG See Table IV	Coronary heart disease
Smoking	Microalbuminuria: albumin/creatinine ratio 3 - 30 mg/mmol	Heart failure
Dyslipidaemia Total cholesterol > 6.5 mmol/l, OR LDL > 4 mmol/l, OR HDL men < 1 and women < 1.2 mmol/l	Slightly elevated creatinine Men 115 - 133 µmol/l Women 107 - 124 µmol/l	Chronic kidney disease: albumin creatinine ratio > 30 mg/mmol
Diabetes mellitus Men > 55 years Women > 65 years		Stroke or transient ischaemic attack
Family history of early onset of cardiovascular disease Men aged < 55 years Women aged < 65 years		Peripheral arterial disease
Waist circumference – abdominal obesity Men ≥ 102 cm Women ≥ 88 cm The exceptions are South Asians and Chinese: men > 90 cm and women > 80 cm.[39]		Advanced retinopathy Haemorrhages OR Exudates Papilloedema

*Based on the European Society of Hypertension/European Society of Cardiology guidelines.[7]
LDL = low-density lipoprotein; HDL = high-density lipoprotein.

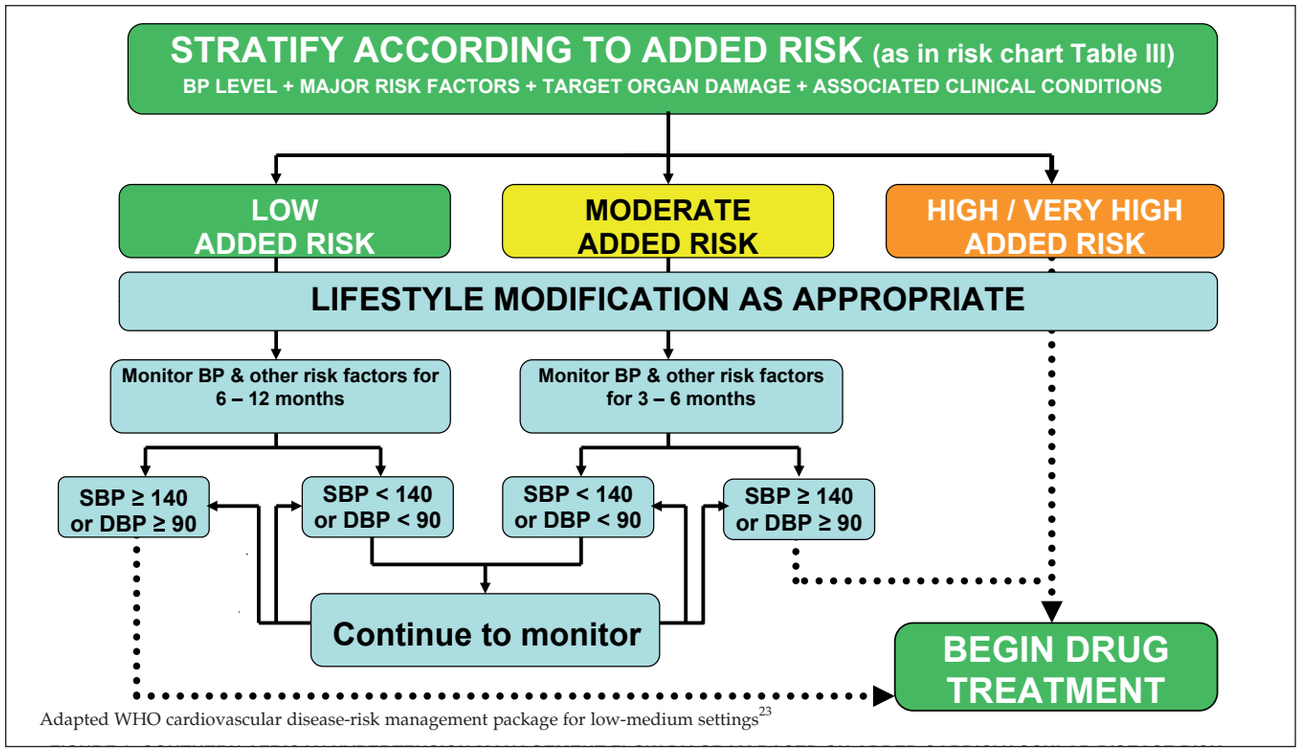

Fig. 1. Southern African hypertension management flow diagram based on added cardiovascular disease risk.

Table III. Stratification of risk to quantify prognosis*

Other risk factors and disease history	BP (mmHg)				
	Normal SBP 120 - 129 or DBP 80 - 84	**High-normal** SBP 130 - 139 or DBP 85 - 89	**Stage 1 Mild hypertension** SBP 140 - 159 or DBP 90 - 99	**Stage 2 Moderate hypertension** SBP 160 - 179 or DBP 100 - 109	**Stage 3 Severe hypertension** SBP > 180 or DBP > 110
No other major risk factors	Average risk	Average risk	Low added risk	Moderate added risk	High added risk
1 - 2 major risk factors	Low added risk	Low added risk	Moderate added risk	Moderate added risk	Very high added risk
≥ 3 major risk factors or target-organ damage or diabetes mellitus	Moderate added risk	High added risk	High added risk	High added risk	Very high added risk
Associated clinical conditions	High added risk	Very high added risk	Very high added risk	Very high added risk	Very high added risk

*Based on the European Society of Hypertension/European Society of Cardiology guidelines.[7]

Table IV. Routine investigations

Investigation	Clinic frequency	Comments
Body weight/overweight		
Body weight	Every visit	
Height	First visit	< 25 kg/m² for men and women. Use supplied body mass
Body mass index	Every visit	index chart (Annexure B) and define level of obesity[38]
Abdominal obesity		
Waist circumference	Every visit	Use correct method of measurement.
OR		Goal: Men < 102 cm, women < 88 cm. South Asians and
Waist-to-hip ratio		Chinese: men < 90 cm, women < 80 cm[39]
		The waist-to-hip ratio has greater predictive value than body mass index or waist circumference for mycardial infarction but may not be practical in many settings[32]
Urine dipstick		
Protein	First visit	Abnormal dipstick
Blood	Yearly if normal	Any one of the following:
Sugar	Repeat at next visit if	Proteinuria ≥ 2+
	abnormal on first visit	Haematuria ≥ 1+
		Refer for immediate further investigation
Microalbuminuria	First visit then yearly	Performed on diagnosis of type 2 diabetes mellitus or 5 years
Diabetes mellitus only		after the diagnosis of type 1[40]
Blood tests		
Creatinine	Yearly if normal	From serum creatinine calculate glomerular filtration rate
Potassium	Yearly if normal	
Glucose	Yearly if normal	Consider glucose tolerance test in patients with abnormal
(fasting preferred)		fasting glucose 5.6 – 7.0 mmol/l
Random total cholesterol	Yearly if normal	Measure fasting lipogram if cholesterol > 5.1 mmol/l or in high-risk groups
ECG (resting)	Yearly if normal	Refer to Southern African Hypertension Society policy brief on left ventricular hypertrophy[41]
Additional investigations	As necessary	If secondary causes suspected at first visit or if refractory
	Referral	hypertension exists, additional investigations should be performed

5.3 Routine baseline investigations

Table IV lists recommended routine basic investigations. Apart from measurements of overweight and obesity, the tests are performed annually unless abnormal. Abnormal or suspicious results must be repeated as clinically indicated.

6. Target BP levels

The target BP levels vary according to number of major risk factors, target-organ damage and/or associated clinical conditions as shown in Table V. These goals should be added to the recommended goals for waist/abdominal circumference, blood sugar and lipid levels in patients with the metabolic syndrome (see section 8.4).

Table V. Targets for BP-lowering treatment

Stage	BP level (mmHg)
All stages	< 140/90* *In isolated systolic hypertension do not lower the DBP to < 65
High-risk patients, for example those with: Diabetes mellitus Renal disease (macroalbuminuria and/or elevated creatinine) Congestive heart failure	< 130/80 **Ideally these targets should be reached in 3 months**

7. Sustainable hypertension management

The Hypertension Working Group has given considerable time, thought and debate to the very real concerns about the economic sustainability of lifelong drug therapy. The issues of affordability are considered important factors in both the private and public sectors in South Africa. This becomes even more relevant given the proposals for the holistic management of hypertension, its complications and disease associations (see section 8.4).

The DOH and SAHS reiterate in the strongest possible terms the importance of lifestyle modification at all stages of hypertension. If there are limited resources then drug

treatment may be delayed in persons with low or moderate added cardiovascular risk[23] (Table III and Fig. 1). Recent studies emphasise the cost effectiveness of both lifestyle and drug management in reducing CVD risk in both developed and lesser-developed regions of the world[42] and the importance of tight BP control in black Americans.[43]

In the current environment the price of antihypertensive and other drugs fluctuates considerably irrespective of market sector. Where possible, generic equivalents or generic drug combinations are encouraged and the cheapest generic in a class should be considered provided they are true equivalents. The patient should not be changed from one generic to another in the same class at frequent intervals solely on the basis of lower price. The DOH and the SAHS believe that poorly managed hypertension, with the undesirable consequences of heart failure, stroke and chronic renal failure, is unacceptable. Health professionals, together with patients, must manage hypertension, its associated risk factors and target organ damage appropriately. Best practice recommendations should be clearly stated and compromises based on limited resources must be made deliberately and transparently.

8. Management

The diagnosis of hypertension may be made if repeat BP measurements performed on 3 separate occasions when either (*i*) the initial SBP is ≥ 140 mmHg or (*ii*) the DBP is ≥ 90 mmHg taken over a period of 2 months. If the SBP is ≥ 180 mmHg or the DBP is ≥ 110 mmHg then refer to section 9 on severe (stage III) hypertension.

Lifestyle information should be given to every person whose BP is measured. Where the BP is elevated, a programme of lifestyle modification should be implemented immediately in every case (Fig. 1 and section 8.1).

These recommendations are further clarified by using Table III and Fig. 1 in conjunction.

8.1 Lifestyle modification[44,45]

A healthy lifestyle remains the cornerstone of the management of hypertension for all levels of BP. A healthy lifestyle decreases BP, enhances antihypertensive drug efficacy and decreases total cardiovascular risk. The DOH's strategy for a healthy lifestyle is supported and includes the following elements that will improve BP (Table VI).

1. Achieve and maintain ideal weight with a BMI between 18.5 and 24.9 kg/m². For weight loss refer to the two local guidelines for the prevention and management of obesity.[38,47]

2. Limit total sodium intake to less than 2 400 mg per day by consuming less than 1 teaspoon of salt per day. Besides table salt, high sodium levels are found in packet soups, stock cubes, gravies, processed cheese, many breakfast cereals, bread, salty snacks and tinned food. Reduce the intake of those foods, remove the salt cellar from the table, and gradually reduce added salt in food preparation. Inform patients that food may taste bland initially, and encourage the use of lemon juice, herbs and spices as alternative seasoning. Taste adaptation to reduced sodium intake occurs with time. Food labels list salt content as sodium. Sodium-free means less than 5 mg per 100 g serving, very low sodium means up to 40 mg per 100 g serving, and low sodium means up to 120 mg per 100 g serving. To calculate the salt content, multiply sodium content by 2.5.

3. Limit alcohol intake to 2 standard drinks per day for men and 1 standard drink per day for women and small men. A standard drink contains about 10 g of ethanol (e.g. 25 ml spirits, 125 ml wine, 340 ml beer, 60 ml sherry, 25 ml liqueur).

4. Follow the nutrition guidelines reported in the global strategy on diet, physical activity and health published by the World Health Organization (WHO).[44,45] These guidelines are incorporated in the various local guidelines.[47-49] These guidelines emphasise a diet low in total fat with high intake of fruit and vegetables (5 portions per day), regular use of low-fat dairy products, a high intake of high-fibre wholegrain foods, fish rather than red meat, the use of products low in saturated fat, low salt, and sparing use of sugar and sugar-containing foods. Intake of beverages with high caffeine levels should be avoided, but the modest use (1 - 2 cups per day) of coffee will not increase BP.

5. Regular moderate-intensity exercise for at least 30 minutes on most or preferably all days of the week. Moderate levels of exercise can be achieved by brisk walking and should be 40 - 60% of peak oxygen consumption (VO_2peak). Exercise bouts can either be continuous or accumulated in shorter time periods throughout the day. The benefit of exercise is dose-response related, and the early adaptations from a sedentary lifestyle to becoming moderately active have the greatest effect. Patients with uncontrolled hypertension should embark on exercise training only after evaluation and initiation of therapy.

6. Stop the use of all tobacco products. The use of snuff is common among South African women who traditionally do not smoke tobacco. Nicotine replacement therapy should be used for patients with hypertension while under medical supervision.

8.2 Drug therapy

In order to use these recommendations for treatment it is essential that the patient's added cardiovascular risk be assessed according to Table III. The level of added cardiovascular risk informs the decision to implement drug therapy according to the decision flow chart (Fig. 1). Drug treatment should be commenced in the following cases: (*i*) low added risk despite a period of 6 - 12 months of lifestyle modification and observation; (*ii*) moderate added risk despite a period of 3 - 6 months of lifestyle modification and observation; and (*iii*) high or very high added risk.

Table VI. Lifestyle modification for hypertension care[6]

Modification	Recommendation	Approximate SBP reduction
Weight reduction	Maintain normal body weight (body mass index* 18.5 - 24.9 kg/m²) by means of limited calorie intake and adequate daily physical activity See Annexure B for body mass index chart	5 - 20 mmHg/10 kg weight loss
Dietary sodium reduction	Reduce dietary sodium intake to ≤ 100 mmol/l (2.4 g sodium or 6 g sodium chloride (salt) per day)	2 - 8 mmHg
Restrict alcohol consumption	Limit consumption to ≤ 2 standard drinks per day in men and 1 standard drink a day in women	2 - 4 mmHg
Limit total fat intake[44]	Limit total fat intake, reduce saturated and trans-fatty acids Recommended maximum fat intake for moderately active adults: Female – normal weight = 70 g/day Female – overweight = 50 g/day Male – normal weight = 95 g/day Male – overweight = 70 g/day OR (preferably) total fat intake should be < 30% of total energy	
Increase fruit and vegetable consumption	Increase fruit, vegetables, legumes, whole grains and nuts to 5 or more helpings a day	Will reduce stroke by up to 26%[46]
Limit free sugars	Reduce free sugars to less than 40 g/day (8 level teaspoons)	
Physical activity[44]	Engage in regular aerobic physical activity such as brisk walking at least 30 minutes per day, most days of the week, minimum of 150 minutes/week	4 - 9 mmHg
Stop using all tobacco products		

There are three important classes of antihypertensive agents for the management of persons without compelling indications: diuretics (thiazide-like and thiazide), angiotensin-converting enzyme inhibitors (ACE-Is) and calcium channel blockers (CCBs). Recent studies[12,13] have led to a reconsideration of the drugs of choice for the management of uncomplicated hypertension. The combination of a thiazide diuretic with a beta-blocker should be discouraged especially where there is abdominal obesity combined with hypertension as both classes of drugs have adverse metabolic consequences and increase the risk of new diabetes.[50,51]

- Step one: low-dose hydrochlorothiazide (12.5 mg preferred up to maximum 25 mg) or thiazide-like diuretic. Higher diuretic doses are not recommended because of the risk of new diabetes.[51]
- Step two and step three: allow for considerations based on the cost of the various drug classes and other patient-related factors such as the presence of major risk factors, associated clinical conditions and target organ damage (Table II). Where there are no other compelling indications, then one of the following is recommended as second- or third-line therapy: (*i*) ACE-I (in ACE-I intolerance use angiotensin II receptor blockers (ARBs)); and (*ii*) CCB long-acting dihydropyridines or non-dihydropyridines.

Table VII lists the clinical considerations for the major drug groups when selecting antihypertensive drug therapy and gives possible contraindications. The issues relating to drug adherence are critical and must be implemented for each patient (see section 14). It is crucial to ensure that the patient understands the importance of adherence to the treatment regimen and brings back drug containers and unused drugs. This should be reinforced frequently. Patient empowerment and single daily dose regimens improve compliance. Where appropriate use fixed-dose combinations. Continued monitoring and management of drug side-effects is essential.

8.2.1 Consensus statement on beta-blockers

Certain beta-blockers (such as atenolol) are no longer considered as routine step one through step three treatment in view of several major considerations, including the failure of atenolol-based therapy in the ASCOT study[13] (see under Combination Therapy, section 8.3). Added to this are the meta-analyses by Carlberg *et al*.[52] and Lindholm *et al*.[53] showing that beta-blockade is relatively ineffective in reducing hard endpoints, not altering several outcomes and reducing stroke only by about half of what was expected by the BP reduction. The proposed mechanisms are that: (*i*) beta-blockers reduce the central aortic systolic pressure less than CCBs and ACE-Is, so that the pressure conveyed to the cerebral circulation and brain

is higher than would be expected from the brachial pressure;[54] and (*ii*) beta-blockers may precipitate new diabetes,[55] especially when used with diuretics.[50,51]

Also note that contrary to expectations, beta-blockers do not reduce cardiovascular mortality or myocardial infarct mortality when given for hypertension.[56] Regarding the use of those beta-blockers not considered in the meta-analysis of Lindholm *et al.*,[53] such as carvedilol, bisoprolol and nebivolol, there are no cardiovascular outcomes in hypertension.

8.2.2 Consensus statement on the use of ACE-I and ARB

Compared with ACE-Is, ARBs provide more effective blockade of the renin-angiotensin system. The DETAIL study in patients with diabetes and microalbuminuria[57] and the VALIANT study[58] in patients post myocardial infarction with heart failure or impaired left ventricular dysfunction or both are the only major published studies comparing ACE-I with ARB on a head-to-head basis. In both studies there was no difference in outcome. The ONTARGET study[59] is a major

Table VII. Indications and contraindications for the major classes of antihypertensive drugs (adapted from the JNC 7 guidelines[6])

		Contraindications	
Class	Conditions favouring use	Compelling	Possible
Diuretics (thiazide; thiazide-like)	Heart failure Elderly hypertensives Isolated systolic hypertension Black hypertensives	Gout	Pregnancy Beta-blockers (especially atenolol)
Diuretics (loop)	Renal insufficiency Heart failure	Not used in other hypertensives	Pregnancy
Diuretics (aldosterone antagonist)	Heart failure Post-myocardial infarction Resistant hypertension	Renal failure Hyperkalaemia	
CCB Long-acting only (dihydropyridine)	Elderly patients Isolated systolic hypertension Angina pectoris Peripheral vascular disease Carotid atherosclerosis Pregnancy Black hypertensives		Tachyarrhythmias Heart failure Antiretroviral therapy
CCB non-dihydropyridine (verapamil, diltiazem)	Angina pectoris Carotid atherosclerosis Supraventricular tachycardia	Atrioventricular block (grade 2 or 3) Heart failure	Constipation (verapamil) Antiretroviral therapy
ACE-Is*	Heart failure Left ventricular dysfunction Post-myocardial infarction Non-diabetic nephropathy Type 1 diabetic nephropathy Type 2 diabetes mellitus Proteinuria	Pregnancy Hyperkalaemia Bilateral renal artery stenosis	
ARBs*	Type 2 diabetic nephropathy Type 2 diabetic microalbuminuria Proteinuria Left ventricular hypertrophy ACE-I cough or intolerance	Pregnancy Hyperkalaemia Bilateral renal artery stenosis	
Beta-blockers	Angina pectoris Post-myocardial infarction Heart failure (only some beta-blockers; must up-titrate) Tachyarrhythmias	Asthma Chronic obstructive pulmonary disease Atrioventricular block (grade 2 or 3)	Peripheral vascular disease Bradycardia Glucose intolerance Metabolic syndrome Athletes and physically active patients Non-dihydropyridine CCBs (verapamil, diltiazem) Pregnancy

*There is considerable overlap in outcomes of ACE-Is and ARBs in the management of type 2 diabetes mellitus. Use the most efficactious class of drug according to patient circumstances.
General note: In resistant (refractory) hypertension centrally acting agents (selective and non-selective) and α-blockers may be required to control BP (see section 9).

study that will further address this issue and is awaited with interest. The ARB produced greater regression of left ventricular hypertrophy compared with a beta-blocker and a 13% reduction in the primary endpoint of cardiovascular morbidity and death, and a 25% reduction in stroke.[60,61] In type 2 diabetes the ACE-Is have prevented the progression from normoalbuminuria to microalbuminuria,[62] and reduced established microalbuminuria.[57] ARBs[63,64] have also reduced microalbuminuria, and delayed the progression of established diabetic nephropathy.[65,66] ACE-I in combination with indapamide,[67] or an ARB[68] were effective in the secondary prevention of stroke. The price of ARBs remains a negative factor until it falls or there is a generic equivalent. However, the overriding importance of drug adherence in BP control must be emphasised.

8.2.3 Compelling indications for a specific drug class

Table VIII outlines the compelling indications (high-risk conditions) that require certain classes of antihypertensive drugs based on randomised controlled trials. The compelling indications for specific classes of drugs apply equally to patients from any ethnic group.[43]

8.3 Combination therapy

The pathophysiology of hypertension is multifactorial. Data on monotherapy show that the reductions in BP produced by monotherapy, i.e. use of a single drug, are often too small to achieve recommended or optimal BP levels. The use of combination therapy is recommended for such patients. Regardless of which drug classes are combined with each other, combination will usually result in an increased BP reduction. Combination therapy may be freely selected flexible or fixed combinations. The latter have specific advantages, allowing BP control to be achieved with a reduced number of daily tablets so that better adherence to therapy is more likely. Flexible dosing of the components allows the dose of one drug to be increased while keeping the other constant, thereby facilitating a greater awareness by the physician of the constituents of the combination, thus reducing the risk of administering contraindicated drugs and allowing better identification of which drug is responsible for the side-effects that may appear during treatment.

A rational fixed-dose antihypertensive combination requires that each component should be safe and that the combination should not elicit additive adverse effects. Each component should contribute to the overall effect and the dosages should be optimised relative to the predictability of bioavailability and the absence of unwanted pharmacokinetic interactions. Additionally, the doses chosen should maintain a high peak-to-trough ratio. The dose-response relationship at the chosen combined dose should be close to the maximal effect of the two doses, and should not cause excessive hypotension. The benefits of combination therapy should include an enhanced antihypertensive effect, a better response rate, fewer adverse effects, lessened metabolic effects and improved outcomes. Effective BP-reducing drug combinations are diuretic and beta-blocker, diuretic and ACE-I or ARB, CCB with ACE-I, and beta- with alpha-blockade. Black patients respond best to diuretic combined with other antihypertensive agents as the majority have low plasma renin activities. Synergism is defined as a co-operative action resulting in a total effect that is greater than the sum of the individual effects. Synergism exists with ACE-Is or ARBs combined with either CCBs or diuretics. Of these classes, ACE-Is, ARBs and diuretics have relatively flat dose-

Table VIII. Recommendations on compelling indications for a specific drug class

Any drug that lowers BP unless absolutely contraindicated (Table VII), will confer protection against target-organ damage. However, the following classes of drugs have additional protective properties in the case of the listed associated clinical conditions/target-organ damage.

Compelling indications	Drug class
Angina	Beta-blocker OR CCB (rate lowering preferred)[69]
Prior myocardial infarct	Beta-blocker AND ACE-I (ARB if ACE-I intolerant).[70,71] Verapamil if beta-blockers contraindicated. If heart failure, see below
Heart failure	ACE-I (ARB if ACE-I intolerant) AND certain beta-blockers[72,73] AND aldosterone antagonist[74,75] For combination ARB and ACE-I see ref.[76] Loop diuretics for volume overload
Left ventricular hypertrophy (confirmed by ECG)	ARB (preferred)[60] OR ACE-I
Stroke: secondary prevention	Low-dose thiazide-like diuretic and ACE-I[67] or ARB[68]
Diabetes type 1 or 2 with or without evidence of microalbuminuria or proteinuria	ACE-I OR ARB[57,62-66,77,78] – usually in combination with a diuretic
Chronic kidney disease	ACE-I OR ARB – usually in combination with a diuretic
Isolated systolic hypertension	Low-dose thiazide or thiazide-like diuretic OR long-acting CCB[79,80]

response BP responses, so that combination with a new drug class is more effective in reducing BP than increasing the dose of the class initially chosen.

8.3.1 Specific combinations

Among combination therapies, that of beta-blockade and diuretics has been standard practice. However, data favouring this practice are based on old studies when comparisons were made versus placebo. The only modern study (the ASCOT trial[13]) comparing this 'old' combination, as atenolol plus thiazide, with the 'new' combination of a CCB (amlodipine) plus an ACE-I (perindopril), had to be stopped because of a mortality and other major outcome advantages for the 'new' combination. Regarding other diuretic combinations, that with a CCB or an ARB was studied in the VALUE trial.[81] The CCB-diuretic combination was better in reducing myocardial infarction than the ARB-diuretic comparator but gave more new diabetes. A third combination that is much used is that of an ACE-I with a diuretic, although there appears to be only one outcome study, namely PROGRESS,[67] in which the combination was more effective than the ACE-I alone in reducing repeat stroke.

8.4 Metabolic syndrome

Of increasing prevalence is the metabolic syndrome, the criteria for which include obesity, dyslipidaemia and type 2 diabetes mellitus occurring together with hypertension. There has been considerable debate about the clinical criteria for this syndrome[34-36] and this guideline recognises that of the American Heart Association/National Heart and Lung and Blood Institutes as shown in Table IX.[37] Thus insulin levels or insulin/glucose ratios have no place in the diagnosis. The metabolic syndrome is not a clearly defined entity with a clear aetiology or underlying mechanism. Therefore, it is recommended that its management should reflect the accepted strategies to reduce cardiovascular risk and each of the components should be managed if present. For example, increased abdominal girth and BP require initial therapy by a combination of intensive diet and exercise.

Table X reflects the current South African norms for the management of dyslipidaemia, obesity and diabetes as communicated by the relevant local professional societies.[38,82,83] The Dietary Approaches to Stop Hypertension (DASH) low-sodium diet will not only lower BP, but will also have a favourable effect on weight, lipids and glycaemic control.[84] There is mounting evidence that so-called normal lipid levels may be inappropriately high in hypertensive patients and certainly are so in patients with hypertensive complications, e.g. stroke and myocardial infarction.[85,86] Lipid-lowering therapy is increasingly part of standard drug therapy in both young and old with vascular disease.[13,87] In some cases, the use of a biguanide, e.g. metformin, may be required in addition to exercise to prevent the progression to frank type 2 diabetes mellitus in hypertensive patients with central obesity.

The indications for and cautions against the use of aspirin, hormone replacement therapy and antioxidants are frequently asked questions. Low-dose aspirin should be used for secondary prevention of transient ischaemic attacks,

Table IX. American Heart Association criteria for clinical diagnosis of the metabolic syndrome[37]

Any 3 of 5 constitute the diagnosis of the metabolic syndrome	Categorical cutpoints
Elevated waist circumference*†	Men > 102 cm, women > 88 cm South Asians and Chinese: Men > 90 cm, women > 80 cm[39]
Elevated triglycerides	≥ 1.7 mmol/l OR On drug treatment for elevated triglycerides
Reduced HDL-C	Men < 1.03 mmol/l, women < 1.29 mmol/l OR On drug treatment for reduced HDL-C‡
Elevated BP	≥ 130/≥ 85 mmHg OR On antihypertensive drug treatment in a patient with a history of hypertension
Elevated fasting glucose	≥ 6.1 mmol/l OR On drug treatment for elevated glucose

*To measure waist circumference, locate top of right iliac crest. Place a measuring tape in a horizontal plane around abdomen at level of iliac crest. Before reading tape measure, ensure that tape is snug but does not compress the skin and is parallel to floor. Measurement is made at the end of a normal expiration.
†Some US adults of non-Asian origin (e.g. white, black, Hispanic) with marginally increased waist circumference (e.g. 94 - 102 cm in men and 80 - 88 cm in women) may have strong genetic contribution to insulin resistance and should benefit from changes in lifestyle habits, similar to men with categorical increases in waist circumference. Lower waist circumference cutpoint (e.g. ≥ 90 cm in men and ≥ 80 cm in women) appears to be appropriate for Asian Americans, south Asians and Chinese.[39]
‡Fibrates and nicotinic acid are the most commonly used drugs for elevated triglycerides and reduced HDL-C. Patients taking one of these drugs are presumed to have high triglycerides and low HDL-C.
HDL-C = high-density lipoprotein cholesterol.

Table X. Current South African norms for dyslipidaemia, obesity and diabetes

LIPID AND TRIGLYCERIDE GOALS[82]

	Lipid	**Current recommended South African levels for different levels of risk** (adopted by the Society for Endocrinology, Metabolism and Diabetes of South Africa)
Established coronary artery disease, diabetes	Total cholesterol	< 4.5 mmol/l
	Triglyceride	< 1.7 mmol/l
OR	HDL-C	> 1 mmol/l men > 1.2 mmol/l women
High cardiovascular disease risk	LDL-C*	< 2.5 mmol/l
Intermediate	Total cholesterol	< 5 mmol/l
OR	Triglyceride	< 1.7 mmol/l
Low cardiovascular disease risk	HDL-C	> 1 mmol/l men > 1.2 mmol/l women
	LDL-C*	< 3 mmol/l

*LDL-C is the primary target of treatment.

HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

OBESITY BODY MASS INDEX[38]

Classification	**Body mass index (kg/m²)**	**Risk of chronic, non-communicable diseases**
Underweight	< 18.5†	Low (but risk of other clinical problems may be greater)
Normal weight	18.5 - 24.9	Average
Pre-obese (overweight)	25.0 - 29.9	Increased
Obese class I	30.0 - 34.9	Moderate
Obese class II	35.0 - 39.9	Severe
Obese class III	≥ 40.0	Very severe

†These values are considered to be independent of age and are the same for men and women.

ABDOMINAL OBESITY WAIST CIRCUMFERENCE (cm)

	Ideal	**Substantial risk**
Men	< 94	> 102
Women	< 80	> 88

DIABETES MELLITUS TYPE 2[83]

Symptoms of diabetes PLUS:
- Casual plasma glucose concentration ≥ 11.1 mmol/l‡

OR
- Fasting plasma glucose ≥ 7.0 mmol/l§

OR
- 2-h plasma glucose ≥ 11.1 mmol/l during an oral glucose tolerance test¶

‡ Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia and unexplained weight loss.
§ Fasting is defined as no caloric intake for at least 8 hours.
¶ The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.
Note: In the absence of unequivocal hyperglycaemia with acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day. The oral glucose tolerance test is not recommended for routine clinical use but as many as 30% of people with diabetes will not be diagnosed if only fasting measurements are done. Different criteria are used to diagnose gestational diabetes in pregnant women.

stroke and myocardial infarction only once the BP is well controlled. Hormone replacement therapy,[88] antioxidants,[89] and homeopathic and complementary medicines are of no benefit in hypertensive patients.

8.5 Holistic management of the complicated patient

The consideration of multiple risk factors leads logically to the concept of treating the related co-morbid conditions. A recently published European CVD prevention guideline[8] outlines a more holistic approach. South Africa requires the development of a similar document that takes into consideration the special needs of its population.

Hypertension can seldom be managed in isolation from other related chronic illnesses. Lifestyle modification, drug therapy and the targets of management should be broadened to include measures of other risk factors and co-morbidities, e.g. measures obesity, blood sugar and lipids as well as BP control (Table IV).

9. Management of severe hypertension

Patients with severe hypertension (stage 3 DBP ≥ 110 mmHg and/or SBP ≥ 180 mmHg) may fall into one of three categories which determine the urgency of their treatment. Patients should be managed or referred to the appropriate level of care and caregiver in accordance with local protocols. Sustained

severe hypertension requires immediate drug therapy, and lifestyle modification must be followed as soon as possible.

9.1 Asymptomatic severe hypertension

These patients are asymptomatic but have severe hypertension with or without evidence of target-organ damage or associated clinical conditions. The patient must be kept in the care setting and BP measurement repeated after resting for 1 hour. If the second measurement is still elevated at the same level, start oral therapy using 2 drugs together, 1 of which should be a low-dose thiazide-like diuretic. The other is usually a dihydropyridine CCB. Follow up within a week or earlier, escalating treatment if needed. Early referral is advised if BP is not controlled within 2 - 4 weeks.

9.2 Hypertensive urgencies and emergencies[90]

Despite advances in chronic hypertension management, hypertensive emergencies and urgencies remain serious complications. Poor compliance with effective antihypertensive management, failure of health professionals to institute effective antihypertensive therapy, failure to refer a patient with resistant hypertension timeously and failure to recognise important secondary causes appear to be critical factors. Hypertensive emergencies and urgencies can also be seen in hypertension in pregnancy and in the preoperative period. Multiple classes of intravenous antihypertensive drugs are available to treat hypertensive emergencies, and specific agents may have an advantage in a given clinical situation. Orally active agents are used to treat hypertensive urgencies and include ACE-Is, CCBs and beta-blockers. Most patients respond well to drug therapy, but problems may arise if BP is normalised rapidly.

While not common, hypertensive emergencies and urgencies are likely to be encountered by all clinicians because of the high prevalence of chronic hypertension. It is essential that all doctors are familiar with treatment. There is a paucity of information from well-conducted studies on the outcomes of various antihypertensive drugs and BP-lowering strategies. Therefore, any recommendation is based on case studies, clinical reports, comparative studies and expert opinion.

9.2.1 Hypertensive urgency[91]

This level of hypertension is symptomatic, usually with severe headache, shortness of breath and oedema. There are no immediate life-threatening neurological, renal, eye or cardiac complications such as are seen in the hypertensive emergencies (9.2.2). Ideally, all patients with hypertensive urgency should be treated in hospital.

Commence treatment with 2 oral agents and aim to lower the DBP to 100 mmHg slowly over 48 - 72 hours. This BP lowering can be achieved with the use of: (*i*) long-acting CCBs; (*ii*) ACE-I, initially used in very low doses, but avoid if there is severe hyponatraemia (serum Na < 130 mmol/l indicates hyperreninaemia and BP may fall dramatically with ACE-I); (*iii*) beta-blockers; and (*iv*) diuretics. Diuretics may potentiate the effects of the other classes of drugs when added. Furosemide should be used if there is renal insufficiency or signs of pulmonary congestion.

9.2.2 Hypertensive emergency

A hypertensive emergency exists when acute elevation of BP is associated with acute and ongoing organ damage to the kidneys, brain, heart, eyes (grade 3 or 4 retinopathy) or vascular system. These patients need rapid (within minutes to a few hours) lowering of BP to safe levels. Once a patient is identified as having a genuine hypertensive emergency, immediate hospitalisation is essential in an intensive care unit with experienced staff and modern facilities. Intravenous antihypertensive therapy, tailored to the specific type of emergency, has become the standard of care except for patients with stroke (see separate recommendations in box). The potential for harm from overzealous lowering of BP exists concurrently with the need for careful and structured BP reduction.

It should be emphasised that the definition of a hypertensive emergency does not explicitly include absolute BP levels, although most adult patients with these emergencies will have SBP > 220 mmHg and/or DBP >140 mmHg. A hypertensive emergency may also be seen at modest BP elevations, for example in a previously normotensive woman during pregnancy (eclampsia) or in the setting of acute glomerulonephritis, especially in children.

Hypertensive emergencies are uncommon, probably occurring in less than 1 - 2% of the hypertensive population, but are more common among blacks and older patients. Most patients know that they are hypertensive, and are on treatment. Hypertensive emergencies and urgencies may be seen in the immediate postoperative period after vascular surgery.

Hypertensive emergencies are poorly understood in terms of initiating factors, but a rapid rise in BP associated with increased vascular resistance is suspected as the initial derangement. Smoking, possibly through mediating endothelial injury, has long been suspected to be a risk factor and smokers have 5 times the risk of developing malignant hypertension. Thrombotic (ischaemic) stroke and intracerebral haemorrhage should be managed according to the South African Stroke Therapy Clinical Guideline (see summary on p. 354).[92]

Some common clinical hypertensive emergencies are described below.

9.2.2.1 Acute cerebrovascular syndromes

Severe hypertension is common in the setting of acute stroke. There is debate about whether or not this should be treated, and if so, to what immediate goal BP. Cerebral autoregulation

Summary management of acute stroke[92]

- Do not lower BP in acute stroke or use antihypertensive medication unless the BP is SBP > 220 mmHg or DBP > 120 mmHg, as a rapid fall may aggravate cerebral ischaemia and worsen the stroke.
- If the BP is above these levels then treatment should aim not to lower the BP by more than 15 - 20% in the first 24 hours.
- Treatment may be given orally but if the patient is unable to swallow then the use of parenteral drugs may be warranted.
- The best parenteral drugs appear to be those that are easily titrated and have a minimal effect on cerebral blood vessels, e.g. labetalol. Sodium nitroprusside should be administered in an intensive care unit because of its rapid onset of action.

is impaired, and too-rapid lowering of BP may result in the extension of an ischaemic stroke. The American Heart Association recommends treating hypertension in the setting of intracerebral bleeding when values > 180/105 mmHg, and that mean arterial pressure (DBP plus 1/3 pulse pressure) be maintained < 130 mmHg. For ischaemic stroke, BP should be observed for at least 1 - 2 hours to see if it will decline spontaneously. Only a DBP persistently > 120 mmHg or SBP > 220 mmHg should be carefully treated with an initial 20% reduction in mean arterial pressure.

9.2.2.2 Hypertensive encephalopathy

Hypertensive encephalopathy is a special cerebrovascular hypertensive emergency characterised by diffuse cerebral dysfunction with headaches, nausea, vomiting and altered levels of conscious and rarely with seizures. Hypertensive encephalopathy is frequently accompanied by retinal findings of malignant hypertension and acute renal dysfunction. Imaging with a computed tomography (CT) scan is usually normal or may show diffuse cerebral oedema. There is consensus that the gradual lowering of BP in this situation usually leads to a fairly rapid improvement in symptoms. Drugs that may be used in this setting include sodium nitroprusside and labetalol. The failure of a patient with presumed hypertensive encephalopathy to improve within 6 - 12 hours of BP reduction should prompt an aggressive additional evaluation for other causes of the encephalopathic process.

9.2.2.3 Acute cardiac syndromes

Severe hypertension in the setting of acute myocardial infarction, unstable angina, or pulmonary oedema should be treated aggressively along with all the other interventions indicated in that setting. BP treatment reduces the ischaemic load on the left ventricle. Intravenous nitroglycerin is an ideal agent for this setting because it reduces myocardial oxygen consumption and increases blood flow beyond a stenotic area. Sodium nitroprusside is a suitable additional drug that can be used alone or in combination with nitroglycerin.

9.2.2.4 Postoperative hypertension[93]

Hypertension is frequent (20 - 75%) in the postoperative state. Such severe hypertension tends to occur more in patients with poor preoperative BP control, autonomic disorders or a history of acute alcohol or cocaine use. Examples of reversible causes of hypertension in the postoperative period include pain, hypoxia, a full bladder, hypervolaemia, hypovolaemia, persistent vomiting and anxiety.

The true emergency situation should preferably be treated by an appropriate specialist. Admit the patient to an intensive care setting for parenteral drug therapy (Table XI) and close monitoring. Do not lower the initial BP by > 25% within 30 - 120 minutes. In the next 2 - 6 hours, aim towards 160/100 mmHg. This may be achieved by the use of intravenous or oral drugs.

10. Resistant (refractory) hypertension[94]

Hypertension that remains > 140/90 mmHg despite the use of 3 antihypertensive drugs in a rational combination at full doses and including a diuretic is known as refractory or resistant hypertension. The therapeutic plan must have included lifestyle measures. In older patients with isolated systolic hypertension, resistant hypertension is diagnosed when triple therapy (as above) has failed to control the BP < 160/90 mmHg. Table XII lists some causes of resistant hypertension in South Africa that should be considered in managing this condition.

The commonest cause of resistant hypertension in South Africa is probably non-adherence (lack of compliance) to lifestyle modification and medication, the unavailability of medication and other drug-related causes. Once the issues relating to lifestyle and adherence to therapy have been satisfactorily managed then consideration should be given to the addition of the fourth-line drug. Resistant hypertension should, where possible, be managed by specialist physicians. Drugs that can be used for fourth-line therapy are listed below and users should be conversant with the pharmacology of these agents:[96] (*i*) direct vasodilators: hydralazine, minoxidil; (*ii*) centrally acting drugs: methyldopa, moxonidine; (*iii*) alpha-blockers: doxazosin; (*iv*) beta-blockers: many cardioselective agents are available; and (*v*) aldosterone antagonists.

11. Special considerations for hypertension in certain populations

11.1 Blacks and Indians

At comparable BP levels blacks are prone to complications such as stroke, heart failure, and renal failure while coronary heart

Table XI. Intravenous and oral drugs for hypertensive emergency

Drug	Dose	Indications and precautions	Effect on BP
Intravenous			
Nitroglycerin (glyceryl trinitrate)	5 - 10 µg/min	Especially useful for myocardial ischaemia	BP lowering occurs in 2 - 5 minutes
Dihydralazine	10 mg every 10 - 15 minutes until either BP is controlled or a maximum of 50 mg given	Avoid in patients with myocardial ischaemia	BP lowering occurs in 10 minutes
Sodium nitroprusside	0.25 - 10 µg/kg/min diluted in 5% dextrose and adjust dose as necessary	Admission to intensive care unit An intra-arterial BP line is desirable	BP control is immediate
Labetalol	2 mg/min to a total dose of 1 - 2 mg/kg	Use where emergency caused by phaeochromocytoma Caution in acute pulmonary oedema	
Furosemide	40 - 80 mg	Acts only for 6 hours Potentiates all of the above drugs	
Oral (use only if IV drugs are not available)			
Nifedipine **Long-acting only**	Long-acting CCBs must be used to prevent rapid and dangerous BP reduction Check dosage according to CCB brand used	Preferred in black persons	
Captopril	6.25 mg as a test dose Increase to 25 mg if BP lowering is not obtained in 15 – 30 minutes	Other rapidly acting ACE-I may be used starting with a low test dose DO NOT USE if bilateral renal artery stenosis is suspected DO NOT USE if pregnancy is suspected	BP lowering in 15 - 30 minutes

disease is increasing in frequency.[97] The prevalence of diabetes mellitus and the metabolic syndrome is higher in Indians than in other ethnic groups.[98] Compared with whites, blacks respond poorly to ACE-I and beta-blockers as monotherapy, but this difference disappears once combined with diuretics. Overall CCBs show the most consistent response in blacks compared with other classes of drugs used as monotherapy.[99,100]

11.2 Children and adolescents[101]

Hypertension in children is an important issue beyond the scope of this guideline. Nevertheless measurement of BP should be a routine part of paediatric examination, and it is essential to use appropriate cuff sizes. Hypertension is defined as the SBP and DBP > 95th percentile for age and sex (Table XIII) and it is seldom primary in childhood. A detailed investigation should be undertaken for an underlying secondary cause. Referral to a specialist for evaluation and treatment is essential.

In adolescents, hypertension is increasingly linked to obesity. The international trend of poor diet and lack of exercise in children is leading to an epidemic of obesity with the early onset of hypertension and even type 2 diabetes. The early recognition of hypertension in these adolescents will be an added motivation for both children and parents to institute important lifestyle changes.

11.3 Persons living with HIV and AIDS

Prolonged highly active antiretroviral therapy (HAART) is associated with a higher prevalence of systolic hypertension.[103] This suggests that individuals taking HAART may be at increased risk of developing hypertension-related conditions and underscores the importance of BP monitoring of these individuals. When antiretroviral drugs are used, the doses of CCBs are variably influenced especially at the start or termination or change of therapy. Frequent BP and dose checks are advised. Two of the three major classes of antiretrovirals, the protease inhibitors and the non-nucleoside reverse transcriptase inhibitors, are involved in many drug interactions by inhibiting and/or inducing of the key hepatic enzyme system, cytochrome P450. CCBs are the major class of antihypertensives affected by such drug interactions.[104,105]

The first-line antiretroviral regimen is currently based on non-nucleoside reverse transcriptase inhibitors (efavirenz or nevirapine), which are enzyme inducers, thereby promoting the metabolism of all currently available CCBs, through a mechanism not well understood,[104] and potentially reducing their antihypertensive effect. If the first-line regimen fails, patients are usually switched to a protease inhibitor regimen which decreases the rate of CCB metabolism to increase CCB blood levels, with risk of hypotension. Thus particular care must be taken when patients are on first-line antiretroviral

Table XII. Causes of resistant hypertension in South Africa (adapted from JNC VI)[95]

Non-adherence to therapy	Instructions not understood
	Side-effects
	Cost of medication and/or cost of attending health care centre
	Lack of consistent and continuous primary care
	Inconvenient and chaotic dosing schedules
	Organic brain syndrome (e.g. memory deficit)
Volume overload	Excess salt intake
	Inadequate diuretic therapy
	Progressive renal damage (nephrosclerosis)
	Fluid retention from reduction of BP
Associated conditions	Smoking
	Increasing obesity
	Sleep apnoea
	Insulin resistance/hyperinsulinaemia
	Ethanol intake of more than 30 g (3 standard drinks) daily
	Anxiety-induced hyperventilation or panic attacks
	Chronic pain
	Intense vasoconstriction (Raynaud's phenomenon), arteritis
Identifiable causes of hypertension	Chronic renal disease
	Renovascular disease
	Primary aldosteronism
	Coarctation
	Cushing's syndrome
	Phaeochromocytoma
Pseudoresistance	'White coat hypertension' or office elevations
	Pseudohypertension in older patients
	Use of regular cuff on very obese arm
Drug-related causes	Doses too low
	Wrong type of diuretic
	Inappropriate combinations
	Rapid inactivation (e.g. hydralazine)
Drug actions and interactions	Non-steroidal anti-inflammatory drugs (NSAIDs)
	Sympathomimetics: nasal decongestants, appetite suppressants
	Cocaine and other recreational drugs, caffeine, oral contraceptives
	Adrenal steroids
	Liquorice (as may be found in chewing tobacco)
	Cyclosporine, tacrolimus, erythropoietin
	Antidepressants (monoamine oxidase inhibitors, tricyclics)

Table XIII. 95th percentile of BP in boys and girls 3 - 16 years of age, according to height[101]*

	Age	Height percentile for boys				Height percentile for girls			
BP	(yrs)	5th	25th	75th	95th	5th	25th	75th	95th
Systolic BP (mmHg)	3	104	107	111	113	104	105	108	110
	6	109	112	115	117	108	110	112	114
	10	114	117	121	123	116	117	120	122
	13	121	124	128	130	121	123	126	128
	16	129	132	136	138	125	127	130	132
Diastolic BP (mmHg)	3	63	64	66	67	65	65	67	68
	6	72	73	75	76	71	72	73	75
	10	77	79	80	82	77	77	79	80
	13	79	81	83	84	80	81	82	84
	16	83	84	86	87	83	83	85	86

* The height percentiles were determined with standard growth curves. Data are adapted from those of the Task Force on High BP in Children and Adolescents.[102]

therapy and the BP is controlled by CCBs – if the antiretroviral therapy should be switched to include a protease inhibitor, dramatic falls in BP may result. It is therefore better to avoid using CCBs with antiretroviral therapy.[104,105]

The metabolism of many beta-blockers may be inhibited by protease inhibitors – this is a theoretical interaction of uncertain significance. It would be prudent to start with a low dose of a beta-blocker in patients on protease inhibitors.

12. Primordial prevention

Prevention of hypertension depends on adopting strict lifestyle measures. 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 which promotes healthy behaviours and population-wide education programmes. They depend, in turn, on many factors, including political commitment, advocacy by health professionals and involvement of community leaders and the mass media.[106] Strategies are to prevent the acquisition or enhancement of CVD risk factors which include changes in lifestyle and diet in blacks brought about by rapid urbanisation. The approach should be non-pharmacological, population-based and lifestyle-linked. There is a need to develop cost-effective methods for the diagnosis and low-cost saving measures for all the risk factors of CVD.

13. Prevalence of hypertension

Prevention of hypertension depends on adopting strict lifestyle measures. There is evidence that the prevalence of hypertension and CVD is increasing rapidly in sub-Saharan Africa.[106] Two recent surveys indicated that in Tanzania[107] just under 20% of hypertensive subjects were aware of their diagnosis, approximately 10% reported receiving treatment and less than 1% were controlled (BP < 140/90 mmHg); 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 (BP < 140/90 mmHg) while 47% of females were aware of their hypertension, 29% were on treatment and only 15% were controlled (BP < 140/90 mmHg).[16]

14. Patient education

The purpose of patient education is to empower the patient to participate in good-quality hypertensive care. People with hypertension have a right to information concerning the status and progress of their condition. Empowering patients to become actively involved in the management of their hypertension is the major objective.

Effective, honest and open two-way communication between the care provider and the patient is critically important in the management of chronic lifelong conditions. Acquisition of communication and counselling skills by health professionals is essential, preferably in the vocabulary of the target population. The checklist (Annexure C) can be used as a guide for the content of hypertension patient education.

Poor adherence to therapy is the most important cause of uncontrolled BP.[108-110] Some of the obstacles to adherence are shown in Table XIV.

15. Ongoing management of the patient with hypertension

- Dose titration or stepwise increase should be carried out after 2 months if the BP remains uncontrolled and adherence is a factor.
- Once a stable target BP has been achieved, follow-up BP measurement should be performed every 3 - 6 months.
- Drug dose should be reduced if the patient presents with symptoms of postural hypotension, i.e. dizziness or > 20 mmHg fall in SBP on standing.
- Refer the patient from the primary care level to higher levels in the following circumstances: (*i*) young patients (18 - 30 years); (*ii*) pregnancy; (*iii*) uncontrolled BP despite use of 3 drugs (resistant hypertension); (*iv*) any patient with severe target-organ damage and/or severe associated clinical conditions (most patients with high added risk or very high

Table XIV. Obstacles to adherence

Treatment characteristics	Patient and illness characteristics
Long duration of therapy	Asymptomatic nature of the condition leaves people feeling
Complicated regimens	that they are not ill
Expensive medications	Chronic conditions require constant attention
Side-effects of medications	There are no immediate consequences of stopping
Lack of specific appointment times	therapy, e.g. one does not feel sick
Long waiting period at clinic or office	Social isolation
Lack of consistent and continuous primary care	Disrupted home situation
Instructions not understood	Psychiatric illnesses
Organic brain syndrome (e.g. memory deficit)	
Medicines not available	

added risk); and (*v*) hypertensive urgency or emergency.
- Most patients with low and moderate added risk can be managed at primary care level (general practitioner or clinic nurse) and can be seen every 6 months. Patients with high and very high added risk with numerous risk factors should be managed by physicians or medical subspecialists (cardiologists, nephrologists, endocrinologists) and those with a special interest in hypertension, and may need frequent visits until the BP is controlled.

16. Strategic implications for the implementation of this guideline

This section was developed by the DOH and is endorsed by the SAHS. It is mainly for those who administer health care facilities or make policy and has been included as Annexure D. The implementation of the guideline is an active process involving more than dissemination and education. It requires the full collaboration and co-operation of policy makers, administrators and funders. Both the DOH and the SAHS are committed to the full implementation of this guideline.

17. Caution

This national clinical guideline is for reference and educational purposes only and is not intended to be a substitute for the advice of appropriate health care professionals or for independent research and judgement. Neither the DOH nor the SAHS accepts responsibility or liability arising from any information contained in or any error of omission from the protocol or from the use of any information contained in it.

18. References

1. Southern African Hypertension Society. Guidelines for the management of hypertension at primary health care level. *S Afr Med J* 1995; **85:** 1321-1325.
2. Southern African Hypertension Society Executive Committee. Hypertension Clinical Guideline 2000. *S Afr Med J* 2001; **91:** 163-172.
3. Milne FJ, Pinkney-Atkinson VJ. Hypertension guideline 2003 update. *S Afr Med J* 2004; **94:** 209-226.
4. Department of Health. *Hypertension: National Programme for Control and Management at Primary Level*. Pretoria: South African Communication Service, 2003.
5. O'Brien E, Asmar R, Beilin L, *et al*. European Society of Hypertension recommendations for conventional, ambulatory and home BP measurement. *J Hypertens* 2003; **21:** 821-848.
6. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP. The JNC 7 Report. *JAMA* 2003; **289:** 2560-2572.
7. Guidelines Committee. 2003 European Society of Hypertension - European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* 2003; **21:** 1011-1053.
8. De Backer G, Ambrosioni, E, Borch-Johnsen K, *et al*. European guidelines on cardiovascular disease prevention in clinical practice. *Eur Heart J* 2003; **24:** 1601-1610.
9. 2003 World Health Organization/International Society of Hypertension (ISH) statement on the management of hypertension. *J Hypertens* 2003; **21:** 1983-1992.
10. Practice Guidelines Working Committee. Practice Guidelines for Primary Care Physicians: 2003 ESH/ESC Hypertension Guidelines. *J Hypertens* 2003; **21:** 1779-1786.
11. Williams B, Poulter NR, Brown NR, *et al*. Guidelines for management of hypertension: report of the fourth working party of the British Hypertension Society, 2004-BHS IV. *J Hum Hypertens* 2004; **18:** 139-185.
12. National Institute for Health and Clinical Excellence. *NICE and British Hypertension Society Confirm Review of Hypertension Guidelines*. Press release NICE 27 October 2005, NICE 2005/026. **www.nice.org.uk/pdf/2005_026_NICE_and_BHS_update_hypertension.pdf** (last accessed 14 March 2006).
13. Dahlof B, Sever PS, Poulter NR, *et al*. Prevention of cardiovascular events with an antihypertensive regimen of amlodipine adding perindopril as required versus atenolol adding bendroflumethiazode as required, in the Anglo-Scandinavian Cardiac Outcomes Trial-BP Lowering Arm (ASCOT-BPLA): a multicentre randomized controlled trial. *Lancet* 2005; **366:** 895-906.
14. Medical Research Council / Department of Health. *South Africa Demographic and Health Survey: Preliminary Report 1998*. Pretoria: Department of Health, 1998.
15. World Health Organization /International Society of Hypertension Guidelines for the management of hypertension. *J Hypertens* 1999; **17:** 151-185.
16. Steyn L, Gaziano T, Bradshaw D, *et al*. Hypertension in South African adults: results from the Demographic and Health Survey, 1998. *J Hypertens* 2001; **19:** 1717-1725.
17. Bradshaw D, Groenewald P, Laubscher R, *et al*. *Initial Estimates from the South African National Burden of Disease Study, 2000*. Medical Research Council Policy Brief, March 2003. Tygerberg: MRC, 2003.
18. Kearney PM, Whelton M, Reynolds K, *et al*. Global burden of hypertension: analysis of worldwide data. *Lancet* 2005; **365:** 217–223.
19. Leeder S, Raymond S, Greenberg H, *et al*. *A Race Against Time. The Challenge of Cardiovascular Disease in Developing Economies*. New York: Columbia University, 2004.
20. World Health Organization. *Reduction of Cardiovascular Burden Through Cost-effective Integrated Management of Comprehensive Cardiovascular Risk*. Geneva: WHO, 2002.
21. Pestana JAX, Steyn K, Leiman A, *et al*. The direct and indirect costs of cardiovascular disease in South Africa in 1991. *S Afr Med J* 1996; **86:** 679-684.
22. Douherty J, McIntyre D, Bloom G. Value for money in South African health care: findings of a review of health expenditure and finance. *Cent Afr J Med* 1996; **42:** 21-24.
23. World Health Organization. *CVD-Risk Management Package for Low- and Medium-Resource Settings*. Geneva: WHO, 2002.
24. O'Brien E, Asmar R, Beilin L, *et al*. on behalf of the European Society of Hypertension Working Group on BP Monitoring. European Society of Hypertension recommendations for conventional, ambulatory and home BP measurement. *J Hypertens* 2003; **21:** 821-848.
25. Pickering TG, Hall JE, Appel LJ, *et al*. Recommendations for BP measurement in humans and experimental animals, part 1: BP measurement in humans: A statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension* 2005; **45:** 142-161.
26. Rosenthal T. Seasonal variations in BP. *Am J Geriatr Cardiol* 2004; **13:** 267-272.
27. O'Brien E. Replacing the mercury sphygmomanometer. Requires clinicians to demand better automated devices. *BMJ* 2000; **320:** 815-816.
28. World Health Organization. *Affordable Technology. BP Measuring Devices for Low Resource Settings*. Geneva: WHO, 2005.
29. Verbeek WJ, Kroon AA, Kessels AGH, de Leeuw PW. Home BP measurement. A systematic review. *J Am Coll Cardiol* 2005; **46:** 743-751.
30. O'Brien E, Atkins N. A website for BP measuring devices. *BP Monitoring* 2003; **8:** 177-180.
31. Bur A, Herkner H, Vlcek M, *et al*. Classification of BP levels by ambulatory BP in hypertension. *Hypertension* 2002; **40:** 817-822.
32. World Health Organization. *Integrated Management of Cardiovascular Risk; Report of a WHO meeting, Geneva, 9-12 July 2002*. Geneva: WHO, 2002.
33. Yusuf S, Hawken S, Ôuppuu S, *et al*. Obesity and the risk of myocardial infarction in 27 000 participants from 52 countries. *Lancet* 2005; **366:** 1640-1649.
34. International Atherosclerosis Society. *Harmonized Guidelines on the Prevention Atherosclerotic Vascular Disease*. 2003. **www.athero.org/current_publications.asp** (last accessed 14 March 2006).
35. International Diabetes Federation. *The IDF Consensus Worldwide Definition of the Metabolic Syndrome*. **http://www.idf.org/webdata/docs/MetSyndrome_FINAL.pdf** (last accessed 14 March 2006).
36. Carr DB, Utzschneider KM, Hull RL, *et al*. Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. *Diabetes* 2004; **53:** 2087–2094.
37. Grundy SM, Cleeman JI, Daniels SR, *et al*. Diagnosis and management of the metabolic syndrome: An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation* 2005; **112:** 2735-2752.
38. Southern African Society for the Study of Obesity. *Guidelines for the Prevention and Management of Overweight and Obesity in South Africa*, 2003. **http://www.hypertension.org.za/ClientFiles/Guidelines/SASSO%20Guidelines.pdf** (last accessed 13 March 2006).
39. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome-a new worldwide definition. *Lancet* 2005; **366:** 1059-1062.
40. Southern African Hypertension Society. Microalbuminuria Policy Brief 2/2005. **http://www.hypertension.org.za/downloads.asp** (last accessed 14 March 2006).
41. Southern African Hypertension Society. ECG-LVH Criteria Policy Brief 1/2004. **http://www.hypertension.org.za/ClientFiles/Guidelines/sahs%20LVh%20criteria%20final.pdf**
42. Murray CJL, Lauer JA, Hutubessy RCW, *et al*. Effectiveness and cost of interventions to lower systolic BP and cholesterol: a global and regional analysis on reduction of cardiovascular disease risk. *Lancet* 2003; **361:** 717-725.
43. Consensus Statement of the Hypertension in African Americans Working Group of the International Society of Hypertension in Blacks. Management of high BP In African Americans. *Arch Intern Med* 2003; **163:** 525-541.
44. World Health Organization. *Global Strategy on Diet, Physical Activity and Health*. 57th World Health Assembly WHA57.17. April 2004.50.
45. World Health Organization. Diet, nutrition and the prevention of chronic diseases. Report of a Joint WHO/FAO Expert Consultation. *World Health Organ Tech Rep Ser* 2003; No. 916.
46. He FJ, Nowson CA, MacGregor GA. Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. *Lancet* 2006; **367:** 320-326.
47. National Department of Health. *National Guideline*: *Prevention and Management of Overweight and Obesity in South Africa*. Pretoria: DOH,
48. National Department of Health. *National Food-based Dietary Guidelines*. Pretoria: DOH,
49. National Department of Health. *National Guideline on Primary Prevention of Chronic Diseases of Lifestyle (CDL)*. Pretoria: DOH,
50. Opie LH, Schall R. Old antihypertensives and new diabetes. *J Hypertens* 2004; **22:** 1453-1458.
51. Mancia G, Grassi G, Zanchetti A. New-onset diabetes and antihypertensive drugs. *J Hypertens* 2006; **24:** 3-10.
52. Carlberg B, Samuelsson O, Lindholm LH. Atenolol in hypertension: is it a wise choice? *Lancet* 2004; **364:** 1684-1689.

53. Lindholm LH, Carlberg B, Samuelsson O. Should beta-blockers remain first choice in the treatment of primary hypertension? A meta-analysis. *Lancet* 2005; **366:** 1545-1553.
54. Morgan T, Lauri J, Bertram D, *et al*. Effect of different antihypertensive drug classes on central aortic pressure. *Am J Hypertens* 2004; **17:** 118-123.
55. Gress TW, Nieto J, Shahar E, *et al*. Hypertension and anithypertensive therapy as risk factors for type 2 diabetes mellitus. *N Engl J Med* 2000; **342:** 905-912.
56. Psaty BM, Smith NL, Siscovick DS, *et al*. Health outcomes associated with antihypertensive therapies used as first-line agents. A systematic review and meta-analysis. *JAMA* 1997; **277:** 739-745.
57. Barnett AH, Bain SC, Bouter P, *et al*. Angiotensin-receptor blockade versus converting-enzyme inhibition in type 2 diabetes and nephropathy. *N Engl J Med* 2004; **351:** 1952-1961.
58. Pfeffer MA, McMurray JJV, Velaquez EJ, *et al*. for the Valsartan in Acute Myocardial Infarction Trial Investigators. Valsartan, captopril or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. *N Engl J Med* 2003; **349:** 1893-1906.
59. Teo K, Yusuf S, Sleight P, *et al*. Rationale, design, and baseline characteristics of 2 large, simple, randomized trials evaluating telmisartan, ramipril, and their combination in high-risk patients: the Ongoing Telmisartan Alone and in Combination with Ramipril Global Endpoint Trial/Telmisartan Randomized Assessment Study in ACE Intolerant Subjects with Cardiovascular Disease (ONTARGET/TRANSCEND) trials. *Am Heart J* 2004; **148:** 52-61.
60. Dahlof B, Devereux R, de Faire U, *et al*. The Losartan Intervention For Endpoint reduction (LIFE) in Hypertension study: rationale, design, and methods. The LIFE Study Group. *Am J Hypertens* 1997; **10:** 705-713.
61. Dahlof B, Devereux RB, Kjeldsen SE, *et al*. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): A randomised trial against atenolol. *Lancet* 2002; **359:** 995–1003.
62. Ruggenenti P, Fassi A, Ilieva AP, *et al*. Preventing microalbuminuria in type 2 diabetes. *N Engl J Med* 2004; **351:** 1941-1951.
63. Parving HH, Lehnert H, Bröchner-Mortensen J, Gomis R, Andersen S, Arner P. The effect of irbesartan on the development of diabetic nephropathy in patients with type 2 diabetes. *N Engl J Med* 2001; **345:** 870-878.
64. Viberti G, Wheeldon NM. Microalbuminuria reduction with valsartan in patients with type 2 diabetes mellitus: a BP-independent effect. *Circulation* 2002; **106:** 672-678.
65. Lewis EJ, Hunsicker LG, Clarke WR, *et al*. Renoprotective effect of the angiotensin-receptor antagonist irbesartan in patients with nephropathy due to type 2 diabetes. *N Engl J Med* 2001; **345:** 851-859.
66. Brenner BM, Cooper ME, de Zeeuw D, *et al*. Effects of losartan on renal and cardiovascular outcomes in patients with type 2 diabetes and nephropathy. *N Engl J Med* 2001; **345:** 861-869.
67. PROGRESS Collaborative Group. Randomised trial of a perindopril-based blood-pressure lowering regiment among 6 105 individuals with previous stroke or transient ischaemic attack. *Lancet* 2001; **358:** 1033-1104.
68. Schrader J, Lüders S, Kulschewski A, *et al*. Morbidity and mortality after stroke, eprosartan compared with nitrendipine for secondary prevention: Principal results of a prospective randomized controlled study (MOSES). *Stroke* 2005; **36:** 1218-1226.
69. Heidenrich PA, McDonanld KM, Hastie T, *et al*. Meta-analysis of trials comparing β-blockers, calcium antagonists and nitrates for stable angina. *JAMA* 1999; **281:** 1927-1936.
70. Gottlieb SS, McCarter RJ, Vogel RA. Effect of beta-blockade on mortality among high-risk and low-risk patients after myocardial infarction. *N Engl J Med* 1998; **339:** 489-497.
71. Dagenais GR, Yusuf S, Bourassa MG, *et al*. HOPE Investigators. Effects of ramipril on coronary events in high-risk persons: results of the Heart Outcomes Prevention Evaluation Study. *Circulation* 2001; **104:** 522-526.
72. Poole-Wilson PA, Swedberg K, Cleland JGF, *et al*. Comparison of carvedilol and metoprolol on clinical outcome in patients with chronic heart failure in the Carvedilol Or Metoprolol European Trial (COMET): randomised control trial. *Lancet* 2003; **362:** 7-13.
73. American College of Cardiology/American Heart Association. *2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult*. A report of the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure): developed in collaboration with the American College of Chest Physicians and the International Society for Heart and Lung Transplantation: endorsed by the Heart Rhythm Society. *Circulation* 2005; **112:** e154-235.
74. Pitt B, Remme W, Zannad F, *et al*. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. *N Engl J Med* 2003; **348:** 1309-1321.
75. Pitt B, Remme W, Cody R, *et al*. The effect of spironolactone on morbidity and mortality with severe heart failure. Randomized Aldactone Evaluation Study. *N Engl J Med* 1999; **341:** 709-717.
76. Young JB, Dunlap ME, Pfeffer MA, *et al*. Candesartan in Heart failure Assessment of Reduction in Mortality and morbidity (CHARM) Investigators and Committees. Mortality and morbidity reduction with Candesartan in patients with chronic heart failure and left ventricular systolic dysfunction: results of the CHARM low-left ventricular ejection fraction trials. *Circulation* 2004; **110:** 2618-2626.
77. Heart Outcomes Prevention Evaluation (HOPE) Study Investigators. Effects of ramipril on cardiovascular and microvascular outcomes in people with diabetes mellitus: results of the HOPE study and MICRO-HOPE substudy. *Lancet* 2000; **355:** 253-259.
78. Lewis EJ, Hunsicker LG, Bain RP, *et al*. The effect of angiotensin-converting enzyme inhibition on diabetic nephropathy. The collaborative study group. *N Engl J Med* 1993; **329:** 1456-1462.
79. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). *JAMA* 1991; **265:** 3255-3264.
80. Staessen JA, Fagard R, Thijs L, *et al*. for the Systolic Hypertension in Europ (Syst-Eur) Trial Investigators. Randomised double-blind comparison of placebo and active treatment for older people with isolated systolic hypertension. *Lancet* 1997; **350:** 757-764.
81. Julius S, Kjeldsen SE, Weber M, *et al*. Outcomes in hypertensive patients at high cardiovascular risk treated with regimens based on valsartan or amlodipine: the VALUE randomised trial. *Lancet* 2004; **363:** 2022-2031.
82. European Guidelines on Cardiovascular Disease Prevention in Clinical Practice. Executive summary. *Eur Heart J* 2003; **24:** 1601-1610.
83. Society for Endocrinology Metabolism and Diabetes of South Africa. Revised SEMDSA Guidelines for diagnosis and management of type 2 diabetes mellitus for primary health care in 2002. **www.semdsa.org.za** (last accessed 14 March 2006).
84. Sacks FM, Svetkey LP, Vollmer WM, *et al*. Effects on BP of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-sodium Collaborative Research Group. *N Engl J Med* 2001; **135:** 1019-1028.
85. Murray CJL, Lauer JA, Hutubessy RCW, *et al*. Effectiveness and cost of interventions to lower systolic BP and cholesterol: a global and regional analysis on reduction of cardiovascular disease risk. *Lancet* 2003; **361:** 717-725.
86. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20 536 high-risk individuals: a randomized placebo-controlled trial. *Lancet* 2002; **360:** 7-22.
87. Cholesterol Treatment Trialists' (CTT) Collaborators, Baigent C, Keech A, Kearney PM, Blackwell L, Buck G, Pollicino C, Kirby A, Sourjina T, Peto R, Collins R, Simes R. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90 056 participants in 14 randomised trials of statins. *Lancet* 2005; **366:** 1267-1278.
88. Manson JE, Hsia J, Johnson KC, *et al*. Estrogen plus progestin and the risk of coronary heart disease. *N Engl J Med* 2003; **349:** 523-534.
89. The ALLHAT Officers and Coordinators for the ALLHAT Cooperative Research Group. Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs. diuretic: The Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). *JAMA* 2002; **288:** 2981-2997.
90. Vidt DG. Hypertension curriculum review: Hypertensive crises: emergencies and urgencies. *J Clin Hypertens* 2004; **6:** 520-525.
91. Handler J. Hypertensive urgency. *J Clin Hypertens* 2006; **8:** 61-64.
92. Neurological Association of South Africa/ South African Medical Association. Stroke Therapy Guideline. *S Afr Med J* 2000; **90:** 276-306.
93. Lindenauer PK, Pekow P, Wang K, *et al*. Perioperative beta-blocker therapy and mortality after major noncardiac surgery. *N Engl J Med* 2005; **353:** 349-361.
94. Kaplan NM. Resistant hypertension. *J Hypertens* 2005; **23:** 1441-1444.
95. Joint National Committee on Detection, Evaluation and Treatment of High BP. The sixth report of the Joint National Committee on Detection, Evaluation and Treatment of High BP (JNC VI). *Arch Intern Med* 1997; **157:** 2413-2446.
96. Gibbons CJ, ed. *South African Medicines Formulary*. 7th ed. Cape Town: South African Medical Association, 2005.
97. Opie LH, Seedat YK. Hypertension in sub-Saharan African populations. *Circulation* 2005; **112:** 3562-3568.
98. Seedat YK. Diabetes mellitus in South African Indians. *British Journal of Diabetes and Vascular Disease* 2005; **5:** 249-251.
99. Sareli P, Radevski IV, Valtchanova ZP, *et al*. Efficacy of different drug classes used to initiate antihypertensive treatment in black subjects: results of a randomized trial in Johannesburg, South Africa. *Arch Intern Med* 2001; **161:** 965-971.
100. Brewster LM, van Montfrans GA, Kleijnen J. Systematic review: antihypertensive drug therapy in black patients. *Ann Intern Med* 2004; **141:** 614-627.
101. Sinaiko RS. Hypertension in children. *N Engl J Med* 1996; **335:** 1968-1973.
102. National Heart, Lung, and Blood Institute's Task Force on BP Control in Children. Report of the Task Force on BP Control in Children – 1977. *Pediatrics* 1977; **59:** Suppl, 797-820.
103. Seaberg EC, Munoz A, Ming L, *et al*. Association between highly active anti-retroviral therapy and hypertension in a large cohort of men followed from 1984 to 2003. *AIDS* 2005; **19:** 953 - 960.
104. Glesby MJ, Bassett R, Alston-Smith B, *et al*. AIDS Clinical Trials Group A5088 Protocol Team. Pharmacokinetic interactions between indinavir plus ritonavir and calcium channel blockers. *Clin Pharmacol Ther* 2005; **78:** 143 - 153.
105. De Maat M, Ekhart GC, Huitema ADR, *et al*. Drug interactions between antiretroviral drugs and comedicated agents. *Clin Phamacokinet* 2003; **42:** 223-282.
106. Lemogoum D, Seedat YK, Mabadeje AF, *et al*. Recommendations for prevention, diagnosis and management of hypertension and cardiovascular risk factors in sub-Saharan Africa. *J Hypertens* 2003; **21:** 1993-2000.
107. Edwards R, Unwin N, Mugusi F, *et al*. Hypertension prevalence and care in an urban and rural area of Tanzania. *J Hypertens* 2000; **18:** 145-152.
108. World Health Organization. *Adherence to Long-term Therapies*: *Evidence for Action*. Geneva: WHO, 2003.
109. Burt VL, Whelton P, Roccella EJ, *et al*. Prevalence of hypertension in the US adult population. Results from the Third National Health and Nutrition Examination Survey, 1988-1991. *Hypertension* 1995; **25:** 305–313.
110. Hershey JC, Morton BG, Davis JB, *et al*. Patient compliance with antihypertensive medication. *Am J Public Health* 1980; **70:** 1081-1089.

Annexure A: Methodology

The Southern African Hypertension Society (SAHS) Advisory Panel Committee and representatives of the National Department of Health (DOH) decided to produce a revised joint hypertension guideline in July 2004. The following Joint National Hypertension Guideline Working Group was appointed: Prof Y K Seedat (chairperson), Ms M A Croasdale,

Prof F J Milne, Prof L H Opie, Dr V J Pinkney-Atkinson, Prof B L Rayner, Prof Y Veriava.

Three meetings of the SAHS Advisory Panel were held in June 2004, March 2005 and 1&2 October 2005 in which the DOH representatives participated. The Joint National Hypertension Guideline Working Group held several meetings.

The most recent version of the guideline was discussed in the light of more recent studies and the South African health care environment and was revised into a new draft. By October 2005 a draft guideline with the relevant references was presented to the Working Group and SAHS Advisory Panel. The purpose of the meetings was to consider the content of the draft guideline and either endorse or amend the document.

The proceedings of the October 2005 were audio recorded and transcribed for future reference.

The final draft document was revised according to the proceedings of the consensus meetings and also incorporates a further series of comments circulated after the meeting. Two versions of the final guideline were circulated widely via the SAHS website and by direct e-mails.

Comments on the guideline were solicited and included after consideration by the Joint National Hypertension Working Group.

The SAHS received no financial support for the development of these guidelines.

Annexure B. Adult body mass index chart

Height	feet	4'10"	4'11"	5'	5'1	5'2"	5'3"	5'4"	5'5"	5'6"	5'7"	5'8"	5'9"	5'10"	5'11"	6'	6'1"	6'2"	6'3"	6'4"	6'5"	6'6"	6'7"
	inches	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79
	cm	147	150	152	155	157	160	163	165	168	170	173	175	178	180	183	185	188	191	193	196	198	201
Weight																							
lb	kg																						
100	45.45	20.9	20.2	19.6	18.9	18.3	17.8	17.2	16.7	16.2	15.7	15.2	14.8	14.4	14.0	13.6	13.2	12.9	12.5	12.2	11.9	11.6	11.3	11.0
105	47.73	22.0	21.3	20.5	19.9	19.2	18.6	18.1	17.5	17.0	16.5	16.0	15.5	15.1	14.7	14.3	13.9	13.5	13.2	12.8	12.5	12.2	11.9	11.6
110	50.00	23.0	22.3	21.5	20.8	20.2	19.5	18.9	18.3	17.8	17.3	16.8	16.3	15.8	15.4	14.9	14.5	14.2	13.8	13.4	13.1	12.7	12.4	12.1
115	52.27	24.1	23.3	22.5	21.8	21.1	20.4	19.8	19.2	18.6	18.0	17.5	17.0	16.5	16.1	15.6	15.2	14.8	14.4	14.0	13.7	13.3	13.0	12.7
120	54.55	25.1	24.3	23.5	22.7	22.0	21.3	20.6	20.0	19.4	18.8	18.3	17.8	17.3	16.8	16.3	15.9	15.4	15.0	14.6	14.3	13.9	13.5	13.2
125	56.82	26.2	25.3	24.5	23.7	22.9	22.2	21.5	20.8	20.2	19.6	19.0	18.5	18.0	17.5	17.0	16.5	16.1	15.7	15.2	14.9	14.5	14.1	13.8
130	59.09	27.2	26.3	25.4	24.6	23.8	23.1	22.4	21.7	21.0	20.4	19.8	19.2	18.7	18.2	17.7	17.2	16.7	16.3	15.9	15.4	15.1	14.7	14.3
135	61.36	28.3	27.3	26.4	25.6	24.7	24.0	23.2	22.5	21.8	21.2	20.6	20.0	19.4	18.9	18.3	17.8	17.4	16.9	16.5	16.0	15.6	15.2	14.9
140	63.64	29.3	28.3	27.4	26.5	25.7	24.9	24.1	23.3	22.6	22.0	21.3	20.7	20.1	19.6	19.0	18.5	18.0	17.5	17.1	16.6	16.2	15.8	15.4
145	65.91	30.4	29.3	28.4	27.5	26.6	25.7	24.9	24.2	23.5	22.8	22.1	21.5	20.8	20.3	19.7	19.2	18.7	18.2	17.7	17.2	16.8	16.4.	16.0
150	68.18	31.4	30.4	29.4	28.4	27.5	26.6	25.8	25.0	24.3	23.5	22.9	22.2	21.6	21.0	20.4	19.8	19.3	18.8	18.3	17.8	17.4	16.9	16.5
155	70.45	32.5	31.4	30.3	29.3	28.4	27.5	26.7	25.8	25.1	24.3	23.6	22.9	22.3	21.7	21.1	20.5	19.9	19.4	18.9	18.4	17.9	17.5	17.1
160	72.73	33.5	32.4	31.3	30.3	29.3	28.4	27.5	26.7	25.9	25.1	24.4	23.7	23.0	22.4	21.7	21.2	20.6	20.0	19.5	19.0	18.5	18.1	17.6
165	75.00	34.6	33.4	32.3	31.2	30.2	29.3	28.4	27.5	26.7	25.9	25.1	24.4	23.7	23.1	22.4	21.8	21.2	20.7	20.1	19.6	19.1	18.6	18.2
170	77.27	35.6	34.4	33.3	32.2	31.2	30.2	29.2	28.3	27.5	26.7	25.9	25.2	24.4	23.9	23.1	22.5	21.9	21.3	20.7	20.2	19.7	19.2	18.7
175	79.55	36.7	35.4	34.2	33.1	32.1	31.1	30.1	29.2	28.3	27.5	26.7	25.9	25.2	24.5	23.8	23.1	22.5	21.9	21.3	20.8	20.3	198	19.3
180	81.82	37.7	36.4	35.2	34.1	33.0	32.0	31.0	30.0	29.1	28.3	27.4	26.6	25.9	25.2	24.5	23.8	23.2	22.5	22.0	21.4	20.8	20.3	19.8
185	84.09	38.7	37.4	36.2	35.0	33.9	32.8	31.8	30.8	29.9.	29.0	28.2	27.4	26.6	25.9	25.1	24.5	23.8	23.2	22.6	22.0	21.4	20.9	20.4
190	86.36	39.8	38.5	37.2	36.0	34.8	33.7	32.7	31.7	30.7	29.8	28.9	28.1	27.3	26.6	25.8	25.1	24.4	23.8	23.2	22.6	22.0	21.4	20.9
195	88.64	40.8	39.5	38.2	36.9	35.7	34.6	33.5	32.5	31.5	30.6	29.7	28.9	28.0	27.3	26.5	25.8	25.1	24.4	23.s	23.2	22.6	22.0	21.5
200	90.91	41.9	40.5	39.1	37.9	36.7	35.5	34.4	33.4	32.3	31.4	30.5	29.6	28.8	28.0	27.2	26.4	25.7	25.1	24.4	23.8	23.2	22.6	22.0
205	93.18	42.9	41.5	40.1	38.8	37.6	36.4	35.3	34.2	35.2	32.2	31.2	30.3	29.5	28.7	27.9	27.1	26.4	25.7	25.0	24.4	23.7	23.1	22.6
210	95.45	44.0	42.5	41.1	39.8	38.5	37.3	36.1	35.0	34.0	33.0	32.0	31.1	30.2	29.4	28.5	27.8	27.0	26.3	25.6	25.0	24.3	23.7	23.1
215	97.73	45.0	43.5	42.1	40.7	39.4	38.2	37.0	35.9	34.8	33.7	32.8	31.8	30.9	30.0	29.2	28.4	27.7	26.9	26.2	25.5	24.9	24.3	23.7
220	100.00	46.1	44.5	43.1	41.7	40.3	39.1	37.8	36.7	35.6	34.5	33.5	32.6	31.6	30.7	29.9	29.1	28.3	27.6	26.8	26.1	25.5	24.9	24.2
225	102.27	47.1	45.5	44.0	42.6	41.2	39.9	38.7	37.5	36.4	35.3	34.3	33.3	32.4	31.4	30.6	29.7	28.9	28.2	27.4	26.7	26.1	25.4	24.8
230	104.55	48.2	46.6	45.0	43.5	42.2	40.8	39.6	38.4	37.2	36.1	35.0	34.0	33.1	32.1	31.3	30.4	29.6	28.8	28.1	27.3	26.6	26.0	25.3
235	106.82	49.2	47.6	46.0	44.5	43.1	41.7	40.4	39.2	38.0	36.9	35.8	34.8	33.8	32.8	31.9	31.1	30.2	29.4	28.7	27.9	27.2	26.5	25.9
240	109.09	50.3	48.6	47.0	45.4	44.0	42.6	41.3	40.0	38.8	37.7	36.6	35.5	34.5	33.5	32.6	31.7	30.9	30.1	29.3	28.5	27.8	27.1	26.4
245	111.36	51.3	49.6	47.9	46.4	44.9	43.5	42.1	40.9	39.6	38.5	37.3	36.3	35.2	34.2	33.3	32.4	31.5	30.7	29.9	29.1	28.4	27.7	27.0
250	113.64	52.4	50.6	48.9	47.3	45.8	44.4	43.0	41.7	40.4	39.2	38.1	37.0	35.9	34.9	34.0	33.1	32.2	31.3	30.5	29.7	29.0	28.2	27.5
255	115.91	53.4	51.6	49.9	48.3	46.7	45.3	43.9	42.5	41.2	40.0	38.9	37.7	36.7	35.6	34.7	33.7	32.8	31.9	31.1	30.3	29.5	28.8	28.1
260	118.18	54.5	52.6	50.9	49.2	47.7	46.2	44.7	43.4	42.1	40.8	39.6	38.5	37.4	36.3	35.3	34.4	33.5	32.6	31.7	30.9	30.1	29.4	28.6
265	120.45	55.5	53.6	51.9	50.2	48.6	47.0	45.6	44.2	42.9	41.6	40.4	39.2	38.1	37.0	36.0	35.0	34.1	33.2	32.3	31.5	30.7	29.9	29.2
270	122.73	56.5	54.6	52.8	51.1	49.5	47.9	46.4	45.0	43.7	42.4	41.1	40.0	38.8	37.7	36.7	35.7	34.7	33.8	32.9	32.1	31.3	30.5	29.7
275	125.00	57.6	55.7	53.8	52.1	50.4	48.8	47.3	45.9	44.5	43.2	41.9	40.7	39.5	38.4	37.4	36.4	35.4	34.4	33.5	32.7	31.8	31.0	30.3
280	127.27	58.6	56.7	54.8	53.0	51.3	49.7	48.2	46.7	45.3	43.9	42.7	41.4	40.3	39.1	38.1	37.0	36.0	35.1	34.2	33.3	32.4	51.6	30.8
285	129.55	59.7	57.7	55.8	54.0	52.2	50.6	49.0	47.5	46.1	44.7	43.4	42.2	41.0	39.8	38.7	37.7	36.7	35.7	34.B	33.9	33.0	32.2	31.4
290	131.82	60.7	58.7	56.8	54.9	53.2	51.5	49.9	48.4	46.9	45.5	44.2	42.9	41.7	40.5	39.4	38.3	37.3	36.3	35.4	34.5	33.6	32.7	31.9
295	134.09	61.8	59.7	57.7	55.9	54.1	52.4	50.7	49.2	47.7	46.3	44.9	43.7	42.4	41.2	40.1	39.0	38.0	36.9	36.0	35.1	34.2	33.3	32.5
300	136.36	62.8	60.7	58.7	56.8	55.0	53.3	51.6	50.0	48.5	47.1	45.7	44.4	43.1	41.9	40.8	39.7	38.6	37.6	36.6	35.6	34.7	33.9	33.0

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Annexure C. Therapeutic education for patient checklist

- The major objective is to empower all patients to participate actively in management of their non-communicable chronic diseases/conditions.
- Provide information to patients so that they can understand hypertension and its consequences if not treated adequately. Involve the patient and family or caregiver in the management.
- Inform patients of the distinction between having a risk factor and having a disease and the benefits of controlling risk factors.
- Reinforce importance of lifestyle modification at each visit.
- Inform patients of their BP reading at every visit and whether BP is controlled or what the target should be.
- Emphasise the importance of adherence to the management protocol.
- Patients must know the name, strength and dose of the drug(s) prescribed, the frequency of doses and the necessity of regular ongoing use.
- Inform patients on how to deal with side-effects.
- Patients must be made aware of drug interactions and food/drug interactions.
- Tell patients to take the morning dose on the day of each visit to the health service.
- Ask patients to return drug containers, even if they are empty, at each visit.
- Support groups for patients are essential and need to be established at all facilities. The focus should be on self-care and self-monitoring, emotional needs, cultural differences, discrimination, change management and behavioural change.
- Counsel patients with hypertension who may have an excessive fear of strokes or other consequences of hypertension.
- Educate patients to inform all health care providers they visit regarding their hypertension and what drugs they are taking.
- Encourage patients to request BP measurement at each visit.

Annexure D. Strategic implications for the implementation of this guideline

This section was developed by the DOH and is endorsed by the SAHS. It is mainly for those who administer health care facilities or make policy. The implementation of the guideline is an active process involving more than dissemination and education. It requires the full collaboration and co-operation of policy makers, administrators and funders. Both the DOH and SAHS are committed to the full implementation of this guideline.

The key elements of focus to improve the management of hypertension and any other non-communicable chronic diseases/conditions are: (*i*) communication; (*ii*) continuity; (*iii*) co-ordination; (*iv*) comprehensiveness; (*v*) community linkages; (*vi*) caring ethos; (*vii*) care of high quality; (*viii*) competence; (*ix*) partnerships – working together to deliver best possible care; (*x*) performance – delivering quality care; (*xi*) professionals – the right people delivering service; (*xii*) patient access – delivering fast and convenient care; (*xiii*) patient empowerment – rights and needs are met; and (*xiv*) prevention – promotion of healthy living.

If the above key elements are considered this will enable the creation of innovative care models suitable for developing communities that are long-term and will prevent non-communicable chronic diseases and associated risk factors from developing.

- Control the progression of the non-communicable chronic diseases or their risk factors, increase survival and enhance quality of life.
- Allow health professionals, patients and families to share complementary knowledge and skills, thus allowing patients to become active partners in management of their non-communicable diseases.
- Allow for a lifestyle, drug and self-management strategy with community and family support.
- Encourage participation of a broad spectrum of care organisations, professionals and informal caregivers.
- Patient-centred care and service is desired where there is one-to-one communication and group education. Inform patients on how, where and when to access help and from whom. A patient-orientated, tolerant, caring, concerned attitude

is required on the part of health care providers. The latter should be sensitive to patients' socio-economic conditions and cultural history. Empathic communication builds trust and is a potent motivator. Implement long-term care models where there is a dedicated care provider, dedicated clinic time, chronic disease register, etc.

- A paradigm shift is needed to adjust the health delivery system. To make a tangible difference in non-communicable disease morbidity and mortality, a paradigm shift is needed. In order to enhance this paradigm shift, a shift from a single risk factor approach to a comprehensive risk management approach is needed and chronic/long-term care models need to be implemented. Effective chronic/long-term care requires a different kind of health care delivery system in which there is co-ordinated comprehensive care. There should be a move from a 'find it and fix it' model to a co-ordinated and comprehensive continuum of care, with extended regular contact. The implementation of dedicated services is a clinic organisational issue. It should be implemented at primary level for chronic diseases. Resources and the provision of care for patients with non-communicable chronic diseases and disabilities should be strengthened and decentralised at and to primary level. The chronic long-term care model differs from acute care models and the delivery of service should reflect this.
- Institute dedicated hypertension service (team and time) by using a team of health professionals specifically assigned to deal with patients with hypertension. As far as possible patients should see the same health care provider at each visit. The team members should be appropriately trained and carefully selected and not rotated. Rotation makes training one of the major cost drivers in the delivery of long-term care. An overall co-ordinator for chronic care/long-term care should be selected and be responsible for the treatment outcomes in the facility. All patients with hypertension require a dedicated hypertension time, e.g. day of week, especially if dedicated hypertension health professionals are not available. Patients should never be treated at the same time as patients with acute, curable disease unless the latter is the reason for visiting the clinic. This will require an effective triage system. The hallmarks of a dedicated hypertension service/care are as follows:
  - Effective, with the right level of care provided at the right time, by the right persons.
  - Efficient, with appropriate integrated care packages across the levels of care. This will require an effective, well-defined referral system, known to patients and health care providers and even an appointment system.
  - Informative, with patients having access to understandable information that allows them to make rational decisions about care options, treatment, lodging of complaints, etc.
  - Equitable, so that all patients benefit from service/care that is consistent, continuous, available and accessible.
  - Promotive and preventive aspects must be included in the integrated hypertension care package, e.g. therapeutic education, life skills training, etc.
- Adequate equipment must be available to perform the tasks and observations as listed in this guideline.
- Drug therapy. Relevant and appropriate drugs should be available at all times and not be changed except by the prescriber. Patients should receive the total amount of drugs as prescribed. Procurement and distribution systems should be more effective.
- National record keeping in the form of chronic disease registers should be implemented at all care facilities to monitor control and compliance of patients.
- Patient/health care provider ratio should be reasonable.
- Patient record/file management should be sequential and systematic with each entry dated and signed as appropriate. All information should be documented on the record and no loose papers or stickers should be allowed.
- Self-monitoring equipment should be made available to those patients as recommended in the discussion on self-measurement of BP (see section 4.3). Each support group must have validated self BP measuring devices for group use.