



المجموعة السعودية لرعاية ضغط الدم

SHMS



اللجنة الوطنية لضغط الدم

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Saudi Hypertension Management Guidelines

2007

Synopsis

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The Group appreciates the unconditional and professional support of Pfizer



Abbreviations

ACCs	Associated Clinical Conditions	ECG	Electro-cardiogram
ACEIs	Angiotensin Converting Enzyme-Inhibitors	HDL-C	High density Lipoprotein-Cholesterol
α-Bs	Alpha-Blockers	HTN	Hypertension
ARBs	Angiotensin Receptors Blockers	I.V.	Intra-venous
β-Bs	Beta-Blockers	LDL-C	Low Density Lipoprotein-Cholesterol
BP	Blood Pressure	LV	Left Ventricle
CCBs	Calcium Channel Blockers	MI	Myocardial Infarction
CBC	Complete Blood Count	NSAIDs	Non-Steroidal Anti-Inflammatory Drugs
CHD	Coronary Heart Disease	OSA	Obstructive Sleep Apnea
CHF	Congestive Heart Failure	SBP	Systolic Blood Pressure
CRD	Chronic Renal Disease	TIA	Transient Ischemic Attack
DBP	Diastolic Blood Pressure	TOD	Target Organ Damage
DM	Diabetes Mellitus		

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Preface by the Minister of Health

Our country aspires to establish a sustainable systematic health development process to all citizens including prevention of common diseases and their complications to ensure a public well being. Therefore, the Government of the Custodian of the Two Holy Mosques was keen to focus on setting up a group of mechanisms with a view to improve and implement an adequate range of comprehensive health services programs through the Ministry of Health and other related health institutions.

As a result, the Ministry of Health has adopted many health policies and programs one of which is establishing a Commission for the Management of Hypertension. This disease affects 20 percent of the Saudi population, children and adults alike, and is a leading cause of cardiovascular, cerebrovascular and renal diseases.

The National Committee for Hypertension has an important role in translating guidelines and programs into practical plans of actions, unified in a protocol for early diagnosis and proper management of this health dilemma and its complications. Another role for the commission is the development of a national awareness and educational programs addressed to health care professionals and the general public.

The committee has to develop, in collaboration with interested groups, research and evidence based knowledge database specific for issues related to our culture and heritage such as the relation of this disease to Ramadan fasting and Hajj. These scientific resources should be made readily accessible online for health care providers. The commission will be responsible for providing brochures and pamphlets for health care providers use.

The committee shall develop locally adopted rules and regulations to measure and control hypertension based on scientific evidence and local circumstances. Such rules and regulations will be disseminated throughout the country with support and participation of existing programs and organizations.

The Ministry of Health is intending to have the program of the National Committee for Hypertension throughout the country at governmental and non-governmental organizations as a mean for continuous quality improvement on the detection and control of Hypertension.

In this regard, I would like to express my deep appreciation to the members of the Saudi Hypertension Management Group (SHMS) for their efforts in developing these guidelines for the direct use by healthcare providers.

May all of us pray to Allah, the Almighty for more success.

Dr. Hamad Bin Abdullah Al-Manea

M.B.B.Ch, PhD (MD)

Minister of Health

Prologue

Hypertension affects more than 20% of the adult Saudi population with expected increasing prevalence. It is an important modifiable risk factor for cardiovascular diseases. Despite overwhelming evidence that lowering BP reduces morbidity and mortality, its management remains frequently sub-optimal. This is largely due to a sinister combination of poor patient compliance and health care provider's indifference. The Saudi Hypertension Management Group (SHMS) was established in 2001 to develop practical guidelines for the management of HTN in Saudi Arabia. The following guidelines were based on the best available evidence in the literature and in accordance with the recent national and international guidelines for management of HTN (1999 WHO/ISH Guidelines for the Management of Hypertension, German Guidelines for Management of Hypertension 2001, 2003 European Guidelines for the Management of Hypertension, JNC-VII 2003). Special attention was given to our local circumstances and cultural background. Our guidelines take into consideration a few issues we think are important for the management of a hypertensive patient in general practice that are usually not included in most guidelines: A special chapter is devoted to correct measurement of BP, the pediatric group with age-specific recommendations, the elderly hypertensive patients and patients with other diseases are some examples, a list of medications for treatment of HTN available currently in the market in Saudi Arabia is attached. As detection of the affected patients is the most important factor in the population related effort to control the disease and as an implementation of any guideline remains as the most critical step, gaining the support of the health authorities is mandatory in lieu of accomplishing our goals. The Ministry of Health, represented by its General Directorate for Non-Communicable Diseases, welcomed the establishment of the group and supported these guidelines and SHMS mission.

The Minister of Health issued an order to establish the National Committee for Hypertension to work on the translation of updated guidelines into practical actions and protocols to optimally detect and treat HTN in Saudi Arabia.

Osman Alfurayh, MD, CPE

Chairman, Saudi Hypertension management Group;
Chairman; National Committee for Hypertension

The Need for Specific Guidelines for the Management of Hypertension in Saudi Arabia

Hypertension is one of the conditions for which guidelines generated by different organizations offer discordant recommendations. Although this may be because different values may be placed on the prevention of certain outcomes by different organizations, concerns have been raised that guidelines variability may reflect methodological deficiencies during their development. Because guidelines generated without a systematic review of the literature and without critical appraisal of the supporting evidence would be more likely to reflect the biases of participants from a certain geographical region, it would not be surprising if they were not similar with other guidelines in the other areas developed by different authorities.

The *Saudi Hypertension Management Group* (SHMS) worked from a team of diverse professionals in the field of HTN for more than two-years. SHMS has reviewed the literature and previously published guidelines from North America and Europe as well as all relevant recent studies on HTN and its management.

Specific issues related to *fasting in Ramadan* and *performing Hajj* was given special attention. The central goal of our group is to close as many gaps in the evidence-finding process and to answer yet many questions related to both subjects.

Most HTN guidelines continue to neglect implementation. Thus we aim to implement specific strategies that help the adoption of these guidelines. These strategies include: Multifaceted interventions (involving reminder systems at the point of care).

- Use of the Internet for dissemination and discussions of the guidelines
- Downloading service to hand-held computers
- Patient-mediated methods are significant for improving guideline implementation

The health care system in Saudi Arabia provides a good opportunity to improve detection and treatment of HTN, as the Ministry of Health is the sole responsible provider for the health care of most individuals living in the country. Our group, in collaboration with the Directorate of Non-Communicable Diseases of Ministry of Health, is establishing training programs aiming at providing health care professionals proper management of the disease.

From a public health point of view, it is not relevant to debate which drugs are more efficacious. The most important issue in HTN management is the initial detection of patients at risk. In our community, many hypertensive patients are either unaware of their disease or are not treated properly for it.

In order to address these problems, our group has initiated a process to provide continuously updated, evidence-based HTN guidelines tailored to the needs of the local community. This process involves regular systematic reviews of the relevant literature, on-line discussion of the reviewed subjects by the upcoming members, as well as organizing meetings to debate the guidelines and any updates. This is the first revision of our guidelines with minor changes and corrections, still your input and critiques are welcomed.

These updated recommendations will be presented regularly at appropriate congresses and scientific meetings. They will also be published in journals for health care professionals and on the Website of the group (www.saudi-hypertension.org). This website will be the forum to present and discuss all chapters for interested professionals.

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Part I: Definition and Classification

Definition

Hypertension is defined as persistent elevation of SBP \geq 140 mm Hg and/or DBP \geq 90 mm Hg in adults not on anti-hypertensive medications.

Classification of Blood Pressure Levels

Based on the most recent evidences, BP levels may be classified as follows:

Table 1: Classification of Blood Pressure Levels.

Category	SBP (mm Hg)		DBP (mm Hg)
Normal	< 120	<i>And</i>	< 80
Pre-Hypertension	120 – 139	<i>Or</i>	80 – 89
Hypertension: Grade I	140 – 159	<i>Or</i>	90 – 99
Hypertension: Grade II	\geq 160	<i>Or</i>	\geq 100

Part II: Clinical Evaluation

The Clinical Evaluation aims at:

- Establishing the diagnosis of HTN
- Identifying secondary HTN
- Detecting additional risk factors of cardiovascular disease
- Determining Target Organ Damage(TOD) and Associated Clinical Conditions (ACCs)

The Clinical Evaluation includes:

- History and physical examination
- BP measurement
- Basic investigations

Blood Pressure measurement:

Accurate BP measurement is of the utmost importance. Patients should have three to five minutes of rest in a quiet environment. BP should be measured in a sitting position with the arm supported at heart level. The measurement of BP in a standing position is

indicated periodically in diabetics and the elderly to look for orthostatic hypotension. The auscultation method of BP measurement with a calibrated device and an appropriate sized cuff (cuff bladder encircling at least 80 % of the arm) should be used. The cuff should be wrapped around the arm smoothly allowing two fingers to be placed under the cuff comfortably. SBP is the pressure recorded when the Korotkoff sound just starts to be audible with the stethoscope at the brachial artery (phase I) and DBP is the pressure recorded at which the Korotkoff sound disappears (phase V).

The diagnosis of HTN is made after the measurement of BP on three different visits. Two BP measurements on each visit should be carried out to obtain an average BP.

Out-of-office BP measurement that includes home BP measurement (self-monitoring) and ambulatory BP monitoring, should be regarded as supplementary and not substitute for office BP measurement.

Basic investigations

Basic laboratory investigations include urine analysis, hemoglobin, fasting plasma glucose, lipid profile, creatinine, uric acid, and potassium as well as ECG.

White Coat Hypertension

“White coat” HTN (isolated office HTN) is a persistent elevation of BP in the physician’s office with normal BP at home or by ambulatory BP monitoring. It affects 10%-20% of grade I and grade II hypertensive patients. As supported in multiple studies, no increase in cardiovascular morbidity or mortality has been noted among patients with white coat HTN over as long as ten years, however they need regular follow up.

Secondary Hypertension

About 10% of cases of HTN are due to secondary causes such as renovascular and renoparenchymal diseases. The main causes of secondary HTN are shown in (Table 2).

Certain clinical and biochemical features suggest the presence of a secondary cause for HTN and warrant further investigations. These include onset of HTN at a young age (< 40 years) or old age (> 65 years), severe or resistant HTN, associated symptoms or signs of possible secondary cause (e.g. bruits over the renal arteries, hypokalemia, and metabolic alkalosis). A renoparenchymal disease is usually recognized by the presence of high blood urea nitrogen and creatinine level and/or significant proteinuria. Renovascular HTN should be suspected in children or young women (fibromuscular dysplasia) or old men (atherosclerotic disease).

Magnetic resonance angiography is becoming a standard approach for the investigation of renal artery stenosis. Other diagnostic tests include captopril enhanced renal scanning, Doppler ultrasonography, and CT angiography.

Table 2: Causes of Secondary Hypertension

Renoparenchymal disease
Renovascular disease
Primary hyperaldosteronism
Cushing`s syndrome
Pheochromocytoma
Thyroid or parathyroid disease
Drug/Substance-Induced (oral contraceptives, NSAIDs, Steroids, liquorice, erythropoietin, cyclosporine, cocaine, amphetamine, excessive alcohol)
Coarctation of aorta
Obstructive sleep apnea

Hypertension Secondary to Reno-Parenchymal Diseases

Hypertension is a frequent finding in patients with CRD, about 70% of patients with CRD have HTN and the prevalence increases with the decrease of glomerular filtration rate. HTN is an important factor in the progression of kidney disease, and its pathogenesis is complex: sodium and fluid retention; over-activity of rennin-angiotensin system and sympathetic nervous system; arterial stiffness; increase intracellular calcium; loss of nocturnal decline in BP; and side effect of medications.

Endocrine Hypertension

Endocrine HTN is an uncommon cause of secondary HTN. Diagnosis of endocrine HTN is costly because appropriate investigations are required to establish the diagnosis. Suspicion of endocrine HTN can be obtained after complete and good history-taking with full physical examination.

The main causes of endocrine hypertension are:

- Oral contraceptives
- Primary hyperaldosteronism
- Cushing`s syndrome
- Pheochromocytoma

Other rare causes of endocrine HTN include thyrotoxicosis, hypothyroidism, hyperparathyroidism, acromegaly, congenital adrenal hyperplasia, Liddle`s syndrome, and apparent mineralocorticoid excess.

Five percent of women taking oral contraceptives develop HTN after five years of continuous use. Treatment of oral contraceptives induced HTN is by stopping the oral contraceptives.

The diagnosis of primary aldosteronism is highly suggested (after stopping diuretics, β -Bs, ACEIs) by low plasma rennin activity, high plasma aldosterone level, and high aldosterone/renin ratio.

Cushing syndrome is often suggested by the typical cushingoid appearance. HTN is present in up to 80% of these patients. Overnight dexamethasone suppression testing is a good screening test and significantly high 24-hour urinary cortisol excretion is diagnostic.

In pheochromocytoma, HTN is present in 90% of patients. Significantly high 24-hour urinary catecholamines or metanephrines excretion is diagnostic. Localization procedures include sonography, CT scan, Magnetic Resonance Imaging (MRI), and Meta-iodo-benzyl-Guanidine (MIBG) scan.

Hypertension and Obstructive Sleep Apnea

Obstructive Sleep Apnea (OSA) is relatively a common sleep disorder. This condition is characterized by repetitive obstruction of the upper airway during sleep associated with oxygen desaturation and interruption of sleep. The two cardinal symptoms of OSA are snoring and excessive daytime sleepiness. Around 50% of OSA patients have HTN and 25-30 % of patients with HTN have OSA. Treating OSA successfully might result into reduction in BP level; however, the data on the impact of treating OSA on HTN are quite limited. Older obese men and those with upper airway abnormalities are at high risk of developing OSA.

Risk Stratification in Hypertensive Patients

Cardiovascular disease risk is determined not only by BP levels but also by the presence or absence of cardiovascular risk factors, TOD, or ACCs. Also patient's personal, medical, and social situation merits consideration.

Factors influencing prognosis *

Risk Factors for Cardiovascular Diseases:

- Levels of SBP and DBP
- Men > 55 years
- Women > 65 years
- Smoking
- Obesity
- Dyslipidemia: (LDL-C more than 130 mg/dl i.e. 3.25 mmol/l and/or HDL less than 40 mg/dl. i.e. 1.0 mmol/l)
- DM **
- Family history of premature cardiovascular disease ***
- C-reactive protein \geq 1 mg/dl

Target Organ Damage:

- LV hypertrophy (ECG, echocardiogram, or chest X-ray)
- Proteinuria or elevated plasma creatinine (men 1.34-1.6 mg/dl i.e. 118-140 µmol/l, women 1.25-1.45 mg/dl i.e. 110-128 µmol/l)
- Ultrasound or radiological evidence of atherosclerotic plaque (aortic, carotid, iliac, or femoral)
- Generalized or focal narrowing of retinal arteries

Associated Clinical Conditions:

- Cerebrovascular disease: (ischemic stroke, cerebral hemorrhage, or TIA).
- Heart disease: (MI, angina, coronary revascularization, or CHF).
- Renal disease: (diabetic nephropathy or renal failure i.e. creatinine (men > 1.6 mg/dl i.e. 140 µmol/l, women > 1.45 mg/dl i.e. 128 µmol/l).
- Vascular disease: (dissecting aortic aneurysm or symptomatic arterial disease)
- Advanced hypertensive retinopathy: (hemorrhages, exudates, or papilledema).

Other Factors Adversely Influencing Prognosis but not used for Risk Stratification:

- Micro-albuminuria in diabetic patient
- Impaired glucose tolerance
- Sedentary lifestyle
- Raised fibrinogen
- High risk socio-economic group
- High risk ethnic group
- High risk geographic region

**Modified from: 1999 WHO/International Society of Hypertension Guidelines for the Management of Hypertension and 2003 European Society of Hypertension-European Society of Cardiology Guidelines for the Management of Arterial Hypertension.*

***Diabetes Mellitus is considered as coronary heart disease equivalent.*

**** First Degree relative; Male < 55 years or Female < 65 years.*

Stratification of hypertensive Patients by Absolute level of Cardiovascular Risk

Table 3: Stratification of Risk to Quantify Prognosis

Levels of BP Other Risk Factors and Disease History	SBP < 120 And DBP < 80 (mm Hg)	SBP 120 – 139 Or DBP 80 – 89 (mm Hg)	SBP 140-159 Or DBP 90-99 (mm Hg)	SBP 160-179 Or DBP 100-109 (mm Hg)	SBP ≥ 180 Or DBP ≥ 110 (mm Hg)
No Other Risk Factors.	Average Risk.	Average Risk.	Low Added Risk.	Moderate Added Risk.	High Added Risk.
1-2 Risk Factors except DM.	Low Added Risk.	Low Added Risk.	Moderate Added Risk.	Moderate Added Risk.	Very High Added Risk.
3 or more Risk Factors, TOD, or DM.	Moderate Added Risk.	High Added Risk.	High Added Risk.	High Added Risk.	Very High Added Risk.
ACCs.	High Added Risk.	Very High Added Risk.	Very High Added Risk.	Very High Added Risk.	Very high Added Risk.

Part III: Therapeutic Approaches

Management Strategy

The primary goal of treatment in hypertensive patients is to achieve the maximum reduction in the total risk of cardiovascular and renal morbidity and mortality. This requires identification and treatment of all reversible risk factors such as smoking, dyslipidemia, or DM and the appropriate management of associated co-morbidities, as well as treatment of the raised BP per se.

In general, the goal is to lower BP to a level below 140/90 mm Hg. In patients with DM or CRD, the goal is to lower BP below 130/80 mm Hg. These goals are achieved through lifestyle modification and, in most cases, drug therapy. Figures 1 and 2 provide algorithms for the treatment of HTN.

Non-Pharmacological Approach

A variety of dietary modifications have been shown to be beneficial in the management of HTN. These include: reduction of salt intake (<6 g sodium chloride/day), avoidance of alcohol, decrease in total and saturated fat consumption with increase in fruits and vegetables intake.

Non-dietary lifestyle modifications include weight reduction and regular physical activity (30-45 min, 3-4 times per week). Cessation of smoking reduces cardiovascular risk.

Herbal Remedies

It is suggested to exercise caution about recommending herbal remedies to treat HTN as the complexity of plant products are unproven and could even have a harmful effect. Also, dosing regimens are difficult to adjust and their intake might jeopardize the regular intake of prescribed medications.

The use of some spices to minimize salt intake can, however, be of benefit in helping reduce the amount of sodium in food.

It has not been proven that garlic or onions have any benefit in the treatment of HTN. However, literature has shown that garlic and olive oil reduce co-morbidity from cardiovascular diseases. In addition hypertensive patients should avoid licorice because it elevates BP.

Pharmacological Approach

All major classes of antihypertensive medications (Diuretics, β -Bs, CCBs, ACEIs and ARBs) are effective in lowering BP and, therefore, are suitable for initiation and maintenance of therapy. They differ, however, in many aspects including mechanism and

duration of action, efficacy, side-effect profile, and cost. The presence of concomitant disease may alter the choice of first line anti-hypertensive agents (see Tables 4 and 5).

Antiplatelet Therapy

Antiplatelet therapy especially in the form of low-dose Aspirin (100 mg daily) is beneficial in terms of reduction of cardiovascular morbidity and mortality when given to patients with high cardiovascular risk (primary prevention) or to patients with previous cardiovascular events (secondary prevention). Aspirin should be given only when BP control has been achieved.

For **primary prevention** low-dose Aspirin can be considered in the following situations:

- Hypertensive patients above the age of 50 years and at high or very high absolute cardiovascular risk, or
- Hypertensive patients with moderate increase in serum creatinine > 1.3 mg/dl i.e. > 114 μ mol/l.

For **secondary prevention** high-dose Aspirin should be given to:

- Patients with status post MI.
- Status post-ischemic stroke.
- Status post angioplasty, post coronary artery bypass graft.

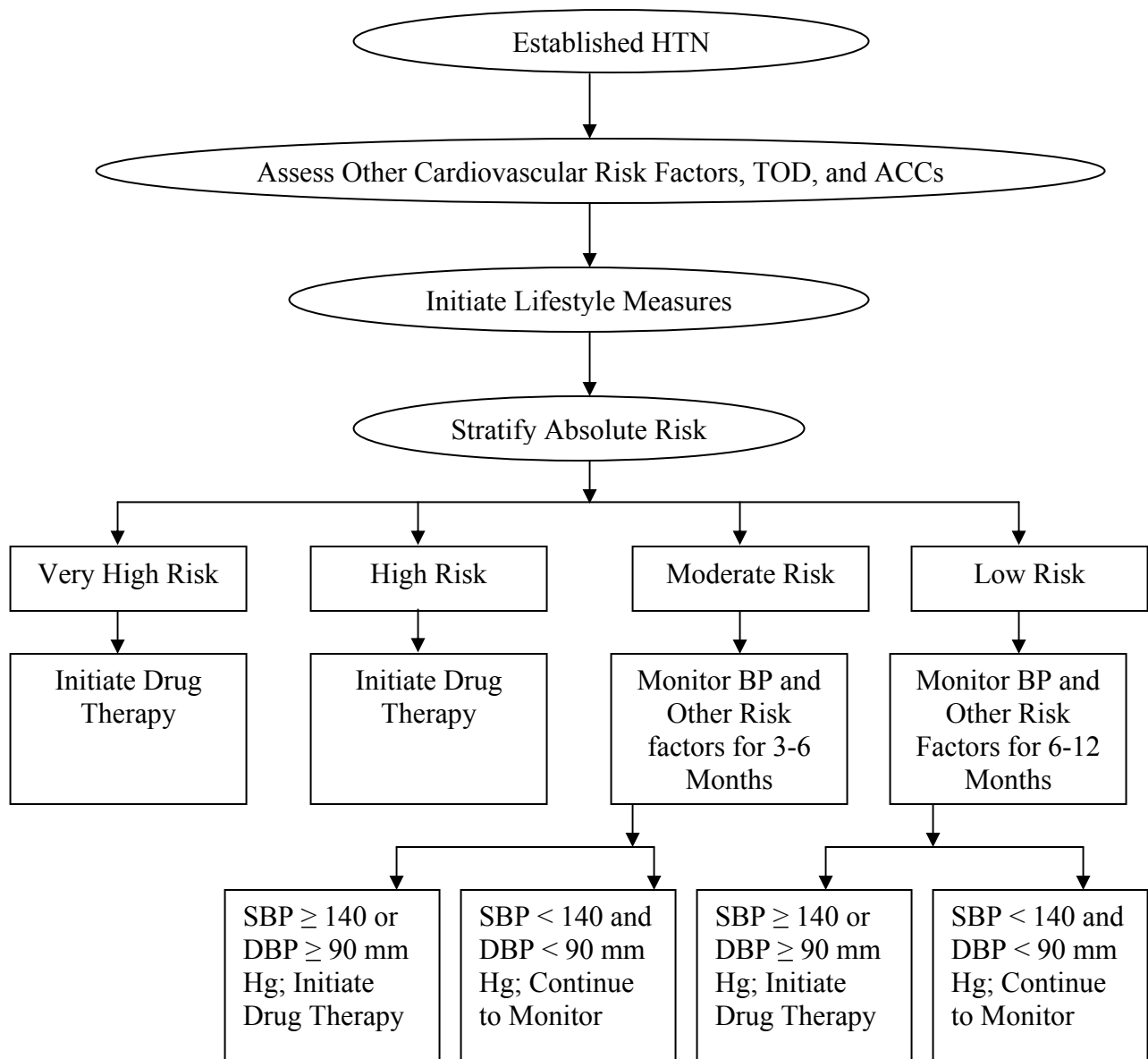


Figure 1: Initiation of Anti-Hypertensive Treatment

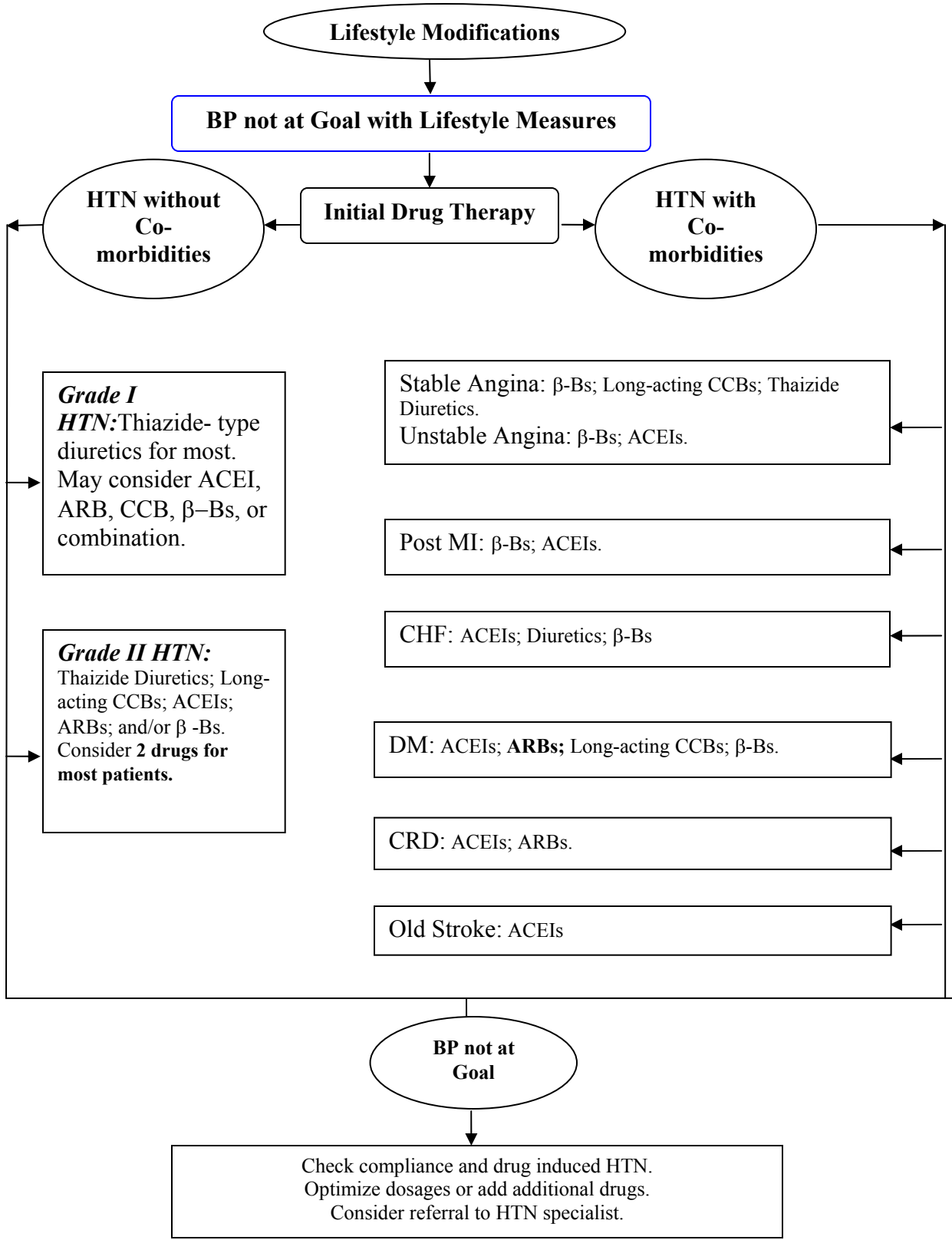


Figure 2: Algorithm for Treatment of Hypertension

Table 4: Anti-Hypertensive Medications – Indications and Contraindications*

Drug Class	Conditions favoring the use	Contraindications	
		Compelling	Possible
Thiazide Diuretics	CHF; Elderly hypertensives; DM; Osteoporosis; hypertensive patients of African origin;	Gout; Hyponatremia	Dyslipidemia; Sexually Active Males; Pregnancy
Loop Diuretics	Renal insufficiency; CHF		
Anti-aldosterone Diuretics	CHF; Post-MI;	Renal failure; Hyperkalaemia	
β -Bs	Angina pectoris; Post-MI; CHF; Pregnancy; DM; Migraine; Essential Tremors; Tachyarrhythmias; Thyrotoxicosis	Asthma; Chronic Obstructive Pulmonary Disease; Atrio-Ventricular block (grade 2 or 3)	Peripheral Vascular Disease; Glucose intolerance; Athletes and physically active patients; dyslipidemia.
Dihydropyridine CCB's	Elderly patients; Angina; Peripheral Vascular Disease; Carotid atherosclerosis; Pregnancy		Atrio-ventricular block (grade 2 or 3); CHF; Tachyarrhythmias.
Non-Dihydropyridine CCB's	Angina pectoris; Carotid atherosclerosis; Supraventricular tachycardia	CHF	
ACEIs	CHF; LV dysfunction; Post-MI; Non-diabetic nephropathy; Type 1 diabetic nephropathy; Proteinuria	Pregnancy; Hyperkalaemia; Bilateral renal artery stenosis Angioedema.	
ARBs	Type 2 diabetic nephropathy; Diabetic microalbuminuria; Proteinuria; LV hypertrophy; ACEIs induced cough	Pregnancy; Hyperkalaemia; Bilateral renal artery stenosis	
α -Bs	Benign Prostatic Hypertrophy; Dyslipidemia	Orthostatic hypotension	CHF

* Modified from 2003 European Society of Hypertension-European Society of Cardiology Guidelines for the Management of Arterial HTN

Table 5: Evidence-Based Antihypertensive Drug Treatment for Special Situations/Populations.

Health Problem / Disease	Target BP	Drugs to Use	Drugs to Avoid
Angina	<130/85	β -Bs (A), non-Dihydropyridine-CCBs, Long Acting DihydropyridineCCBs (B).	Short Acting Dihydropyridine CCBs (C).
Asthma	<140/90	Thiazide diuretics (B), Potassium sparing Diuretics (B).	β -Bs (A).
Atrial Fibrillation	<140/90	β -B (B), ARBs, non-Dihydropyridine-CCBs (B).	NSR.
CHF	<130/85	ACEIs (A), Diuretics (A), ARBs (A), Isosorbide Dinitrate with Hydralazine (A), Dihydropyridine-CCBs (A).	Non-Dihydropyridine-CCBs(A).
Conduction Defects	<140/90	NSR.	β -Bs, Dihydropyridine-CCBs.
Depression	<140/90	NSR.	β -Bs, Reserpine.
DM	<130/80	ACEIs (A), Cardioselective β -Bs (A), Low Dose-Diuretics (B), Long-Acting Dihydropyridine-CCBs (B), Indapamide (C), ARBs (C), α -Bs (C), Loop diuretic (C).	High Dose-Diuretics.
Dyslipidemia.	<140/90	Low Dose-Diuretics (B), ACEIs (B), β -Bs with Intrinsic Sympathomemtic Activity (B), α -Bs (D), ARBs (D), Dihydropyridine-CCBs (D).	β -Bs without Intrinsic Sympathomemtic Activity, High-dose Diuretics.
Elderly	<140/90	Low Dose-Diuretics (A), Long-Acting Dihydropyridine-CCBs (A), ACEIs (B), ARBs (D).	High Dose-Diuretics.
Gout	<140/90	NSR.	Thiazide diuretics (D), Loop diuretic.
Hyperthyroidism	<140/90	β -Bs.	NSR.
Liver disease	<140/90	NSR.	Labetalol, Methyldopa.
LV Hypertrophy	<140/90	ACEIs.	Hydralazine (C), Minoxidil (C).
MI	<130/85	β -Bs without Intrinsic Sympathomemtic Activity (A), ACEIs (A), Verapamil (A), Diltiazem (C).	Short Acting Dihydropyridine-CCB (C).
Migraine	<140/90	Non Cardioselective β -Bs, non-Dihydropyridine-CCBs.	NSR.
Osteoporosis	<140/90	Thiazide diuretics	NSR.
Preoperative	<140/90	β -Bs.	NSR.
Proteinuria >1gm/d	<125/75	ACEIs (A), Indapamide, Loop Diuretics, non-Dihydropyridine-CCBs (B).	NSR.
PVD	<140/90	CCBs (B), α -Bs (B).	β -Bs (B), ACEIs (B).
Renal disease	<130/80	ACEIs (A), Indapamide, Loop Diuretics, non-Dihydropyridine-CCBs (B).	NSR.
Smoking	<140/90	Thiazide diuretics, ACEIs.	β -Bs.
Stroke; old	<140/90	ACEIs(A), Dihydropyridine-CCBs (A).	NSR.
Stroke; recovery	<140/90	ACEIs.	NSR.

(A) Highly Recommended; (D) Least Recommended; NSR: No Specific Recommendations.

Part IV: Hypertension during Ramadan and Hajj

Hypertension and Ramadan Fasting

Some small-scale studies have looked at the effects of fasting on BP in hypertensive patients. One study looked at the changes in the cardiovascular system and its regulatory mechanisms in 150 patients with Grade II HTN. Iakovlev et al. observed a beneficial effect of fasting on BP. They hypothesized that this effect is due to the inhibition of the basic metabolism and the weakening of sympathetic influences on the myocardium and smooth muscle cells of the resistance vessels, with reduced contractility.

In another study with 11 moderately obese hypertensive women who fasted for 48 hours, SBP decreased from 158 to 146 mm Hg and DBP from 96 to 89 mm Hg. The differences were statistically significant.

Vertes and Hazelton studied the effects of weight reduction and fasting on BP in 99 morbidly obese hypertensive women. Reduction in BP was noted in 85 of these women by the end of one-week of in-hospital fasting.

Goldhamer et al. studied the effect of medically supervised fasting except for water on BP in 174 patients with HTN. The duration of fasting was 10 to 11 days. Almost 90% of the subjects achieved a BP less than 140/90 mm Hg. They concluded that medically supervised, water-only fasting appears to be a safe and effective means of normalizing BP.

The most relevant study in this regard with 99 hypertensive patients was carried out by Habbal et al. in Casablanca (Morocco) during Ramadan in 1998. All patients had ambulatory BP measurements before and during Ramadan. No statistically significant difference was noted between these two periods, either for SBP or DBP, for the all over 24-hour BP or for the diurnal and nocturnal periods. They concluded that in patients with uncomplicated essential HTN, Ramadan fasting was well tolerated. The variations of BP are minimal and are probably related to the changes in sleep, activity, and eating patterns. In addition, another study that was conducted in Jerusalem in 1998 looked at the effects of fasting on a small number of treated hypertensive patients. A 24-hour BP monitoring was carried out twice: before Ramadan and during the last week of the month. All patients continued their medications, which were all administered once daily. A 24-hour MBP as well as average awake and average sleep BP were compared. There was no difference between the MBP before or during Ramadan. The authors concluded that treated hypertensive patients might be assured that, with continuation of prescribed medications, fasting during Ramadan could be safely undertaken.

General Recommendations for Hypertensive Patients during Ramadan

Based on the scarce available data, the following recommendations can be reasonably made (expert opinion)

- Physician's advice and management should be individualized
- Patient education should emphasize the need to maintain compliance with non-pharmacological and pharmacological measures
- Diuretics are better avoided, especially in hot climates or to be administered in the early evening
- Patients are encouraged to seek medical advice before fasting in order to adjust their medications if needed
- A once daily dosage schedule with long acting preparations is recommended.
- Patients with HTN should be advised to take a low salt, low fat diet
- Patients with difficult to control HTN, should be advised not to fast until their BP is reasonably controlled
- Patients with hypertensive emergencies should be treated appropriately regardless of fasting

Hypertension and Hajj (Pilgrimage)

Based on the scarce available data, the following recommendations can be reasonably made:

- Hypertensive Pilgrims should have a medical check-up before they leave home for Hajj, especially the elderly and those with other co-morbidities.
- Patients with severe HTN should be considered unfit for a long journey such as Hajj.
- Once daily medication regimens are preferable.
- Due to the hot climate in the Makkah region and the possibility of dehydration, diuretics are better avoided (unless indicated for other reasons).
- To keep the BP under control, patients should take their BP medications as directed. Patients should check their BP regularly and try to reduce stress during the Hajj.

Part V: Hypertension and Co-Morbidities

Hypertension and Diabetes Mellitus

Approximately 60% to 80% of diabetic patients will develop HTN. Strict control of BP in these patients is as important as the control of blood sugar. Studies have shown that BP above 130/80 mm Hg is associated with significant risk for microvascular and macrovascular complications. In general, BP control is probably more important than the drug used. Thiazide diuretics, ACEIs, ARBs, β -Bs and CCBs have all been shown to be effective agents in diabetic patients to reduce microvascular and macrovascular complications. ACEIs and ARBs are particularly effective when there is diabetic nephropathy, as evidenced by microalbuminuria or proteinuria.

- The goal BP is < 130/80 mm Hg in diabetic patients
- In a patient with no evidence of microvascular or macrovascular damage, there is no clear benefit of one drug over the other
- In patients with microalbuminuria or proteinuria ACEIs or ARBs provide the best renoprotection
- Multiple drugs are often needed to achieve the target BP

Hypertension and Chronic Renal Disease

Treatment of HTN can slow the progression of CRD. In addition to sodium and fluid restriction, all antihypertensive medications can be used to lower BP in CRD, and usually multiple medications are required. Loop diuretics in a good dose help to control volume overload. Some physicians are reluctant to administer ACEIs in spite of their proven efficacy in preventing progression of CRD for the initial rise of serum creatinine. ACEIs are the preferred medications, particularly with proteinuria, as they reduce proteinuria by 30 to 50%, prevent progression of CRD, and reduce cardiovascular morbidity and mortality as evidenced by several randomized controlled trials.

Recommendations:

- Strict control of BP (< 130/80 mm Hg) should be achieved in all patients with CRD and < 125/75 mm Hg in patients with proteinuria (> 1 gm/day).
- Decrease proteinuria < 60% of the baseline with lowest achievable level is targeted, preferably with ACEIs and/or ARBs.
- Monitor renal function and potassium level within one week after initiation of therapy.
- Titrate-up with small doses gradually to maximum dose level to achieve reduction in proteinuria and control of BP.
- If that is not achieved, additional therapy with diuretics or non-dihydropyridine CCBs could be used.
- Labetolol is the agent of choice for pregnant women with CRD.
- Additionally, control of dyslipidemia, cessation of smoking, and moderate protein restriction should be followed to decrease the rate of progression of CRD.

Hypertension and Coronary Heart Disease

The risk of recurrent cardiovascular events in patients with CHD has a direct relationship to BP level, but excessive and rapid lowering of BP should be avoided especially if associated with reflex tachycardia.

Hypertension and Angina:

First choice: β -Bs.

Second choice: Long-acting CCBs.

HTN and Recent MI:

- β -Bs preferably without intrinsic sympathomimetic activity and/or ACEIs.
- Diltiazem or Verpamil could be used if β -Bs are contraindicated especially in non-Q MI and normal LV systolic function.
- There is a trend towards using ACEIs or ARBs in all patients with CHD even without HTN and normal LV systolic function.
- Aldosterone Antagonists in small dose proved to be useful even without HTN.

Hypertension and Dyslipidemia

In hypertensive patients, elevated total cholesterol and LDL-C are major risk factors for cardiovascular events.

Lifestyle modification is the first approach for management of HTN and dyslipidemia.

In hypertensive patients, the presence of any of the following factors is an indication for lowering LDL-C to below 130 mg/dl i.e. 3.3 mmol/l:

- men over 45 years
- women over 55 years
- positive family history of premature cardiovascular disease
- smoking
- HDL-C < 40 mg/dl (<1 mmol/l)

Hypertensive patients with history of CHD or DM irrespective of the presence or absence of other risk factors should have their LDL-C level reduced to < 100 mg/dl (<2.6 mmol/l). This last recommendation is based on the finding that these patients have a risk of MI 20 times more than those without CHD.

Effect of antihypertensive medications on lipid profile:

- **Neutral effects:** ACEIs, ARBs, CCBs and Central Adrenergic Agonists
- **Beneficial effects:** α -Blockers.
- **Transient adverse effect:** Hydrochlorothiazide or β -Bs especially in large doses.

The choice of antihypertensive therapy should not be significantly affected by consideration of patient's lipid profile as the beneficial effects of these drugs outweigh their minimal transient adverse effects.

Hypertension and Congestive Heart Failure

Congestive Heart Failure is five times more common in hypertensive patients as compared to normotensive persons. LV dysfunction is related to the level of BP even in the normal range.

Treatment of choice:

- ACEIs and diuretics
- Combined α - and β -Bs (Carvedilol), and β -Bs (Metoprolol or Bisoprolol) have been proved to be useful in the whole spectrum of LV systolic dysfunction

Alternative treatment:

- Isosorbide dinitrate + Hydralazine: if ACEIs are contraindicated
- ARBs are alternatives to ACEIs if not tolerated especially in elderly
- Aldosterone antagonists are useful in severe CHF
- Long-acting dihydropyridin CCBs may be added to control hypertension

Hypertension and Cerebrovascular Disease

Reduction of BP in hypertensive patients is effective in primary and secondary prevention of stroke.

Treatment of HTN in the acute stroke settings:

- i) **In acute ischemic stroke:** There is a general agreement that these patients don't need fast aggressive lowering of BP.

Early use of antihypertensive medications is warranted when:

- Mean BP is more than 130 mm Hg
- DBP is more than 120 mm Hg
- SBP more than 200 mm Hg

SBP should be < 180 and DBP should be < 100 if thrombolytic therapy is to be initiated. The best agent to be used is labetalol starting with a bolus of 10 mg over 1-2 min. followed by an infusion of 2-8 mg/min. till the desired BP is achieved.

- BP should be lowered if Hypertensive encephalopathy is suspected.
- The use of sublingual BP lowering agent nifedipine is contraindicated.

- ii) **In hemorrhagic stroke:** BP should also be lowered with caution. In patients with known HTN, a SBP ≤ 180 or a DBP ≤ 105 mm Hg can be tolerated. Mean arterial BP should be lowered by 20-25%, especially in the case of subarachnoid hemorrhage.

Obese patients

Obesity is a risk factor for HTN and other cardiovascular diseases. As Body Mass Index (BMI) increases, so does the risk of HTN. It is important to assess BMI and waist circumference in each individual. Using BMI, patients can be classified as normal weight (BMI 18.5-24.9 kg/m²), overweight (25-29.9 kg/m²) or obese (≥ 30 kg/m²). For those obese patients a weight management plan should be constructed and discussed with the patient. Options available include lifestyle modification (including behaviour therapy), pharmacotherapy, and bariatric surgery.

Metabolic Syndrome

Criteria for diagnosis; 3 or more of the following;

1. Waist circumference of > 80 cm in women or > 94 cm in men. Less in certain populations; South-East Asian ethnicity, family history of DM where > 80 cm in women and > 90 cm in men.
2. Elevated Triglycerides; 150 mg (1.7 mmol/l) or more, or receiving drug treatment for triglycerides.
3. An HDL-C < 50 mg/dl (1.25 mmol/l) in women, or < 40 mg/dl (1 mmol/l) in men or receiving drug treatment for low HDL-C.
4. A blood pressure level of > 130 mm Hg systolic or > 85 diastolic or receiving anti-hypertensive therapy.
5. A fasting blood sugar > 100 mg/dl (> 5.6 mmol/l) or more or receiving drug treatment for raised glucose.

Part VI: Hypertension in Special Populations

Hypertension in Children

BP increases gradually with age and height, therefore, standard nomograms are necessary for interpretation of BP in children. Most children track in a constant percentile around the mean. (See percentiles charts Appendix III).

Secondary HTN is more common in younger children while essential HTN is more common in older children and adolescents.

Most hypertensive patients are asymptomatic or have non-specific symptoms; measurement of BP with appropriate cuff should be part of the routine pediatric clinical evaluation.

The primary investigation for hypertensive children should include: CBC, urinalysis, urine culture, blood urea nitrogen, creatinine, electrolytes, lipid profile, ECG, chest X-ray, echocardiogram, as well as renal ultrasound and Doppler study.

Treatment should be guided by the following:

- Transient or persistent HTN.
- Cure of the secondary causes might cure or eliminate HTN.
- Non-pharmacological measures should be tried.
- Selection of the antihypertensive agent should take into consideration the possible pathophysiology.
- In children, dose adjustment of antihypertensive drugs is imperative.
- Severe HTN (BP > 99th percentile for age, gender, and height) or malignant HTN (marked HTN with retinal hemorrhage, exudate, papilledema, seizure with or without renal involvement) should be treated immediately and the treatment should go hand in hand with the investigations.

In general, hypertensive children should be referred to a specialized pediatrician.

Hypertension in Women

i) Pregnancy:

There are four types of HTN in pregnancy:

1. Pre-existing HTN: Diagnosed before pregnancy or before the 20th week of gestation and persists post-delivery.
2. Gestational HTN: HTN occurs for the first time in the second half of pregnancy without proteinuria and normalizes by 12 weeks post-partum.
3. Pre-eclampsia: Two BP readings of > 140/90 mm Hg, (six-hours apart) with proteinuria.

The following indicate severe disease which warrant hospitalization and consideration of urgent delivery: SBP > 160 or DPB > 110 mm Hg, 24 hours urine protein \geq 2g (2+ or 3+ on qualitative examination), serum creatinine >

1.2 mg/dl, platelets $<100,000$ cells/mm³, evidence of microangiopathic hemolytic anemia, elevated hepatic enzymes, persistent headache or other cerebral or visual disturbances, persistent epigastric pain, convulsions (eclampsia).

4. Pre-eclampsia superimposed on chronic HTN.

Treatment:

1. **Non-pharmacological therapy:** Bed-rest without salt-restriction.
2. **Pharmacological therapy:**
 - Methyldopa and Labetalol are drugs of choice.
 - Labetalol is the agent of choice for pregnant women with CRD.
 - Other drugs which could be used include other β -Bs, CCBs and Hydralazine.
 - Diuretics use is controversial but should not be used in pre-eclampsia.
 - During breast feeding, drugs for mild pre-eclampsia could be withheld with close monitoring of blood pressure. Methyldopa and Hydralazine are safe.
 - ACEIs and ARBs are contraindicated in pregnancy and breast feeding.
 - Severe hypertension: IV Hydralazine, IV labetalol, or oral Nifedipine.
 - Sodium Nitroprusside can be used in rare cases not responding to the above mentioned medications.

ii) Women using Hormone Replacement Therapy:

Hormone replacement therapy has the potential to worsen BP in hypertensive women. It should be withheld in women with resistant HTN in order to assess its contribution to the increase in BP. It is advisable to monitor BP two to three times in the first six months after starting hormone replacement therapy, then twice a year thereafter. Recent recommendations advise against the use of hormone replacement therapy for cardiovascular protection in menopausal females.

Hypertension in the Elderly

The definition of HTN in the general adult population applies to the elderly (Age > 65 years). Among older persons, elevation in SBP or increase pulse-pressure is a better predictor of cardiovascular morbidity and mortality than elevated DBP. Primary HTN is by far the most common form of HTN in older persons. However, in the case of clinical suspicion (or when the onset of HTN occurs at old age) a secondary cause should be sought. Renal parenchymal disease is the most common secondary cause to be considered, followed by renal artery stenosis. The later is suspected in any patient with

resistant HTN and known atherosclerosis in other arteries. The goal of treatment in older patients with no co-morbidity should be the same as in the general population, i.e. < 140/90 mm Hg.

Special notes:

- Pseudo-HTN, orthostatic hypotension even without treatment, or white coat HTN especially among elderly women is more common than in the young patient.
- Sodium reduction is especially effective in the elderly because of their greater sensitivity to sodium intake.
- The starting dose of medications in older patients should be about half of that used in younger patients: “Start low and go slow”.
- Medications with once daily dosage are preferred for better compliance and in order to keep the drug regimen as simple as possible.
- Low-dose thiazide therapy (12.5 - 25 mg of hydrochlorothiazide or equivalent) can be prescribed as the first-line treatment of HTN.
- Long acting CCBs are second choice
- β -Bs are less appropriate as first line therapy for HTN in the elderly.
- Drugs that exaggerate postural hypotension (α -Bs, high dose diuretics) or drugs that can cause cognitive dysfunction (central α -2 agonists) should be used with great caution.
- Presence of other co-morbidities dictates the choice of the first line drugs.

Minority populations

Socio-economic factors and lifestyle may influence BP control in some minority patients. However, there are no studies published that address BP control in these populations in Saudi Arabia. American studies have indicated that prevalence, severity, and impact of HTN are increased in Blacks, who also demonstrate somewhat reduced BP responses to monotherapy with β -Bs, ACEIs, or ARBs compared with diuretics or CCBs. Three major clinical trials suggest that CCBs are most effective in Black people. South-East Asian patients tend to consume large amount of sodium monoglutamate salt that may interfere with BP control.

Resistant Hypertension

Resistant HTN is defined as failure to achieve adequate BP control despite life style measures and drug regimen with three or more anti hypertensive medications near or at maximum doses. One of these medications has to be a diuretic. Common causes of resistant HTN include:

- Suboptimal therapy
- Extracellular volume expansion
- Poor compliance with medical or dietary therapy
- Secondary HTN
- White Coat HTN
- Pseudohypertension in the elderly
- Ingestion of substances that can elevate BP

Part VII: Hypertensive Urgencies and Emergencies

Hypertensive urgencies are those conditions with critically elevated BP (SBP \geq 200, DBP \geq 120 mm Hg) without evidence of acute TOD. In hypertensive urgencies critically elevated BP should be lowered gradually within 24 hours.

Hypertensive emergencies are those conditions with critically elevated BP (SBP \geq 200, and/or DBP \geq 120 mm Hg) with evidence of acute TOD or one of the following conditions: pheochromocytoma, vasculitis and clonidine withdrawal, head trauma and life threatening arterial bleeding, etc.

In hypertensive emergencies critically elevated BP should be lowered rapidly (within 15-30 minutes) to avoid and limit the risk of serious complications, but controlled (reduction of MBP by 25%, aim DBP 100-110, SBP 160 mm Hg) to avoid sudden drop of BP and reduction of perfusion to vital organs (brain, heart).

Initial medical treatment depends on the clinical presentation as shown in the Table 6. Patients with hypertensive emergencies require immediate hospitalization for further management.

Table 6: Initial Medical Treatment for Hypertensive Emergencies

Clinical Presentation	Initial Treatment
Acute Myocardial Infarction	Nitroglycerine 5 mg sublingual, Nitroglycerine infusion
Acute Renal Failure	Furosemide 40-80 mg IV
Clonidine Withdrawal	Clonidine 0.1 mg IV slowly
Dissecting Aortic Aneurysm	Nitroprusside 0.25-8 μ g/kg/min + Esmolol 100 μ g/kg/min
Eclampsia	Hydralazine 10 mg IV slowly
Hypertensive Encephalopathy, Stroke, Vasculitis	Captopril 25 mg orally, Nifedipine 10 mg orally, Clonidine 0.1mg orally
Phaeochromocytoma	Phenoxybenzamine 10 mg oral, Phentolamine 2-5 mg IV
Pulmonary Edema	Furosemide 40-80 mg IV
<i>Pediat.</i> : Hypertensive encephalopathy	Labetalol 1-3 mg IV (adjust according to response) Nitroprusside 0.5-8 μ g/kg/min IV infusion Diazoxide 1-3 mg/kg IV
<i>Pediat.</i> : Sudden and severe hypertension	Labetalol 1-3 mg IV Nitroprusside 0.5-8 μ g/kg/min IV infusion Nifedipin 0.25-0.5 mg/kg P.O.

Table 7: Recommended Antihypertensive Drugs for Hypertensive Emergencies

Clinical Condition	Preferred Treatment	Dosages of I.V. Antihypertensive Medications
Acute Pulmonary Edema	Furosemide 40 mg I.V.	<p>Enalaprilat; I.V.; 1.25 mg over 5 min every 6 h, titrated by increments of 1.25 mg at 12-24 h intervals to a maximum of 5 mg every 6 h.</p> <p>Esmolol; Loading dose of 500 mg/kg over 1 min, followed by an infusion at 25 to 50 mg/kg/min, which may be increased by 25 mg/kg/min every 10 to 20 min until the desired response to a maximum of 300 mg/kg/min.</p> <p>Fenoldopam; initial dose of 0.1 mg/kg/min, titrated by increments of 0.05 to 0.1 mg/kg/min to a maximum of 1.6 mg/kg/min.</p> <p>Hydralazine; may be administered in doses of 10 to 20 mg.</p> <p>Labetalol; Initial bolus 20 mg, followed by boluses of 20 to 80 mg or an infusion starting at 2 mg/min; maximum cumulative dose of 300 mg over 24 h.</p> <p>Nicardipine; 5 mg/h; titrate to effect by increasing 2.5 mg/h every 5 min to a maximum of 15 mg/h.</p> <p>Nitroglycerin (up to 200 µg /min)</p> <p>Nitroprusside; 0.5 mg/kg/min; titrate as tolerated to maximum of 2 mg/kg/min.</p> <p>Phentolamine; 1-5 mg boluses; maximum dose, 15 mg.</p> <p>Trimethaphan; 0.5 to 1 mg/min; titrate by increasing by 0.5 mg/min as tolerated; maximum dose, 15 mg/min.</p>
Acute Myocardial Ischemia	Labetalol or Esmolol in combination with Nitroglycerin (up to 200 µg /min). Nicardipine may be added if pressure is controlled poorly with Labetalol/Esmolol alone.	
Hypertensive Encephalopathy	Labetalol or Nicardipine	
Acute Aortic Dissection	Labetalol or combination of Nitroprusside and Esmolol.	
Eclampsia	Hydralazine (traditional). In the ICU, Labetalol or Nicardipine is preferred.	
Acute Renal Failure	Nicardipine or Fenoldopam	
Microangiopathic Hemolytic Anemia	Nicardipine or Fenoldopam	
Pheochromocytoma	Phentolamine I.V. followed by oral Phenoxybenzamine	
Clonidine withdrawal	Oral Cloindine (0.1 mg every 20 min)	

Part VIII: Specialist Referral

Specialist referral is indicated if there is a possible underlying cause or presenting as:

- sudden onset
- worsening of hypertension
- resistance to multi-drug regimen three or more drugs
- Hypertension diagnosed in young age (< 35 years)
- persistent noncompliance

Appendices

Appendix I: Anti-hypertensive Drugs Available in Saudi Arabia (June 2004):

Oral Antihypertensive Drugs Available in Saudi Arabia			
Generic Name	Brand Name & Package Size & Price in Riyals	Usual Dose Range, mg/d	Daily Frequency
DRUG CLASS: THIAZIDE DIURETICS			
Chlorthalidone	Hygroton, 50mg. (20 tablet=12.3 SR)	12.5-50	1
Hydrochlorothiazide	ESIDREX 25 MG (20 tablet= 11.85 SR) MONOZIDE 25MG (30 tablet= 13.8 SR) MONOZIDE 12.5MG (30 tablet= 8.15 SR) MODURETIC-50MG (30 tablet= 17.3 SR)	12.5-50	1
Indapamide	Natrillix 2.5 mg (30 tablet=24.2 SR) Natrillix 1.5 mg S.R. (30 tablet=27.05 SR)	1.25-2.5	1
DRUG CLASS: LOOP DIURETICS			
Bumetanide	BURINEX TABLETS 1 MG (20 tablet=6.7 SR)	0.5-2	2
Furosemide	DIUSEMIDE 40 MG (30 tablet= 6.8 SR) IMPUGAN 40 MG (24 tablet= 5.05 SR) LASIX 40 MG. (20 tablet = 15.55 SR) OEDEMAX 40 MG. (10 tablet=3.75 SR & 50 tablet=18.65 SR)	20-80	2
DRUG CLASS: POTASSIUM-SPARING DIURETICS			
Amiloride	Available in combination products	5-10	1-2
Triamterene	Available in combination products	50-100	1-2
DRUG CLASS: ALDESTERONE-RECEPTOR BLOCKERS DIURETICS			
Spironolactone	Aldactone 25 MG (20 tablet= 12.7 SR) Aldactone 100 MG (10 tablet= 23.75 SR) Noractone 25 MG (30 tablet= 8.8 SR)	25-50	1-2
DRUG CLASS: β-Bs			
Atenolol	Apo-Atenolol 50 mg.(30 tablets=16.75 SR ,100 tablets=47.95 SR) Apo-Atenolol 100 mg(30 tablets=29.05 SR ,100 tablets=83.35SR). Bolkium 100 mg.(15 tablets=13.15.95 SR). Canar 50 mg.(28 tablets=18 SR) Canar 100 mg.(28 tablets=28.8 SR) Cardol 100 mg.(20 tablets=28.3 SR) Hypoten 50 mg.(28 tablets=21.8 SR) Hypoten 100 mg.(14 tablets=21.8 SR) Normoten 50 mg (28 tablets=20 SR) Normoten 100 mg (14 tablets=16.4 SR) Normoten 100 mg (28 tablets=32 SR) Novo-Atenolol 50 mg.(30 tablets=18.45 SR ,100 tablets=52.9 SR) Tensoten 50 mg (30 tablets=16.2 SR)	25-100	1
Bisoprolol	Concor 5 mg. (30 tablets=24.95 SR) Concor 10 mg.(30 tablets=35.3 SR) Concor-5-Plus (Bisoprolol 5mg+Hydrochlorothiazide5mg.) (20 tablets=21.65 SR)	2.5-10	1

Oral Antihypertensive Drugs Available in Saudi Arabia			
Generic Name	Brand Name & Package Size & Price in Riyals	Usual Dose Range, mg/d	Daily Frequency
Metoprolol	Lopressor 50 mg.(40 tablets=24.35 SR) Lopressor 100 mg.(20 tablets=24.35 SR) Lopressor 200 mg.(14 tablets=31 SR)	50-100	1-2
Propranolol	Inderal 10 mg.(50 tablets=4.45 SR) Inderal 40 mg.(50 tablets=11.15 SR) Inderal 80 mg.(100 tablets=42.5 SR) Indicardin 10 mg.(50 tablets=3.45 SR) Indicardin 40 mg.(50 tablets=7.10 SR)	40-160	2
DRUG CLASS: β-Bs WITH INTRINSIC SYMPATHOMIMETIC ACTIVITY			
Pindolol	Visken 5mg.(30 tablets=19.6 SR) Viskaldix=Pindolol 10mg+Clopamide 5 mg)(30 tablets=26.45 SR)	10-40	2
DRUG CLASS: COMBINED α- and β-Bs			
Carvedilol	Dilatrend 25 mg.(30 tablets=51.85 SR) Dilatrend 6.25 mg.(30 tablets=19.45 SR)	12.5-50	2
Labetalol	Trandate 100 mg,(25 tablets=13.05 SR, 100 tablets=44.65 SR) Trandate 200 mg.(25 tablets=18.25 SR, 100 tablets=66.55)	200-800	2
DRUG CLASS: ACEIs			
Benazepril	Cibacen 5 mg (14 tablets=19.6 SR) Cibacen 10 mg (14 tablets=33.25 SR) Cibacen 20 mg (14 tablets=54.5 SR)	10-40	1-2
Captopril	Capocard 25 mg(20 tablets=18.45 SR) Capocadr 50 mg(20 tablets=30.85 SR) Capoten 25 mg(20 tablets=26.15 SR) Capoten 50 mg(20 tablets=44.2 SR) Capril 25 mg(20 tablets=19.6 SR) Capril 50 mg(20 tablets=33.15 SR) Miniten 25 mg(20 tablets=15.3 SR) Miniten 50 mg(20 tablets=25.1 SR) Novo-Captopril 12.5 mg.(20 tablets=9.6 SR) Novo-Captopril 25 mg.(20 tablets=16.25 SR) Novo-Captopril 50 mg.(20 tablets=26.6 SR) Capozide 50+5 mg(28 tablets=62.1 SR)	25-100	2
Enalapril	Angiotec 5 mg (30 tablets=29.05 SR) Angiotec 10 mg(30 tablets=39.55 SR) Angiotec 20 mg(30 tablets=47.55 SR) Enapril 5 mg (30 tablets=25.55 SR) Enapril 10 mg (30 tablets=43.3 SR) Esopress 10 mg (30 tablets=48.1 SR) Narapril 5 mg (28 tablets=19.3 SR) Narapril 10 mg (28 tablets=32.7 SR) Narapril 20 mg (28 tablets=43.15SR) Renitec 5 mg (28 tablets=47.1 SR) Renitec 10 mg(28 tablets=66.05 SR) Renitec 20 mg(14 tablets=43.15 SR & 28 tablets=78.45 SR) Riapril 5mg (30 tablets=23 SR) Riapril 10mg (30 tablets=38.95 SR) Riapril 20mg (30 tablets=51.4 SR)	2.5-40	1-2

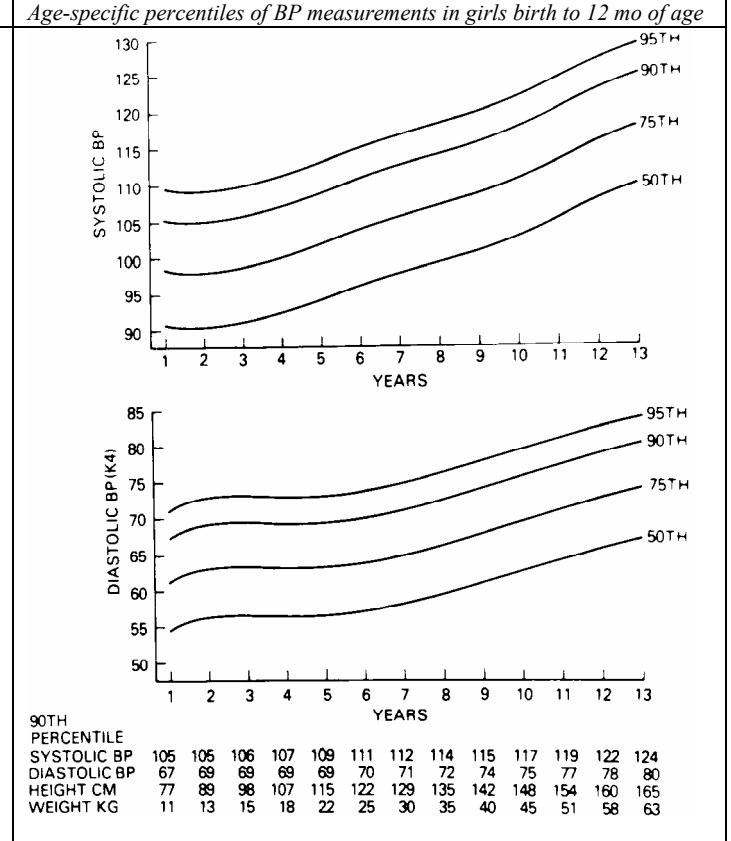
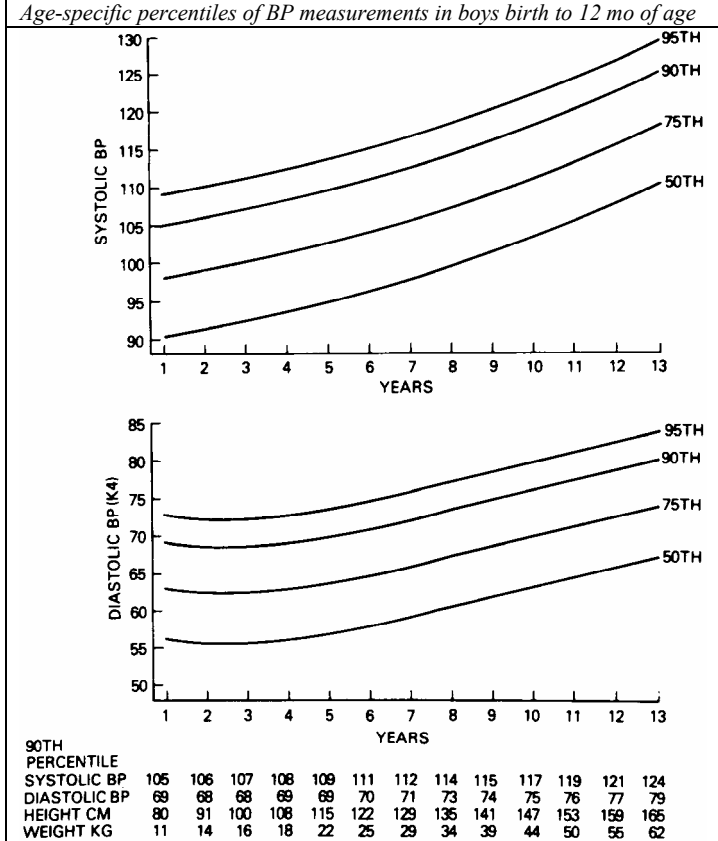
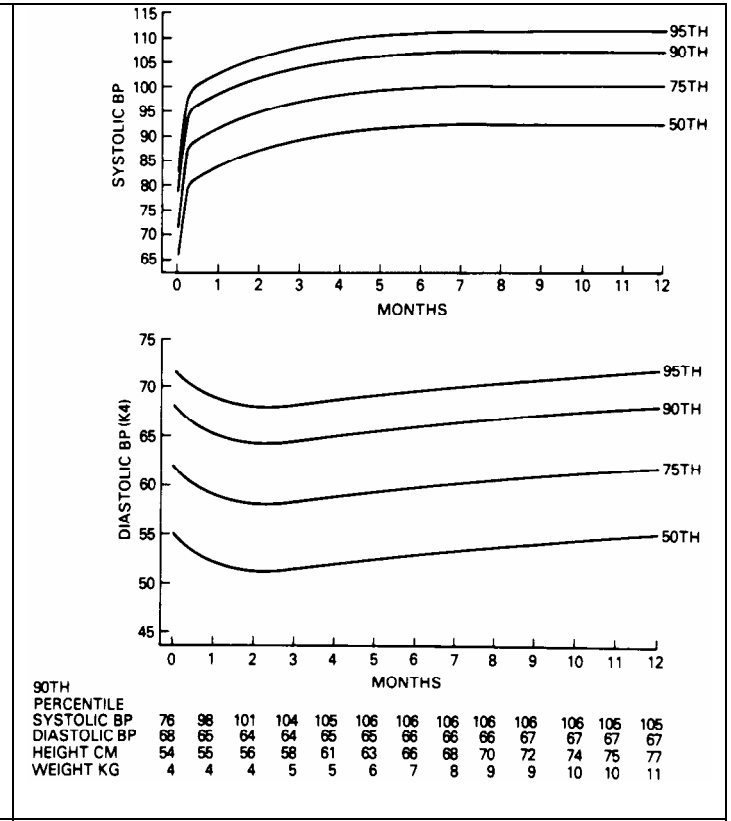
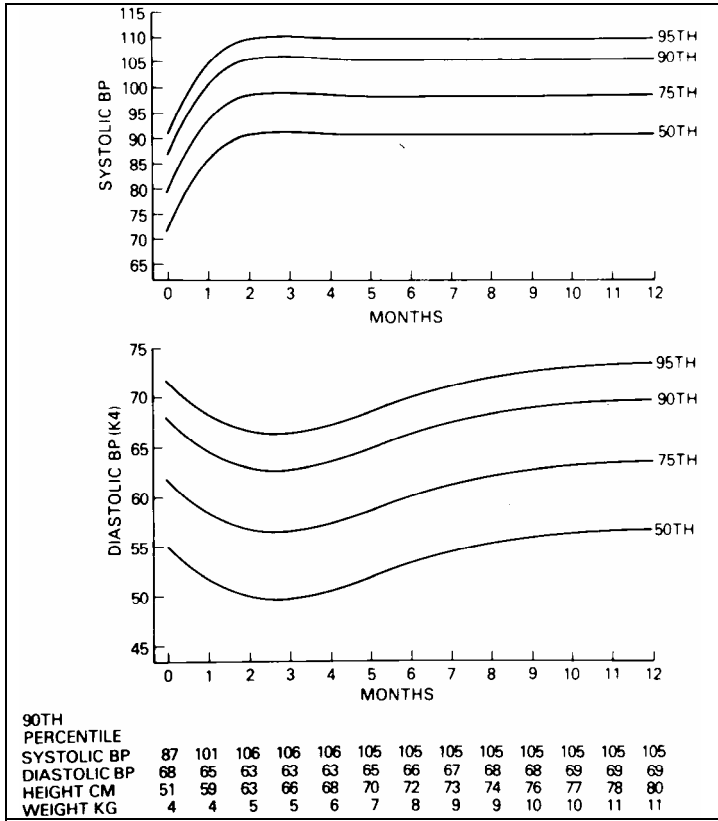
Oral Antihypertensive Drugs Available in Saudi Arabia			
Generic Name	Brand Name & Package Size & Price in Riyals	Usual Dose Range, mg/d	Daily Frequency
Fosinopril	Staril 10 mg (30 tablets=85.95 SR) Staril 20 mg(30 tablets=129.3 SR)	10-40	1
Lisinopril	Zestril 5 mg(28 tablets=29.8 SR) Zestril 10 mg(28 tablets=71.8 SR) Zestril 20 mg(28 tablets=90.2 SR) Zinopril 5 mg (28 tablets=20.25 SR) Zinopril 10 mg (28 tablets=48.85 SR) Zinopril 20 mg (28 tablets=61.3 SR) Zinopril 40 mg (28 tablets=100.55 SR)	10-40	1
Perindopril	Coversyl 2 mg.(30 tablets=25.9 SR) Coversyl 4 mg.(30 tablets=45.95 SR)	4-8	1-2
Quinapril	Acuitel 10 mg.(30 tablets=38.15 SR) Acuitel 20 mg.(30 tablets=58.85 SR)	10-40	1
DRUG CLASS: ARBs			
Candesartan	Atacand 4 mg(28 tablets=50.5 SR) Atacand 8 mg(28 tablets=60.45SR) Atacand 16mg(28 tablets=73.8 SR) Blopess 8 mg(28 tablets=54.4 SR) Blopess 16 mg(28 tablets=66.4 SR)	8-32	1
Eprosartan	Teveten 600mg (28 tablets=93.05 SR)	400-800	1-2
Losartan	Cozaar 50 mg.(28 tablets=79.25 SR)	25-100	1-2
Irbesartan	Aprovel 150 mg (28 tablets=78.10 SR) Aprovel 300 mg (28 tablets=95.9 SR)	150-300	1
Telmisartan	Micardis 40 mg.(28 tablets=67.75 SR) Micardis 80 mg.(28 tablets=88.4 SR)	20-80	1
Valsartan	Diovan 80 mg.(28 capsules=76.55 SR) Diovan 160 mg.(28 capsules=94.6 SR)	80-320	1
DRUG CLASS: CCBs-NON-DIHYDROPYRIDINES			
Diltiazem	Apo-Diltaz 30 mg(30 tablets=9.5 SR) Apo-Diltaz 60 mg(30 tablets=14.85 SR) Bi-tildiem 90 mg(28 tablets=22.9 SR) Bi-tildiem 120 mg(28 tablets=29 SR) Dilzem 90 mg(30 tablets=24.25 SR) Dilzem 60 mg(30 tablets=19.05 SR) Mono-Tildiem SR 200 mg (28 tablets=41.75 SR) Mono-Tildiem SR 300 mg (28 tablets=58 SR) Novo-Diltazem 30 mg.(30 tablets=10.7 SR &100 tablets=30.7 SR) Novo-Diltazem 60 mg.(30 tablets=16.65 SR &100 tablets=47.8 SR)	120-420	1
Verapamil	Isoptin 40 mg.(50 tablets=18.65 SR) Isoptin 80 mg.(20 tablets=14.3 SR) Isoptin –Retard 120 mg.(20 tablets=20.8 SR) Isoptin-SR 240 mg.(20 tablets=30.65 SR)	80-320	1-2
DRUG CLASS: CCBs-DIHYDROPYRIDINES			
Amlodipine	Amlor 5 mg.(25 tablets=65 SR) Amvasc 2.5 mg (30 tablets=27.6 SR) Amvasc 5 mg (30 tablets=46.8 SR) Amvasc 10 mg (30 tablets=76.75 SR)	2.5-10	1
Felodipine	Plendil SR 5 mg.(30 tablets=43.45 SR) Plendil SR 10 mg.(30 tablets=58.45 SR)	2.5-20	1

Oral Antihypertensive Drugs Available in Saudi Arabia			
Generic Name	Brand Name & Package Size & Price in Riyals	Usual Dose Range, mg/d	Daily Frequency
Isradipine	Lomir 2.5 mg(28 tablets=40.2 SR &56 tablets=80.45 SR) Lomir SR 5mg.(30 capsules=70.5 SR)	2.5-10	2
Nifedipine	Adalat-Retard 20 mg(30 tablets=40.45 SR) Adalat-LA 30mg.(30tablets=42.4 SR) Adalat-LA 60mg.(30tablets=53.5 SR)	30-60	1
DRUG CLASS: ADRENERGIC INHIBITORS: α-Bs			
Doxazosin	Cardura 1 mg. (20 tablets=26 SR) Cardura 2 mg. (20 tablets=44.1 SR) Cardura 4 mg. (20 tablets=58.9 SR)	1-16	1
Prazosin	Minipress 1 mg. (30 tablets=7.6 SR,100 tablets=20.1) Minipress 2 mg.(30 tablets=12.6 SR,100 tablets=33.65) Minipress 5 mg.(30 tablets=22.75 SR, 100 tablets=64.6 SR)	2-20	2-3
Terazosin	Hytrin 2 mg. (30 tablets=34.9 SR) Hytrin 5 mg. (14 tablets=40.75 SR)	1-20	1-2
DRUG CLASS: CENTRAL α-AGONISTS AND OTHER CENTRALLY ACTING DRUGS			
Clonidine	CATAPRES 0.150 mg.	0.1-0.8	2
Methyldopa	Aldomet 250 mg. (30 tablets=12.85 SR, 100 tablets=38 SR) Dopamet 250 mg. (24 tablets=13.3 SR,100 tablets=55SR)	250-1000	2
DRUG CLASS: DIRECT VASODILATORS			
Hydralazine	Hydralazine 25 mg.(100 tablets=13.35 SR)	25-100	2

Appendix II: Combination Drugs for Hypertension Available in Saudi Arabia (June 2004)

Combination Type	Brand Name & Package Size & Price in Riyals
ACEIs and Diuretics	Captopril 50 mg + 25 mg Hydrochlorothiazide Capozide 50/25 (28 tablets=62.1 SR) Enalapril 20 mg + 12.5 mg Hydrochlorothiazid Co-Renitic 20/12.5 mg (30 tablets=84.05 SR) Lisinopril 20 mg + 12.5 mg Hydrochlorothiazide Zestoretic 20/12.5 mg (28 tablets=54.85 SR) Perindopril 2mg + 0.625mg Indapamide Preterax 2/0.625 mg (30 tablets=41.85 SR) Perindopril 4mg + 1.25 mg Indapamide Preterax 4/1.25 mg (30 tablets=57.15 SR) Quinapril 20 mg + 12.5 mg Hydrochlorothiazide Accuzide 20/12.5 mg (30 tablets=61 SR)
ARBs and Diuretics	Candesartan 16mg Hydrochlorothiazide 12.5mg tablet Atacand plus (28 tablets =108.3 SR) Losartan 50 mg 12.5 mg Hydrochlorothiazide Hyzaar 50/12.5 mg.(28 tablets=81.8 SR) Irbesartan 50 mg + hydrochlorothiazide 12.5 mg tablet Fortzaar 100/12.5 mg (28 tablets=172.2 SR) Irbesartan 100 mg + hydrochlorothiazide 12.5 mg tablet Co-Aprovel 150 / 12.5 mg (28 tablets=80.65 SR) CoAprovel 300/ 12.5 mg (28 tablets=99 SR) Valsartan 80 mg + 12.5 mg Hydrochlorothiazide Co-Diovan 80/12.5 mg.(28 tablets=87.5 SR) Valsartan 160 mg + 12.5 mg Hydrochlorothiazide Co-Diovan 160/12.5 mg.(28 tablets=102.4 SR) Valsartan 160 mg + 25 mg Hydrochlorothiazide Co-Diovan 160/25 mg.(28 tablets=102.4 SR)
Beta-Blockers and Diuretics	Bisoprolol 5mg + Hydrochlorothiazide5mg. Concor-5-Plus (20 tablets=21.65 SR) Pindolol 10mg + Clopamide 5 mg) Viskaldix (30 tablets=26.45 SR)
Beta-Blockers and CCBs	Atenolol 50mg + Nifedipine 20mg caps Nif-Ten (28 tablets=43 SR)
Centrally Acting Drug and Diuretic	Reserpine 0.1mg+ Dihydralazin10mg +Hydrochlorothiazide 10mg. Esidrex (30 Tablet=10.55)
Diuretic and Diuretic	Triamtrene 50 MG + Hydrochlorothiazide 25 MG. Dyazide (20 tablet=8.85 SR) Amiloride 5 MG + Hydrochlorothiazide 50 MG. Amuretic (20 tablet=8.65 SR) Amiloride 5 MG + Hydrochlorothiazide 50 MG. Apo-Amilzide (30 tablet=13 SR)

Appendix III: BP Percentile Charts for Children:



Age-specific percentiles for BP measurements in boys 1-13 yr of age

Age-specific percentiles of BP measurements in girls 1-13 yr of age

“IN MEMORIAM”



Sameer Bin Huraib

Born in Taif, Saudi Arabia on the 13th of October 1954. Died in Riyadh on the 15th of April 2003, at the age of 48.

We lost a prominent member-founder of our group on 13 April 2003. Professor Sameer Bin Huraib's, struggle with his disease was lengthy, but he went through it with his spirit high, patience and a great belief in the passion of Almighty God. We lost not only a friend, but also a founder who had contributed with great enthusiasm to the establishment of this group.

The short life of Dr. Huraib was full of professional achievements that go well beyond the borders of his native land. He graduated in Medicine from King Saud University, Riyadh (1979). He earned a scholarship at the University of Toronto, Ontario, Canada (September 1985) where he did his Fellowship training and then received the American Board of Internal Medicine (ABIM); Certificate of Competence in Nephrology Royal College of Physicians, Canada (1987); Fellow of the Royal College of Physicians (FRCPC) (1988); and Fellowship of the American College of Physicians (FACP) (January 1991).

Dr. Huraib was an example of diligence and thoughtfulness. His research work on glomerulonephritis and the epidemiology of hepatitis C in dialysis patients brought him national and regional fame. He was appointed as associate professor, and later to full professor in Internal Medicine and Nephrology at the King Saud University. In 1996 he moved to the King Fahad National Guard Hospital (KFNGH), Riyadh. Under his leadership, the renal services improved remarkably. He was appointed as the Chairman of the Department of Medicine and Head, Section of Nephrology. Here again, Professor Huraib excelled with dedication and hard work.

Professor Huraib served as the Chairman of The Riyadh Nephrology and Transplantation Club for one year. He also helped in the planning, founding and implementation of the Renal and Organ Transplantation Center. At the regional level, Professor Huraib was a founder member of the Arab Society of Nephrology and Renal Transplantation of which he was the President-Elect.

The Arab Society of Nephrology & Renal Transplantation has established "Bin Huraib's Prize" to be given during each of the Society's congresses for the best abstract submitted.

Our Saudi Hypertension Management Group is deeply indebted for Sameer as he was the first to think of its foundation. He gathered with his excellent social and professional contacts diverse professionals interested in hypertension to form our group. Thank to his dynamic personality and his good relations to many of us he was able to encourage many to join. In the spirit of his personality and character our group shall continue its work and remembers him with gratitude and thanks.

Acknowledgement

The group wishes to thank *Dr. Magdi Mohsen, Dr. Ala'a El-Alem, Dr. Tamer Heiba, Mr. Ashraf Samih, Mr. Jamal Al-Shafie, and Mr Nabil Al-Dubais from Pfizer, Saudi Arabia* for their unlimited support, profound professionalism, and truly ethical conduct while supporting our work.

We owe *Ms. Hala Merhi*, our administrative coordinator, many thanks and appreciation for her energetic support, for organization of our meetings, as well as the coordination of our various activities and preparing our guidelines and home page.

Our thank goes further to *Ms. Mona Alhamdan and Mr Fathi Alhaj* for their **administrative and logistic support**.



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