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List of Abbreviations

ABPM  Ambulatory Blood Pressure Monitor
ACEI  ACE Inhibitors
ARB  Angiotensin Receptor Antagonists
BB  Beta-Blockers
BMI  Body Mass Index
BP  Blood Pressure
CA  Calcium Antagonists
CHD  Coronary Heart Disease
CVD  Cardiovascular Disease
DBP  Diastolic Blood Pressure
DM  Diabetic Mellitus
ECG  Echocardiograph
ESC  European Society of Cardiology
ESH-ESC  European Society of Hypertension
ESRD  End Stage Renal Failure
HTN  Hypertension
ISH  Isolated Systolic Hypertension
LVH  Left Ventricular Hypertrophy
MS  Metabolic Syndrome
OD  Organ Damage
SBP  Systolic Blood Pressure
TOD  Target Organ Damage
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PREFACE

The purpose of the 2010 Gulf Hypertension Association (GHA) Guidelines for the Management of Arterial Hypertension is to provide an adequate review of the current clinical evidences in relation to classification, diagnoses, and management of hypertension and to make these evidences available for both primary care and specialist physicians in the Arabian Gulf Countries.

It is important to note that the contents of these guidelines, a significant amount of which are exerts from international guidelines, are meant to serve as general guideline and should therefore be used in conjunction with appropriate diagnoses and treatment approaches for hypertension.

The GHA task force for hypertension understands that the treating clinician’s judgment in diagnosis, treatment, and follow up of the cases remains the single most important approach for an effective management of hypertension.

*Gulf Heart Association*
Classification of Hypertension:

Although there have been substantial studies in the field of hypertension, no universal agreement exists on the definition of hypertension. Historically, the term hypertension has been synonymous with an elevation of arm cuff blood pressure (BP) beyond an arbitrary cut off BP level. In 2005, the Hypertension Writing Group defines hypertension as:

- Progressive cardiovascular syndrome arising from complex and interrelated etiologies.
- Early markers of the syndrome are often present before blood-pressure elevation is sustained; therefore, hypertension cannot be classified solely by discreet blood-pressure thresholds.
- Progression is strongly associated with function and structural cardiac and vascular abnormalities that damage the heart, kidneys, brain, vasculature and other organs and lead to premature morbidity and death.

The 2007 European Society of Hypertension (ESH) / European Society of Cardiology (ESC) guidelines defined and classified hypertension in adults, as shown in Table 1.
Table 1: Classification of Hypertension According to BP Levels

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>and &lt;80</td>
</tr>
<tr>
<td>Normal</td>
<td>120–129</td>
<td>and/or 80–84</td>
</tr>
<tr>
<td>High normal</td>
<td>130–139</td>
<td>and/or 85–89</td>
</tr>
<tr>
<td>Grade 1</td>
<td>140–159</td>
<td>and/or 90–99</td>
</tr>
<tr>
<td>Grade 2</td>
<td>160–179</td>
<td>and/or 100–109</td>
</tr>
<tr>
<td>Grade 3</td>
<td>≥ 180</td>
<td>≥ 110</td>
</tr>
<tr>
<td>Isolated systolic hypertension</td>
<td>≥ 140</td>
<td>&lt;90</td>
</tr>
</tbody>
</table>

Isolated systolic hypertension should be graded (1, 2, and 3) according to systolic BP values in the ranges indicated, provided that diastolic values are < 90mmHg.

High BP known as hypertension is highly prevalent in the Arabian Gulf countries. Hypertension is a powerful independent risk factor for the development of cardiovascular disease (CVD), and may have serious adverse prognostic effects. Hence, high priority should be given to the prevention, and treatment of hypertension.

White Coat Hypertension
It is defined as a persistently elevated clinic or office BP (>140/90 mm Hg) together with a normal daytime ambulatory pressure (<135/85 mm Hg).
Masked Hypertension
Some individuals with normal office BP (<140/90mmHg) may have elevated ambulatory or home BP values, a condition termed as a masked hypertension. These patients should be treated with antihypertensive drugs, particularly if end organ damage is evident.

Total Cardiovascular Risk Factors
All patients should be classified not only in relation to the grades of hypertension, but also in terms of the total cardiovascular risk resulting from the coexistence of different risk factors, organ damage and disease.
The Framingham Study indicated that hypertension is often associated with cluster of other risk factors. Less than 20% of the time, hypertension cases occur in isolation. Whereas, most of the time (50%) hypertension occurs with a cluster of two or three major risk factors., a rate twice that expected by chance.
The INTERHEART study reported that hypertensive patients with more than three risk factors had much higher (> 20-fold) increase of cardiovascular risk. Therefore, risk assessment is essential for making decisions on the type and intensity of therapy for hypertension.
Factors Influencing Prognosis

Presence of risk factors

- Age (Male >55 years; Female >65 years)
- Male gender
- Smoking
- Family history of premature cardiovascular disease (Male at age <55 years; Female at age <65 years)
- Dyslipidaemia
- Sedentary lifestyle
- Unhealthy eating
- Khat Chewing (Tree leaves chewed daily by a high proportion of the adult population in Yemen)
- Abdominal obesity
- Dysglycemia (diabetes, impaired glucose tolerance, impaired fasting glucose)

Presence of target organ damage

- Microalbuminuria or proteinuria
- Carotid wall thickening
- Left ventricular hypertrophy
- Chronic kidney disease (glomerular filtration rate < 60 ml/min/1.73 m2)
**Presence of atherosclerotic vascular disease**
- Previous stroke or TIA
- Coronary heart disease
- Peripheral arterial disease

**Table 2: Stratification of cardiovascular risk in four categories.**

<table>
<thead>
<tr>
<th>Blood Pressure (mm Hg)</th>
<th>Normal SBP 120-129 Or DBP 80-84</th>
<th>Normal SBP 120-129 Or DBP 80-84</th>
<th>Grade 1 HT SBP 140-159 Or DBP 90-99</th>
<th>Grade 2 HT SBP 160-179 Or DBP 100-109</th>
<th>Grade 3 HT SBP ≥180 DBP ≥110</th>
</tr>
</thead>
<tbody>
<tr>
<td>No other risk factors</td>
<td>Average risk</td>
<td>Average risk</td>
<td>Low added risk</td>
<td>Moderate added risk</td>
<td>High added risk</td>
</tr>
<tr>
<td>1-2 risk factors</td>
<td>Low added risk</td>
<td>Low added risk</td>
<td>Moderate added risk</td>
<td>Moderate added risk</td>
<td>very high added risk</td>
</tr>
<tr>
<td>3 or more risk factors, MS, OD or DM</td>
<td>Moderate added risk</td>
<td>High added risk</td>
<td>High added risk</td>
<td>High added risk</td>
<td>very high added risk</td>
</tr>
<tr>
<td>Established CV or renal disease</td>
<td>very high added risk</td>
<td>very high added risk</td>
<td>very high added risk</td>
<td>very high added risk</td>
<td>very high added risk</td>
</tr>
</tbody>
</table>

In table 2 total CV Risk is stratified in four categories. Low, moderate, high and very high risks refer to 10 year risk of a CV fatal or non-fatal event. The term “added risk” is used to emphasize that in all categories relative risk is greater than average risk.

*OD: subclinical organ damage; MS: metabolic syndrome.*
Diagnosis and Evaluation of Hypertension

Objectives of initial evaluation

1. Establish the diagnosis and grade of hypertension (Including office and none office BP reading)
2. The likelihood of secondary hypertension
3. The presence of target organ damage
4. The level of global CVD risk

History

(a) Duration and previous levels of high BP.
(b) General symptomatology; Hypertensive individuals are commonly asymptomatic but can present with non specific symptoms.
(c) Symptoms of organ damage;
   1. Brain and eyes: headache, vertigo, impaired vision, transient ischemic attacks, sensory or motor deficit
   2. Heart: palpitation, chest pain, shortness of breath, swollen ankles
   3. Kidney: thirst, polyuria, nocturia, hematuria
   4. Peripheral arteries: cold extremities, intermittent claudication
(d) Symptoms suggestive of secondary causes
(e) Intake of drugs or substances that can raise BP; Such as nasal drops, cocaine, amphetamines (e.g. Khat*), oral contraceptives, steroids, nonsteroidal anti-inflammatory drugs, erythropoietin, and cyclosporin;
(f) Lifestyle factors; Life style factors such as dietary intake of fat (animal fat in particular), salt and alcohol, quantification of smoking and physical activity, weight gain since early adult life;
(g) Sleep history; Is commonly associated with the development of hypertension. Sleep apnea should be suspected in obese individuals with disrupted sleep patterns. Snoring is a frequent finding but is oftentimes more reliably reported by the sleep partner.
(h) Past history; History or current symptoms of coronary disease, heart failure, cerebrovascular or peripheral vascular disease, renal disease, DM gout, dyslipidaemia, and asthma or any other significant illnesses, and drugs used to treat those conditions.
(i) Previous antihypertensive therapy; its results and adverse effects
(j) Personal, family and environmental factors that may influence BP, cardiovascular risk, as well as the course and outcome of therapy.
(k) A comprehensive family history should be obtained with particular attention to hypertension, DM, dyslipidaemia, premature coronary heart disease (CHD), stroke, peripheral artery or renal disease.

*Commonly used in Yemen*
Physical Examination

Cardiac examination
(a) Heart rate and rhythm should be noted. Ectopic beats and atrial fibrillation are common findings.
(b) An accentuated aortic second sound occurs frequent.
(c) A fourth heart sound suggests atrial enlargement and increased ventricular stiffness; a third heart sound suggests dilated cardiomyopathy and reduced left ventricular function.
(d) Certain murmurs are associated with hypertension such as pulmonic flow murmurs in conditions of high cardiac output.

Abdomen
(a) Periumbilical or flank bruits may suggest the presence of renal arterystenosis.
(b) Active, forceful pulsation along the aorta suggests an abdominal aorticaneurysm in older individuals.
(c) Polycystic kidney is usually palpable in the flanks and the related renalininsufficiency may be the etiology of the patient›s hypertension.
Neurologic Examination
A basic screening examination for motor and cranial nerve function, gait, stance, and coordination is important to establish a baseline for therapeutic follow-up.

Peripheral Pulses
The carotid arteries should be palpated and auscultated for the presence of bruits. Peripheral arteries: absence, reduction, or asymmetry of pulses, cold extremities, ischaemic skin lesions.

Blood Pressure Measurement
In general, the diagnosis of hypertension should be based on at least 2 BP measurements per visit and at least 2 to 3 visits, however, in some severe cases the diagnosis can be based on BP measurements taken at a single visit. Use of standardized measurement techniques is recommended when assessing BP (table 3).
Table 3: Recommended Technique for Measuring BP

1. The patient should be resting comfortably for 5 minutes in the seated position with back support. The arm should be bare or free of restrictive clothing or other materials and supported at heart level, as a lower position will result in an erroneously higher SBP and DBP. There should be no talking, and patients’ legs should not be crossed. The patient should avoid exertion, temperature extremes, eating, caffeine, or smoking for 1 hour before BP measurement.

2. Choose a cuff with an appropriate bladder size matched to the size of the arm. For measurements taken by auscultation, bladder width should be close to 40% of arm circumference and bladder length should cover 80 – 100% of arm circumference. When using an automated device, select the cuff size as recommended by its manufacturer.

3. Place the cuff so that the lower edge is 3 cm above the elbow crease and the bladder is centered over the brachial artery. At least three measurements should be taken in the same arm with the patient in the same position. The first reading should be discarded and the latter two averaged. BP also should be assessed after 2 minutes standing (with arm supported) and at times when patients report symptoms suggestive of postural hypotension.

4. Supine BP measurements may also be helpful in the assessment of elderly and diabetic patients.

5. Increase the pressure rapidly to 30 mm Hg above the level at which the radial pulse is extinguished (to exclude the possibility of a systolic auscultatory gap).
6. Place the bell or diaphragm of the stethoscope gently and steadily over the brachial artery.

7. Open the control valve so that the rate of deflation of the cuff is approximately 2 mm Hg per heart beat. A cuff deflation rate of 2 mm Hg per beat is necessary for accurate systolic and diastolic estimation.

8. Read the systolic level — the first appearance of a clear tapping sound (phase I Korotkoff) — and the diastolic level (the point at which the sounds disappear (phase V Korotkoff)). Continue to auscultate at least 10 mm Hg below phase V to exclude a diastolic auscultatory gap. Record the BP to the closest 2 mm Hg on the manometer (or 1 mm Hg on electronic devices) as well as the arm used and whether the patient was supine, sitting or standing.

9. If Korotkoff sounds persist as the level approaches 0 mm Hg, then the point of muffling of the sound is used (phase IV) to indicate the diastolic pressure.

10. In the case of arrhythmia, additional readings may be required to estimate the average systolic and diastolic pressure. Isolated extra beats should be ignored. Note the rhythm and pulse rate.

11. Leaving the cuff partially inflated for too long will fill the venous system and make the sounds difficult to hear. To avoid venous congestion, it is recommended that at least one minute should elapse between readings.

12. BP should be taken in both arms on at least one visit and if one arm has a consistently higher pressure, that arm should be subsequently used for BP measurement and interpretation.
Table 4: Ambulatory Blood Pressure Monitor (ABPM)

Beyond the diagnosis of hypertension, ABPM measurement may also be considered for selected patients for the management of hypertension (HTN).

**Untreated patients**
- Mild (Grade 1) to moderate (Grade 2) clinic BP elevation and without target organ damage.

**Treated patients**
- BP that is not below target values despite receiving appropriate antihypertensive therapy.
- Symptoms suggestive of hypotension.
- Fluctuating office BP readings.

**How to interpret?**
- Mean daytime ambulatory BP $\geq 135/85$ mmHg is considered elevated.
- Mean 24 h ambulatory BP $\geq 130/80$ mmHg is considered elevated.
- A drop in nocturnal BP of $<10\%$ is associated with increased risk of CV events.
Table 5: Home Blood Pressure

(1) Home BP readings can be used in the diagnosis of hypertension.

(2) The use of home BP monitoring on a regular basis should be considered for patients with hypertension, particularly those with:
   a) Diabetes mellitus
   b) Chronic kidney disease
   c) Suspected nonadherence
   d) Demonstrated white coat effect
   e) BP controlled in the office but not at home (masked hypertension).

(3) When white coat hypertension is suggested by home monitoring, its presence should be confirmed with ABPM.

(4) Patients should be advised to purchase and use only home BP monitoring devices appropriate for the individual and meet international standards.

(5) Home SBP $\geq 135$ mm Hg, or DBP $\geq 85$ mm Hg should be considered to be elevated and associated with an increased overall mortality risk analogous to office SBP readings of 140 mm Hg or higher or DBP 90 mm Hg or higher.
(6) Health care professionals should ensure that patients who measure their BP at home have adequate training.

(7) The accuracy of all individual patients’ validated devices (including electronic devices) must be regularly checked against a device of known calibration.

(8) Home BP values for assessing white coat hypertension or sustained hypertension should be based on duplicate measurements, morning and evening, for an initial seven-day period. First day home BP values should not be considered.

**Table 6: BP thresholds (mmHg) for definition of hypertension with different types of measurement**

<table>
<thead>
<tr>
<th></th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Office or clinic</td>
<td>140</td>
<td>90</td>
</tr>
<tr>
<td>24-hour</td>
<td>130</td>
<td>80</td>
</tr>
<tr>
<td>Day</td>
<td>135</td>
<td>85</td>
</tr>
<tr>
<td>Night</td>
<td>120</td>
<td>70</td>
</tr>
<tr>
<td>Home</td>
<td>135</td>
<td>85</td>
</tr>
</tbody>
</table>
**Table 7: Laboratory Investigations**

### Routine tests
- Fasting plasma glucose
- Fasting lipid profile
- Serum potassium
- Serum uric acid
- Serum creatinine
- Estimated creatinine clearance
- Hemoglobin and hematocrit
- Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination)
- Electrocardiogram

### Other tests
- Echocardiogram
- Carotid ultrasound
- Quantitative proteinuria (if dipstick test positive)
- Ankle-brachial BP Index
- Fundoscopy
- Glucose tolerance test (if fasting plasma glucose >5.6 mmol/L (100 mg/dL)
- Home and 24 h ambulatory BP monitoring
- Pulse wave velocity measurement (where available)
Extended evaluation (domain of the specialist)

- Further search for cerebral, cardiac, renal and vascular damage. Mandatory in complicated hypertension
- Search for secondary hypertension when suggested by history, physical examination or routine tests.

**Echocardiography in Hypertension**

Echocardiography is more accurate than ECG in the assessment of cardiac TOD related to hypertension, thus leading to a more precise stratification of total CVD risk. However, routine echocardiographic evaluation of all hypertensive patients is not recommended. Echocardiographic assessment of left ventricular mass, as well as of systolic and diastolic left ventricular function, is recommended for hypertensive patients suspected to have left ventricular dysfunction or coronary artery disease.

**Fundoscopy**

Examination of eye grounds is recommended in severe hypertensives only.
Mild retinal changes are largely non-specific except in young patients.
Hemorrhages, exudates and papilledema, only present in severe hypertension, are associated with increased CV risk.
Secondary Causes of Hypertension

A skillful physician may elicit clinical clues during history taking and physical examination which heightens suspicion to most secondary forms of HTN; presence of abdominal bruit (renal artery stenosis), reduced or delayed femoral pulses (coarctation of aorta), abdominal masses (polycystic kidney), abdominal striae (Cushing disease), paroxysmal headaches, pallor and palpitations (pheochromocytoma); and the use of contraceptive medications or illicit drug use (drug induced HTN).

Difficult to control HTN requiring multiple agents remains the most common reason for initiating secondary HTN workup. Patients with hypertension secondary to obstructive sleep apnea often have the typical features of loud snoring, daytime sleepiness, and obesity. Weight loss, continuous positive airway pressure, and aldosterone antagonists are effective in lowering BP in hypertensive patients with sleep apnea, which (if left untreated) has a poor prognosis.

Table 8, summarizes the clinical clues suggesting secondary hypertension and the recommended diagnostic procedures.
### Table 8: Some Causes of Secondary Hypertension

<table>
<thead>
<tr>
<th>Condition</th>
<th>Signs and Symptoms</th>
<th>Diagnostic Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hyperaldosteronism</strong></td>
<td>Refractory HTN Hypokalemia, orthostatic BP drop</td>
<td>Plasma and urinary potassium; plasma renin and aldosterone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Plasma or urinary aldosterone after saline load; adrenal CT/ MRI scans</td>
</tr>
<tr>
<td><strong>Renovascular</strong></td>
<td>Sudden onset of severe HTN in young women (fibromuscular disease)</td>
<td>Renal sonography Duplex Doppler sonography</td>
</tr>
<tr>
<td></td>
<td>Severe coronary, peripheral arterial or cerebrovascular disease; cigarette smoking</td>
<td>Magnetic resonance or computed tomography (CT) angiography</td>
</tr>
<tr>
<td></td>
<td>history (atherosclerotic disease) Holosystolic bruit with or without diastolic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>component</td>
<td></td>
</tr>
<tr>
<td><strong>Pheochromocytoma</strong></td>
<td>Paroxysmal and/ or refractory HTN. Anxiety, tremor, headaches, sweating, rapid</td>
<td>Plasma metanephrine; urine metanephrine</td>
</tr>
<tr>
<td></td>
<td>pulse, recent weight loss, orthostatic BP drop Multiple endocrine neoplasia: thyroid</td>
<td>Urinary catechols; plasma catechols (basal and after 0.3 mg clonidine); adrenal CT scans</td>
</tr>
<tr>
<td></td>
<td>or parathyroid enlargement, neurofibromas, café au lait spots</td>
<td>and scintiscans</td>
</tr>
<tr>
<td><strong>Cushing’s syndrome</strong></td>
<td>Obesity, unusual truncal distribution of fat and abdominal striae, excessive body</td>
<td>Morning plasmacortisol after 1mg dexamethasone at bedtime</td>
</tr>
<tr>
<td></td>
<td>or facial hair (Cushing’s syndrome)</td>
<td>Urinary cortisol after variable doses of dexamethasone; adrenal CT scans and scintiscans</td>
</tr>
<tr>
<td><strong>Aortic coarctation</strong></td>
<td>Absent or diminished femoral pulses</td>
<td>BP in legs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Echocardiography, magnetic resonance imaging or contrast aortography</td>
</tr>
</tbody>
</table>
Management of Hypertension

Initially elevated BP above 140 mm Hg systolic or 90 mm Hg diastolic must always be re-measured at least three times over at least 4 weeks to ensure that hypertension is present. Only if the level is very high (>180/110 mm Hg) or if symptomatic target organ damage is present should therapy be begun before the diagnosis is carefully established.

Non Pharmacological Treatment

Table 9: Lifestyle therapy in Hypertension

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduce foods with added sodium</td>
<td>&lt; 2300 mg/day</td>
</tr>
<tr>
<td>Weight loss</td>
<td>BMI &lt; 25 kg/m2</td>
</tr>
<tr>
<td>Physical activity</td>
<td>30-60 minutes 4-7 days/week</td>
</tr>
<tr>
<td>Dietary patterns</td>
<td>DASH diet</td>
</tr>
<tr>
<td>Smoking cessation</td>
<td>Smoke free environment</td>
</tr>
<tr>
<td>Waist Circumference Middle East (arbitrary)</td>
<td>Men  &lt; 94 cm</td>
</tr>
<tr>
<td></td>
<td>Women &lt; 80 cm</td>
</tr>
</tbody>
</table>
Pharmacological Treatment

Initiation of drug therapy

<table>
<thead>
<tr>
<th>Blood pressure (mmHg)</th>
<th>Other risk factors OD or disease</th>
<th>No other risk factors</th>
<th>1-2 risk factors</th>
<th>≥3 risk factors, MS or OD</th>
<th>Diabetes</th>
<th>Established CV or kidney disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal SBP 120-129 Or DBP 80-84</td>
<td>High normal SBP 130-139 Or DBP 85-89</td>
<td>No BP intervention</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes</td>
<td>Lifestyle changes + Immediate drug treatment</td>
</tr>
</tbody>
</table>

2007 European guidelines for the management of Arterial Hypertension
<table>
<thead>
<tr>
<th>Grade 1 HT SBP 140-159 Or DBP 90-99</th>
<th>Grade 2 HT SBP 160-179 Or DBP 100-109</th>
<th>Grade 3 HT SBP $\geq$ 180 Or DBP $\geq$ 110</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lifestyle changes for several months then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes for several weeks then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes + Immediate drug treatment</td>
</tr>
<tr>
<td>Lifestyle changes for several months then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes for several weeks then drug treatment if BP uncontrolled</td>
<td>Lifestyle changes + Immediate drug treatment</td>
</tr>
<tr>
<td>Lifestyle changes + Immediate drug treatment</td>
<td>Lifestyle changes + Immediate drug treatment</td>
<td>Lifestyle changes + Immediate drug treatment</td>
</tr>
<tr>
<td>Lifestyle changes + Immediate drug treatment</td>
<td>Lifestyle changes + Immediate drug treatment</td>
<td>Lifestyle changes + Immediate drug treatment</td>
</tr>
</tbody>
</table>
Hypertension Treatment without Compelling Indications

Monotherapy versus combined therapy

- Regardless of the drug employed, monotherapy allows achieving BP target in only a limited number of hypertensive patients.
- Use of more than one agent is necessary to achieve target
BP in the majority of patients. A vast array of effective and well tolerated combinations is available.

- Initial treatment can make use of monotherapy or combination of two drugs at low doses with a subsequent increase in drug doses or number, if needed.
- Monotherapy could be the initial treatment for a mild BP elevation with a low or moderate total cardiovascular risk. A combination of two drugs at low doses should be preferred as first step treatment when initial BP is in the grade 2 or 3 range or total cardiovascular risk is high or very high.
- Fixed combinations of two drugs can simplify treatment schedule and favour compliance.
- In several patients BP control is not achieved by two drugs, and a combination of three or more drugs is required.
- In uncomplicated hypertensives and in the elderly, antihypertensive therapy should normally be initiated gradually. In higher risk hypertensives, goal BP should be achieved more promptly, which favours initial combination therapy and quicker adjustment of doses.
- Based on the ONTARGET data we recommend not to regularly use a combination of ACEI and ARB, at least at the full doses employed in this trial, because of lack of additional cardiovascular benefit and increased risk of renal dysfunction (as well as of other adverse events).
### Table 10: Antihypertensive treatment: Preferred drugs

<table>
<thead>
<tr>
<th>Subclinical Organ Damage</th>
<th>ACEI, CA, ARB</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic atherosclerosis</td>
<td>CA, ACEI</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>ACEI, ARB</td>
</tr>
<tr>
<td>Renal dysfunction</td>
<td>ACEI, ARB</td>
</tr>
<tr>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic atherosclerosis</td>
<td>CA, ACEI</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>ACEI, ARB</td>
</tr>
<tr>
<td>Renal dysfunction</td>
<td>ACEI, ARB</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Event</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous stroke</td>
<td>ACEI, ARB, diuretics</td>
</tr>
<tr>
<td>Previous MI</td>
<td>BB, ACEI, ARB</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>BB, CA</td>
</tr>
<tr>
<td>Heart failure</td>
<td>diuretics, BB, ACEI, ARB, anti-aldosterone agents</td>
</tr>
<tr>
<td>Recurrent</td>
<td>ARB, ACEI</td>
</tr>
<tr>
<td>Permanent</td>
<td>BB, non-dihydropiridine CA</td>
</tr>
<tr>
<td>ESRD/proteinuria</td>
<td>ACEI, ARB, loop diuretics</td>
</tr>
<tr>
<td>Peripheral artery disease</td>
<td>CA</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Condition</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ISH (elderly)</td>
<td>diuretics, CA</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>ACEI, ARB, CA</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>ACEI, ARB</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>CA, methyldopa, BB</td>
</tr>
<tr>
<td>Blacks</td>
<td>diuretics, CA</td>
</tr>
</tbody>
</table>

Abbreviations: LVH: left ventricular hypertrophy; ISH: isolated systolic hypertension; ESRD: renal failure; ACEI: ACE inhibitors; ARB: angiotensin receptor antagonists; CA: calcium antagonists; BB: b-blockers
Hypertension treatment with compelling evidences

Anti-hypertensive Treatment in Diabetes

**Treatment of Hypertension in Association with diabetes Mellitus: Summary**
Threshold equal or above 130/80 mmHg and TARGET below 130/80 mmHg

- **Diabetes**
  - With Nephropathy
    - ACE Inhibitor
    - Or
    - ARB
  - Without Nephropathy
    - 1. ACE Inhibitor or ARB
    - Or
    - 2. Thiazide diuretic or DHPCCB

≥2 - drug combinations

A combination of 2 first line drugs may be considered as initial therapy if BP is 20 mmHg systolic or ≥10 mmHg diastolic above target.

Monitor serum potassium and creatinine carefully in patients with CKD prescribed ACEI or ARB.
Combination of ACEI and ARB are specifically not recommended in the absence of proteinuria.
Antihypertensive Treatment in Patients with Renal Dysfunction

Treatment of Hypertension in patients with Non Diabetic Chronic Kidney Disease

Target BP: < 130/80 mmHg

- Chronic Kidney Disease and Proteinuria*

  - ACEI or ARB (If ACEI is tolerated)

  - Addictive therapy: Thiazide diuretics

  - Alternate: If volume overload: loop diuretic

  - Combination with other agents

- *albumin creatinine ratio [ACR] > 30 mg/mmol or urinary protein > 500 mg/24 hours.

Monitor serum potassium and creatinine carefully in patients with CKD prescribed ACEI or ARB. Combination of ACEI and ARB are specifically not recommended in the absence of proteinuria.
Antihypertensive Treatment in Patients with Cerebrovascular Disease

Treatment of Hypertension in Patients with Cerebrovascular Disease

Strongly consider BP reduction in all patients after the acute phase of stroke or TIA.

- Stroke TIA
- An ACEI/ARB/diuretic combination is preferred
- Combinations of an ACEI with an ARB are not recommended
Antihypertensive Treatment in Patients with Heart Failure

### Treatment of Hypertension with Left Ventricular Systolic Dysfunction

| Systolic cardiac dysfunction | ACEI and Beta blocker  
| If ACEI intolerant: ARB  
| Titrate doses of ACEI or ARB to those used in clinical trails  
| If additional therapy is needed:  
| • Diuretic (Thiazide for hypertension; Loop for volume control)  
| • For CHF class III-IV or post MI; Aldosterone Antagonist  
| If ACEI and ARB are contraindicated: Hydralazine and Isosorbide dinitrate in combination  
| Non dihydropyridine CCB  
| If additional antihypertensive therapy is needed:  
| • ACEI/ARB Combination  
| • Long-acting DHP-CCB (Amlodipine) |
Treatment of Hypertension in Patients with coronary artery diseases:

A. Stable angina

| Stable angina | • Beta-blocker  
               | • Long-acting CCB |

ACEI are recommended for most patients with established CAD*:

- Caution should be exercised when combing a non DHP-CCB and a betablocker
- If abnormal systolic left ventricular function: avoid non DHP-CCB (Verapamil or Diltiazem)
- Combination of an ACEI with an ARB is not recommended in the absence of heart failure.

Those at low risk with well controlled risk factors may not benefit from ACEI therapy

Short-acting nifedipine

Not recommended and have deleterious effect.
Hypertension in Pregnancy
Hypertensive disorders are one of the most common complications of pregnancy and may be associated with significant maternal and fetal morbidity and mortality. Although the etiology of these disorders is becoming increasingly better understood, interventions to prevent hypertensive disorders of pregnancy have had poor results.
The diagnosis of a hypertensive disorder in a pregnant woman depends, in part, upon the gestational age at presentation. **Preeclampsia** refers to the syndrome of new onset of hypertension and proteinuria after 20 weeks of gestation in a previously normotensive woman, or worsening hypertension with new onset proteinuria in a woman with preexisting hypertension (superimposed preeclampsia).

**Preexisting hypertension** is defined as systolic pressure $\geq 140$ mmHg and/or diastolic pressure $\geq 90$ mmHg that antedates pregnancy, is present before the 20th week of pregnancy, or persists longer than 12 weeks postpartum.

**Gestational hypertension** refers to elevated BP first detected after 20 weeks of gestation in the absence of proteinuria. Over time, some patients with gestational hypertension will develop proteinuria and be considered preeclamptic, while others will be diagnosed with preexisting hypertension because of persistent BP elevation postpartum.

Preeclampsia is associated with substantial risk to the mother and the fetus. Recognized risk factors include primiparity and preexisting chronic hypertension.

The “cure” for preeclampsia is delivery of the fetus, but delivery earlier than necessary poses major risks to the fetus; management involves close followup of the mother and the fetus to prolong the pregnancy as long as is safely possible.

Non-pharmacological management (including close supervision and
restriction of activities) should be considered for pregnant women with SBP 140–149mmHg or DBP 90–95mmHg. In the presence of gestational hypertension (with or without proteinuria) drug treatment is indicated at BP levels $\geq$140/90mmHg. SBP levels $\geq$ 170 or DBP $\geq$110mmHg should be considered an emergency requiring hospitalization.

In non-severe hypertension, oral methyldopa, labetalol, calcium antagonists and (less frequently) b-blockers are drugs of choice. In pre-eclampsia with pulmonary edema, nitroglycerine is the drug of choice. Diuretic therapy is inappropriate because plasma volume is reduced.

As emergency, intravenous labetalol, oral methyldopa and oral nifedipine are indicated. Intravenous hydralazine is no longer the drug of choice because of an excess of perinatal adverse effects. Intravenous infusion of sodium nitroprusside is useful in hypertensive crises, but prolonged administration should be avoided.

Calcium supplementation, fish oil and low dose aspirin are not recommended. However, low dose aspirin may be used prophylactically in women with a history of early onset pre-eclampsia.
Antihypertensive Treatment in the Elderly

Randomized trials in patients with systolic-diastolic or isolated systolic hypertension aged ≥ 60 years have shown that a marked reduction in cardiovascular morbidity and mortality can be achieved with antihypertensive treatment.

Drug treatment can be initiated with thiazide diuretics, calcium antagonists, angiotensin receptor antagonists, ACE inhibitors, and β-blockers, in line with general guidelines.

Trials specifically addressing treatment of isolated systolic hypertension have shown the benefit of thiazides and calcium antagonists but subanalysis of other trials also show efficacy of angiotensin receptor antagonists.

Initial doses and subsequent dose titration should be more gradual because of a greater chance of undesirable effects, especially in very old and frail subjects.

BP goal is the same as in younger patients, i.e. <140/90mmHg or below, if tolerated.

Because of the increased risk of postural hypotension, BP should always be measured also in the erect posture.
Resistant Hypertension
Resistant hypertension is defined as failure to achieve goal BP when a patient adheres to the maximum tolerated doses of 3 antihypertensive drugs including a diuretic.

**Step 1: Confirm true resistant hypertension**
A careful evaluation of the patient to confirm the diagnosis and exclude factors associated with “pseudo-resistance,” (table 10).

**Step 2: Education**
Education and reinforcement of life-style issues that affect BP, such as sodium restriction, and weight loss if obese, are critical in treating resistant hypertension.

**Step 3: Identify and reverse factors contributing to true resistance**
(a) Specifically ask the patient about use of any pharmacological or herbal (Khat) agents that may increase BP; in case of identification of such a substance, discontinue or minimize its use.
(b) Evaluate the level of renal function with estimation of glomerular filtration rate and modify treatment accordingly.
(c) Perform a thorough search for secondary hypertension; if an identifiable cause is present, treat accordingly.

**Step 4: Treat aggressively with optimal doses of appropriate antihypertensive medications (including drug combinations) according to patient characteristics.**
Table 11: Causes of Pseudo-Resistant Hypertension

1. Improper BP measurement
2. Heavily calcified or arteriosclerotic arteries that is difficult to compress (in elderly persons)
3. White-coat effect
4. Poor patient adherence
   - Side effects of medication
   - Complicated dosing schedules
   - Poor relations between doctor and patient
   - Inadequate patient education
   - Memory or psychiatric problems
   - Costs of medication
5. Related to antihypertensive medication
   - Inadequate doses
   - Inappropriate combinations
6. Physician inertia (failure to change or increase dose regimens when not at goal).
### Table 12: Factors Contributing to Resistant Hypertension

1. **Drug-induced**
   - Nonsteroidal anti-inflammatory drugs (including cyclooxygenase-2 inhibitors)
   - Sympathomimetics (decongestants, anorectics)
   - Cocaine, amphetamines, other illicit drugs
   - Oral contraceptive hormones
   - Adrenal steroid hormones
   - Erythropoietin
   - Cyclosporine and tacrolimus
   - Licorice (included in some chewing tobacco)
   - Over-the-counter dietary and herbal supplements (e.g., ginseng, yohimbine, ma huang, bitter orange)

2. **Excess alcohol intake**

3. **Volume overload**
   - Excess sodium intake
   - Volume retention from kidney disease
   - Inadequate diuretic therapy
(4) **Associated conditions**
- Obesity
- Diabetes mellitus
- Older age

(5) **Identifiable causes of hypertension**
- Renal parenchymal disease
- Renovascular disease
- Primary aldosteronism
- Obstructive sleep apnea
- Pheochromocytoma
- Cushing’s syndrome
- Thyroid diseases
- Aortic coarctation
- Intracranial tumors
Hypertension Emergencies and Urgencies Definitions

Severe Hypertension BP > 180/110 mm Hg

Progress Target Organ Damage?

Yes

- HTN Emergency
- Parenteral Rx Admit to ICU

No

- 1st Episode HTN Urgency
- Oral Rx in ED Clinic appt: 24th

- Frequent Episodes
- Refill Rx Clinic in 72h
### Table 13: Examples of hypertension emergencies

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accelerated/malignant hypertension</td>
</tr>
<tr>
<td>Hypertensive encephalopathy</td>
</tr>
<tr>
<td>Acute left ventricular failure</td>
</tr>
<tr>
<td>Acute aortic dissection</td>
</tr>
<tr>
<td>Intracranial hemorrhage</td>
</tr>
<tr>
<td>Pheochromocytoma crisis</td>
</tr>
<tr>
<td>Monoamine oxidase inhibitor + tyramine interaction</td>
</tr>
<tr>
<td>Eclampsia</td>
</tr>
<tr>
<td>Substance/drug-induced acute hypertension</td>
</tr>
</tbody>
</table>

### Table 14: Examples of hypertension urgencies

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accelerated/malignant hypertension*</td>
</tr>
<tr>
<td>Severe hypertension associated with coronary artery disease</td>
</tr>
<tr>
<td>Severe hypertension in the organ transplant patient</td>
</tr>
<tr>
<td>Preoperative hypertension</td>
</tr>
<tr>
<td>Hypertension associated with burns</td>
</tr>
<tr>
<td>Severe, uncontrolled hypertension</td>
</tr>
</tbody>
</table>

*Can also be considered an emergency on the basis of acute target organ dysfunction.
### Table 15: Initial Evaluation of Patients with Hypertensive Emergency

<table>
<thead>
<tr>
<th>History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior diagnosis and treatment of hypertension</td>
</tr>
<tr>
<td>Intake of pressor agents: street drugs, sympathomimetics</td>
</tr>
<tr>
<td>Symptoms of cerebral, cardiac, and visual dysfunction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
</tr>
<tr>
<td>Funduscropy</td>
</tr>
<tr>
<td>Neurologic status</td>
</tr>
<tr>
<td>Cardiopulmonary status</td>
</tr>
<tr>
<td>Body fluid volume assessment</td>
</tr>
<tr>
<td>Peripheral pulses</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Laboratory evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematocrit and blood smear</td>
</tr>
<tr>
<td>Urine analysis</td>
</tr>
<tr>
<td>Automated chemistry: creatinine, glucose, electrolytes</td>
</tr>
<tr>
<td>Plasma renin activity and aldosterone (if primary aldosteronism is suspected)</td>
</tr>
<tr>
<td>Plasma renin activity before and 1 h after 25 mg captopril (if renovascular hypertension is suspected)</td>
</tr>
<tr>
<td>Spot urine or plasma for metanephrine (if pheochromocy toma is suspected)</td>
</tr>
<tr>
<td><strong>Chest radiograph</strong> (if heart failure or aortic dissection is suspected)</td>
</tr>
<tr>
<td><strong>Electrocardiogram.</strong></td>
</tr>
</tbody>
</table>
**Goal of Therapy**

The initial aim of treatment in hypertensive crises is to rapidly lower the diastolic pressure to about 100 to 105 mmHg; this goal should be achieved within two to six hours, with the maximum initial fall in BP not exceeding 25 percent of the presenting value. This level of BP control will allow gradual healing of the necrotizing vascular lesions. More aggressive hypotensive therapy is both unnecessary and may reduce the BP below the autoregulatory range, possibly leading to ischemic events (such as stroke or coronary disease).

**Treatment**

For many patients with urgent hypertension without symptoms of major target organ dysfunction, initiation of therapy with two oral agents is appropriate to lower BP to an intermediate target over 24 to 72 hours. Agents that reliably cause an immediate fall in BP include central sympatholytics (clonidine 0.1–0.2 mg), labetalol (200–400 mg), and amlodipine (2.5–5 mg). Responses to angiotensin-converting enzyme (ACE) inhibitors are more variable. In keeping with the vasoconstrictive nature of hypertensive emergencies, the parenteral drugs that are safest and most effective are listed in Table 13. Of the available choices, parenteral labetalol is particularly attractive because it does not require intra arterial BP monitoring, tends to protect the heart, and counteracts the marked sympathetic over activity and tachycardia that often accompany a hypertensive emergency. Sodium
nitroprusside is particularly attractive in hypertensive encephalopathy. With the exception of pulmonary edema or marked fluid overload, diuretics are not indicated for initial therapy in hypertensive emergencies (owing to the volume contraction that usually accompanies the condition). Loop-diuretics in particular are not recommended for the routine treatment of hypertensive urgencies or emergencies in the absence of fluid overload because they can cause additional reflex vasoconstriction.
# Table 16: Parenteral Antihypertensive Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset of Action</th>
<th>Duration of Action</th>
<th>Adverse Effects</th>
<th>Special Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VASODILATORS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>0.25-10.00 μg/kg per min as i.v. infusion</td>
<td>immediate</td>
<td>1-2 min</td>
<td>Nausea, vomiting, muscle twitching, sweating, thiocyanate and cyanide intoxication</td>
<td>Most hypertensive emergencies; caution with high intracranial pressure or azotemia</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>5-100 μg per min as i.v. infusion</td>
<td>2-5 min</td>
<td>5-10 min</td>
<td>Headache, vomiting, methemoglobinemia, tolerance with prolonged use</td>
<td>Coronary ischemia</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>5-15 mg per h as i.v.</td>
<td>5-10 min</td>
<td>1-4 h</td>
<td>Headache, nausea, flushing, tachycardia, local phlebitis</td>
<td>Most hypertensive emergencies; caution with acute heart failure</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>5-20 mg as i.v. 10-40 mg IM</td>
<td>10-20 min</td>
<td>1-4 h</td>
<td>Tachycardia, flushing, headache, vomiting, aggravation of angina</td>
<td>Eclampsia; caution with high intracranial pressure</td>
</tr>
<tr>
<td>ADRENERGIC INHIBITORS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td><strong>Phentolamine</strong></td>
<td>5-15 mg as i.v</td>
<td>1-2 min</td>
<td>3-10 min</td>
<td>Tachycardia, flushing, headache</td>
<td>Catecholamine excess</td>
</tr>
<tr>
<td><strong>Esmolol</strong></td>
<td>200-500 μg/kg per min for 4 min, then 50-300 μg/kg per min as i.v.</td>
<td>10-20 min</td>
<td>10-20 min</td>
<td>Hypotension, nausea</td>
<td>Aortic dissection, after operation</td>
</tr>
<tr>
<td><strong>Labetalol</strong></td>
<td>20-80 mg as i.v. bolus every 10 min or 2 mg per min as i.v. infusion</td>
<td>5-10 min</td>
<td>3-6 h</td>
<td>Vomiting, scalp tingling, burning in throat, dizziness, nausea, heart block, orthostatic hypotension</td>
<td>Most hypertensive emergencies except acute heart failure</td>
</tr>
<tr>
<td><strong>Furosemide</strong></td>
<td>20-40 mg in 1-2 min, repeated and higher doses with renal insufficiency</td>
<td>5-15 min</td>
<td>2-3 h</td>
<td>Volume depletion, hypokalemia</td>
<td>Usually needed to maintain efficacy of other drugs</td>
</tr>
</tbody>
</table>
Vascular Protection for Hypertension Patients

Lipid Lowering Agents
All hypertensive patients with established atherosclerotic vascular disease (previous stroke or TIA, or Coronary heart disease, or peripheral arterial disease ) renal disease or with type 2 diabetes should be considered for statin therapy aiming at serum total and LDL cholesterol levels of, respectively, <4.5mmol/l (175 mg/dl) and <2.5 mmol/l (100 mg/dl), and lower, if possible.

Hypertensive patients without established atherosclerotic vascular disease, renal disease or type 2 diabetes but with 3 or more of the risk factors in table should also be considered for statin treatment even if their baseline total and LDL serum cholesterol levels are not elevated.
Table 17: Cardiovascular Risk Factors for Consideration of Statin Therapy in Non-dyslipidemic Patients with Hypertension

1- Male
2- Age $\geq$55 years
3- LVH
4- Other ECG abnormalities:
   - LBBB,
   - LV strain pattern,
   - abnormal Q-waves or ST-T changes compatible with IHD
5- Microalbuminuria or proteinuria
6- Smoking
7- Family history of premature CVD
8- Total cholesterol to HDL cholesterol ratio $\geq$ 6 mmol/l
Anti-platelet Therapy

Antiplatelet therapy, in particular low-dose aspirin, should be prescribed to hypertensive patients with previous cardiovascular events.

Low-dose aspirin should also be considered in hypertensive patients without a history of cardiovascular disease if older than 50 years, with a moderate increase in serum creatinine or with a high cardiovascular risk.

In all these conditions, the benefit-to-risk ratio of this intervention (reduction in myocardial infarction greater than the risk of bleeding) has been proven favourable.

To minimize the risk of haemorrhagic stroke, antiplatelet treatment should be started after achievement of BP control.
G.H.A

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