**Draft Version 1**

**Iraqi Hypertension Guidelines 2016 / Iraqi Hypertension Society**

**Introduction**

Limited data are available on the prevalence of hypertension and the temporal trends of BP values in Iraq. Overall the prevalence of hypertension appears to be around 40.4% of the adult population (more than 24y old), with a steep increase with ageing and slightly more in males.

Blood pressure is normally distributed in the population and there is no natural cut-off point above which 'hypertension' definitively exists and below which it does not. The risk associated with increasing blood pressure is continuous, with each 2 mmHg rise in systolic blood pressure associated with a 7% increased risk of mortality from ischaemic heart disease and a 10% increased risk of mortality from stroke. Hypertension is one of the most important preventable causes of premature morbidity and mortality, and is a major risk factor for ischaemic and haemorrhagic stroke, myocardial infarction, heart failure, chronic kidney disease, cognitive decline and premature death. Untreated hypertension is usually associated with a progressive rise in blood pressure. The vascular and renal damage that this may cause can culminate in a treatment-resistant state.

**Definitions**

In this guideline the following definitions of hypertension stages are used.

Stage 1 hypertension Clinic blood pressure is 140/90mmHg or higher and subsequent ambulatory blood pressure monitoring (ABPM) daytime average or home blood pressure monitoring (HBPM) average blood pressure is 135/85 mmHg or higher.

Stage 2 hypertension Clinic blood pressure is 160/100mmHg or higher and subsequent ABPM daytime average or HBPM average blood pressure is 150/95 mmHg or higher.

Severe hypertension Clinic systolic blood pressure is 180mmHg or higher or clinic diastolic blood pressure is 110 mmHg or higher.

**Measuring blood pressure**

Measurement of BP at the upper arm is preferred and cuff and bladder dimensions should be adapted to the arm circumference. In the event of a significant (.10 mmHg) and consistent SBP difference between arms, which has been shown to carry an increased CV risk, the arm with the higher BP values should be used. A between-arms difference is meaningful if demonstrated by simultaneous arm measurement; if one gets a difference between arms with sequential measurement, it could be due to BP variability. In elderly subjects, diabetic patients and in other conditions in which orthostatic hypotension may be frequent or suspected, it is recommended that BP be measured 1 min and 3 min after assumption of the standing position. Orthostatic hypotension—defined as a reduction in SBP of ≥20 mmHg or in DBP of ≥10 mmHg within 3 min of standing—has been shown to carry a worse prognosis for mortality and CV events. If feasible, automated recording of multiple BP readings in the office with the patient seated in an isolated room, though providing less information overall, might be considered as a means to improve reproducibility and make office BP values closer to those provided by daytime ABPM or HBPM. BP measurements should always be associated with measurement of heart rate, because resting heart rate values independently predict CV morbid or fatal events in several conditions, including hypertension

**Home BP measurement**

1. Home BP monitoring 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: i. Diabetes mellitus; ii. Chronic kidney disease; iii. Suspected nonadherence; iv. Demonstrated white coat effect; v. BP controlled in the office but not at home (masked hypertension).

3. When white coat hypertension is suggested by home BP monitoring, its presence should be confirmed by repeat home BP monitoring or ambulatory BP monitoring before treatment decisions are made.

4. Home SBP values 135mmHg or DBP values 85mmHg should be considered to be elevated and associated with an increased overall mortality risk.

5. Home BP monitoring for assessing white coat hypertension or sustained hypertension should be on the basis of duplicate measures, morning and evening, for an initial 7- day period. First-day home BP values should not be considered.

**Ambulatory BP measurement**

Recommendations

1. Ambulatory BP monitoring can be used in the diagnosis of hypertension. Ambulatory BP monitoring should be considered when an office-induced increase in BP is suspected in treated patients with: i. BP that is not below target despite receiving appropriate chronic antihypertensive therapy; ii. Symptoms suggestive of hypotension (Grade C); iii. Fluctuating office BP readings (Grade D).

Automated devices may not measure blood pressure accurately if there is pulse irregularity (for example, due to atrial fibrillation), palpate the radial or brachial pulse before measuring blood pressure. If pulse irregularity is present, measure blood pressure manually using direct auscultation over the brachial artery. ensure that devices for measuring blood pressure are properly validated, maintained and regularly recalibrated according to manufacturers' instructions, and an appropriate cuff size for the person's arm is used. When measuring blood pressure in the clinic or in the home, standardise the environment and provide a relaxed, temperate setting, with the person quiet and seated, and their arm outstretched and supported.

2. Ambulatory BP monitoring upper arm devices that have been validated independently using established protocols must be used (see www.dableducational.org).

3. Therapy adjustment should be considered in patients with a mean 24-hour ambulatory BP monitoring SBP of 130 mm Hg and/or DBP of 80 mm Hg, or a mean awake SBP of 135 mm Hg and/or DBP of 85 mm Hg.

4. The magnitude of changes in nocturnal BP should be taken into account in any decision to prescribe or withhold drug therapy on the basis of ambulatory BP monitoring because a decrease in nocturnal BP of < 10% is associated with increased risk of cardiovascular events.

**Medical history**

The medical history should address the time of the first diagnosis of arterial hypertension, current and past BP measurements and current and past antihypertensive medications. Particular attention should be paid to indications of secondary causes of hypertension. Women should be questioned about pregnancy-related hypertension, and OCP or HRT. Hypertension translates into an increased risk of renal and CV complications (CHD; heart failure; stroke; PAD; CV death), especially when concomitant diseases are present. Therefore, a careful history of CVDs should be taken in all patients, to allow assessment of global CV risk, including concomitant diseases such as diabetes, clinical signs or a history of heart failure, CHD or PAD, valvular heart disease, palpitations, syncopal episodes, neurological disorders with an emphasis on stroke and transient ischaemic attack (TIA). A history of CKD should include the type and duration of kidney disease. Nicotine abuse and evidence for dyslipidaemia should be sought. A family history of premature hypertension and/or premature CVD is an important first indicator of familial (genetic) predisposition to hypertension and CVD.

**Physical examination**

**A**ims to establish or verify the diagnosis ofhypertension, establish current BP, screen for secondary causes ofhypertension and refine global CV risk estimation. All patients should undergo auscultation of the carotid arteries, heart and renal arteries. Murmurs should suggest further investigation (carotid ultrasound, echocardiography, renal vascular ultrasound, depending on the location of the murmur). Height, weight, and waist circumference should be measured with the patient standing, and BMI calculated. Pulse palpation and cardiac auscultation may reveal arrhythmias. In all patients, heart rate should be measured while the patient is at rest. An increased heart rate indicates an increased risk of heart disease. An irregular pulse should raise the suspicion of atrial fibrillation, including silent atrial fibrillation.

**White-coat (isolated office) hypertension and masked (isolated ambulatory) hypertension**

Office BP is usually higher than BP measured out of the office, which has been ascribed to the alerting response, anxiety and/or a conditional response to the unusual situation, and in which regression to the mean may play a role. Although several factors involved in office or out-of-office BP modulation may be involved, the difference between the two is usually referred to although somewhat improperly as the ‘white-coat effect’, whereas ‘white-coat-’or ‘isolated office-’ or ‘isolated clinic hypertension’ refers to the condition in which BP is elevated in the office at repeated visits and normal out of the office, either on ABPM or HBPM. Conversely, BP may be normal in the office and abnormally high out of the medical environment, which is termed ‘masked-’ or ‘isolated ambulatory hypertension’. The terms ‘true-’ or ‘consistent normotension’ and ‘sustained hypertension’ are used when both types of BP measurement are, respectively, normal or abnormal. Whereas the cut-off value for office BP is the conventional 140/90 mmHg, most studies in white-coat or masked hypertension have used a cut-off value of 135/85 mmHg for out-of-office daytime or home BP and 130/ 80 mmHg for 24-h BP. Notably, there is only moderate agreement between the definition of white-coat or masked hypertension diagnosed by ABPM or HBPM. It is recommended that the terms ‘white-coat hypertension’ and ‘masked hypertension’ be reserved to define untreated individuals. Factors associate white coat hypertension are: age, female sex and non-smoking. Prevalence is lower in the case of target OD or when office BP is based on repeated measurements or when measured by a nurse or another healthcare provider. The prevalence is also related to the level of office BP: for example, the percentage of white-coat hypertension amounts to about 55% in grade 1 hypertension and to only about 10% in grade 3 hypertension. ODis less prevalent in white coat hypertension than in sustained hypertension and prospective studies have consistently shown this to be the case also for CV events.

Masked hypertension prevalence averages about 13%. Several factors may raise out-of-office BP relative to office BP, such as younger age, male gender, smoking, alcohol consumption, physical activity, exercise-induced hypertension, anxiety, job stress, obesity, diabetes, CKD and family history of hypertension and the prevalence is higher when office BP is in the high normal range. Masked hypertension is frequently associated with other risk factors, asymptomatic OD and increased risk of diabetes and sustained hypertension

In people with symptoms of postural hypotension (falls or postural dizziness): measure blood pressure with the person either supine or seated measure blood pressure again with the person standing for at least 1 minute prior to measurement. If the systolic blood pressure falls by 20 mmHg or more when the person is standing: review medication, measure subsequent blood pressures with the person standing, consider referral to specialist care if symptoms of postural hypotension persist.

When considering a diagnosis of hypertension, measure blood pressure in both arms. If the difference in readings between arms is more than 20 mmHg, repeat the measurements. If the difference in readings between arms remains more than 20 mmHg on the second measurement, measure subsequent blood pressures in the arm with the higher reading. If blood pressure measured in the clinic is 140/90 mmHg or higher: Take a second measurement during the consultation. If the second measurement is substantially different from the first, take a third measurement. Record the lower of the last two measurements as the clinic blood pressure.

If the clinic blood pressure is 140/90 mmHg or higher, offer ambulatory blood pressure monitoring (ABPM) to confirm the diagnosis of hypertension. If a person is unable to tolerate ABPM, home blood pressure monitoring (HBPM) is a suitable alternative to confirm the diagnosis of hypertension. If the person has severe hypertension, consider starting antihypertensive drug treatment immediately, without waiting for the results of ABPM or HBPM.

While waiting for confirmation of a diagnosis of hypertension, carry out investigations for target organ damage (such as left ventricular hypertrophy, chronic kidney disease and hypertensive retinopathy) and a formal assessment of cardiovascular risk using a cardiovascular risk assessment tools .

If hypertension is not diagnosed but there is evidence of target organ damage such as left ventricular hypertrophy, albuminuria or proteinuria, consider carrying out investigations for alternative causes of the target organ damage.

If hypertension is not diagnosed, measure the person's clinic blood pressure at least every 6 months subsequently, and consider measuring it more frequently if the person's clinic blood pressure is close to 140/90mmHg.

When using ABPM to confirm a diagnosis of hypertension, ensure that at least two measurements per hour are taken during the person's usual waking hours (for example, between 08:00 and 22:00).Use the average value of at least 14 measurements taken during the person's usual waking hours to confirm a diagnosis of hypertension. Atrial fibrillation may prevent accurate blood pressure measurement with automated devices.

When using HBPM to confirm a diagnosis of hypertension, ensure that: for each blood pressure recording, two consecutive measurements are taken, at least 1minute apart and with the person seated and blood pressure is recorded twice daily, ideally in the morning and evening and blood pressure recording continues for at least 4 days, ideally for 7 days. Discard the measurements taken on the first day and use the average value of all the remaining measurements to confirm a diagnosis of hypertension.

Initial investigations should include: test for GUE, for microalbuminuria, measure plasma glucose, electrolytes, creatinine, estimated glomerular filtration rate, serum total cholesterol and HDL cholesterol examine the fundi for the presence of hypertensive retinopathy arrange for a 12-lead electrocardiograph to be performed.

For people aged under 40 years with stage 1 hypertension and no evidence of target organ damage, cardiovascular disease, renal disease or diabetes, consider evaluation of secondary causes of hypertension and a more detailed assessment of potential target organ damage. This is because 10-year cardiovascular risk assessments can underestimate the lifetime risk of cardiovascular events in these people.

**Monitoring treatment and blood pressure targets**

Use clinic blood pressure measurements to monitor the response to antihypertensive treatment with lifestyle modifications or drugs. Aim for a target clinic blood pressure below 140/90 mmHg in people aged under 60 years, and 150/90 above 60 years with treated hypertension. For people identified as having a 'white-coat effect' or masked hypertension, consider ABPM or HBPM as an adjunct to clinic blood pressure measurements to monitor the response to antihypertensive treatment with lifestyle modification or drugs. When using ABPM or HBPM to monitor response, aim for a target average blood pressure during the person's usual waking hours of: below 135/85 mmHg for people aged under 80 years below 145/85mmHg for people aged 80 years and over.

**Routine laboratory tests for the;** investigation of patients with hypertension

1. Routine laboratory tests that should be performed for the investigation of all patients with hypertension include the following. i. Urinalysis; ii. Blood chemistry (potassium, sodium, and creatinine); iii. Fasting blood glucose and/or glycated hemoglobin; iv. Serum total cholesterol, LDL, HDL, non-HDL cholesterol, and triglycerides; lipids may be drawn fasting or non-fasting; v. Standard 12-lead electrocardiography.

2. Assess urinary albumin excretion in patients with diabetes.

3. During the maintenance phase of hypertension management, tests (including those for electrolytes, creatinine, and fasting lipids) should be repeated with a frequency reflecting the clinical situation.

**Secondary Hypertension**

For people aged under 40 years with stage 1 hypertension and no evidence of target organ damage, cardiovascular disease, renal disease or diabetes, consider evaluation of secondary causes of hypertension and a more detailed assessment of potential target organ damage. This is because 10-year cardiovascular risk assessments can underestimate the lifetime risk of cardiovascular events in these people.

**Assessment for renovascular hypertension**

1. Patients presenting with 2 of the following clinical clues, suggesting renovascular hypertension, should be investigated: i. Sudden onset or worsening of hypertension and age > 55 or < 30 years; ii. Presence of an abdominal bruit; iii. Hypertension resistant to 3 drugs; iv. Increase in serum creatinine level 30% associated with use of an angiotensin converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB); v. Other atherosclerotic vascular disease, particularly in patients who smoke or have dyslipidemia; vi. Recurrent pulmonary edema associated with hypertensive surges.

2. When available, the following tests are recommended to aid in the usual screening for renal vascular disease: captoprilenhanced radioisotope renal scan, Doppler sonography, magnetic resonance angiography, and computed tomography (CT) angiography (for those with normal renal function).

**Endocrine hypertension**

A. Hyperaldosteronism: screening and diagnosis

1. Screening for hyperaldosteronism should be considered for the following patients: i. Hypertensive patients with unexplained spontaneous hypokalemia (Kþ < 3.5 mmol/L) or marked diuretic-induced hypokalemia (Kþ < 3.0 mmol/L); ii. Patients with hypertension refractory to treatment with 3 drugs; iii. Hypertensive patients found to have an incidental adrenal adenoma.

2. Screening for hyperaldosteronism should include; assessment of plasma aldosterone and plasma renin activity or plasma renin.

3. A diagnosis of primary hyperaldosteronism should be established by presence of inappropriate autonomous hypersecretion of aldosterone, When the diagnosis is established, the abnormality should be localized .

4. In patients with primary hyperaldosteronism and a definite adrenal mass who are eligible for surgery, adrenal venous sampling is recommended to assess for lateralization of aldosterone hypersecretion. AVS should be performed exclusively by experienced teams working in specialized centres.

B. Pheochromocytoma and paraganglioma: screening and diagnosis

1. If pheochromocytoma or paraganglioma is strongly suspected, the patient should be referred to a specialized hypertension centre, particularly if biochemical screening tests have already been found to be positive .

2. The following patients should be considered for screening for pheochromocytoma or paraganglioma:

i. Patients with paroxysmal, unexplained, labile, and/ or severe (BP 180/110 mm Hg) sustained hypertension refractory to usual antihypertensive therapy; ii. Patients with hypertension and multiple symptoms suggestive of catecholamine excess (eg, headaches, palpitations, sweating, panic attacks, and pallor); iii. Patients with hypertension triggered by b-blockers, monoamine oxidase inhibitors, micturition, changes in abdominal pressure, surgery, or anaesthesia; iv. Patients with an incidentally discovered adrenal mass; v. Patients with a predisposition to hereditary causes (eg, multiple endocrine neoplasia 2A or 2B, von Recklinghausen neurofibromatosis type 1, or Von Hippel-Lindau disease); vi. For patients with positive biochemical screening tests, localization of pheochromocytomas or paragangliomas should use magnetic resonance imaging (preferable), CT (if magnetic resonance imaging unavailable), and/ or iodine I-131 meta-iodobenzylguanidine scintigraphy.

**MANAGEMENT**

**Lifestyle interventions**

Lifestyle advice should be offered initially and then periodically to people undergoing assessment or treatment for hypertension. Ascertain people's diet and exercise patterns because a healthy diet and regular exercise can reduce blood pressure. Offer appropriate guidance and written or audiovisual materials to promote lifestyle changes and management of obesity. Relaxation therapies can reduce blood pressure and people may wish to pursue these as part of their treatment. Ascertain people's alcohol consumption and encourage a reduced or stop intake if they drink excessively, because this can reduce blood pressure and has broader health benefits. Discourage excessive consumption of coffee and other caffeine-rich products. Encourage people to keep their dietary sodium intake low, either by reducing or substituting sodium salt, as this can reduce blood pressure. Do not offer calcium, magnesium or potassium supplements as a method for reducing blood pressure. ????? Offer advice and help to smokers to stop smoking.

**Choosing antihypertensive drug treatment**

Where possible, recommend treatment with drugs taken only once a day. Prescribe non-proprietary drugs where these are appropriate and minimize cost. Offer people with isolated systolic hypertension (systolic blood pressure 160 mmHg or more) the same treatment as people with both raised systolic and diastolic blood pressure.

If diuretic treatment is to be initiated or changed, offer a thiazide-like diuretic, such as chlortalidone (12.5–25.0 mg once daily) or indapamide (1.5 mg modified-release or 2.5 mg once daily) in preference to a conventional thiazide diuretic such as bendroflumethiazide or hydrochlorothiazide. For treatment of resistant hypertension at step 4: Consider further diuretic therapy with low-dose spironolactone (25 mg once daily) if the blood potassium level is 4.5 mmol/l or lower. Use particular caution in people with a reduced estimated glomerular filtration rate because they have an increased risk of hyperkalaemia. Consider higher-dose thiazide-like diuretic treatment if the blood potassium level is higher than 4.5mmol/l.

Beta-blockers are not a preferred initial therapy for hypertension. However, beta-blockers may be considered in younger people, particularly: those with an intolerance or contraindication to ACE inhibitors and angiotensin II receptor antagonists or women of child-bearing potential or people with evidence of increased sympathetic drive. If therapy is initiated with a beta-blocker and a second drug is required, add a calcium-channel blocker rather than a thiazide-like diuretic to reduce the person's risk of developing diabetes.

**Choice of therapy for adults with hypertension**

without compelling indications for specific agents Recommendations

A. Recommendations for individuals with diastolic and/or systolic hypertension, In the general population <60 years, initiate pharmacologic treatment to lower BP at DBP above 90mmHg and treat to a goal DBP<90mmHg, and initiate pharmacologic treatment to lower BP at SBP above140 mmHg and treat to a goal SBP <140mmHg.

1. Initial therapy should be monotherapy with a thiazide/thiazide-like diuretic, an ACE inhibitors, a long acting CCB; or an ARB. If there are adverse effects, another drug from this group should be substituted. Hypokalemia should be avoided in patients treated with thiazide/thiazide-like diuretic monotherapy.

2. Additional antihypertensive drugs should be used if target BP levels are not achieved with standard-dose monotherapy. Add-on drugs should be chosen from first-line choices. Useful choices include a thiazide/thiazide-like diuretic or CCB with either: ACE inhibitor, ARB, or b-blocker

3. Combination therapy using 2 first-line agents may also be considered as initial treatment of hypertension, if SBP is 20 mm Hg greater than target or if DBP is 10 mm Hg greater than target. However, caution should be exercised in patients in whom a decrease in BP from initial combination therapy is more likely to occur or in whom it would be poorly tolerated (eg, elderly patients).

4. Before considering 3 drug regimen, review medication to ensure that 2 drug treatment is at optimal or best tolerated doses. If treatment with three drugs is required, the combination of ACE inhibitor or angiotensin II receptor blocker, calcium-channel blocker and thiazide-like diuretic should be used. Do not use ACE Inhibitors with ARB. If BP not controlled treat as resistant hypertension.

B. Recommendations for individuals with isolated systolic hypertension

1. Initial therapy should be single-agent therapy with a thiazide/thiazide-like diuretic, a long-acting dihydropyridine CCB, or an ARB. If there are adverse effects, another drug from this group should be substituted. Hypokalemia should be avoided in patients treated with thiazide/thiazide-like diuretic monotherapy.

2. Additional antihypertensive drugs should be used if target BP levels are not achieved with standard-dose monotherapy. Add-on drugs should be chosen from first-line options.

3. If BP is still not controlled with a combination of 2 first-line agents, or there are adverse effects, other classes of drugs (such as a-blockers, ACE inhibitors, centrally acting agents, or nondihydropyridine CCBs) may be added or substituted.

4. Possible reasons for poor response to therapy should be considered.

5. a-Blockers are not recommended as first-line agents for uncomplicated isolated systolic hypertension; and b-blockers are not recommended as first-line therapy for isolated systolic hypertension in patients aged 60 years. However, both agents may be used in patients with certain comorbid conditions or in combination therapy.

**Hypertension in Elderly**

there is no evidence that different classes are differently effective in the younger vs. the older patient. In the general population aged more than 60 years, initiate pharmacologic treatment to lower blood pressure (BP) at systolic blood pressure (SBP)below150 mmHg or diastolic blood pressure (DBP) below 90mmHg and treat to a goal SBP <150 mm Hg and goal DBP <90 mm Hg. In the general population aged above 60years, if pharmacologic treatment for high BP results in lower achieved SBP (eg, <140mmHg) and treatment is well tolerated and without adverse effects on health or quality of life, treatment does not need to be adjusted.

**White Coat Hypertension treatment**

In the absence of additional CV risk factors, intervention may be limited to lifestyle changes only, but this decision should be accompanied by a close follow-up of the patients (including periodical out-of-office BP monitoring) because, in white-coat hypertensive subjects, out-of-office BP is often higher than in truly normotensive subjects and white-coat hypertensives have a greater risk of developing OD and to progress to diabetes and sustained hypertension

**Treatment of hypertension in association with ischemic heart disease**

A. Recommendations for hypertensive patients with CAD

1. For most hypertensive patients with CAD, an ACE inhibitor or ARB is recommended.

2. For hypertensive patients with CAD, but without coexisting systolic heart failure, the combination of an ACE inhibitor and ARB is not recommended (Grade B).

3. For high-risk hypertensive patients, when combination therapy is being used, choices should be individualized. The combination of an ACE inhibitor and a dihydropyridine CCB is preferable to an ACE inhibitor and a thiazide/thiazide-like diuretic in selected patients.

4. For patients with stable angina pectoris but without previous heart failure, myocardial infarction, or coronary artery bypass surgery, either a b-blocker or CCB can be used as initial therapy.

5. Short-acting nifedipine should not be used.

6. When decreasing SBP to target levels in patients with established CAD (especially if isolated systolic hypertension is present), be cautious when the DBP is 60 mm Hg because of concerns that myocardial ischemia might be exacerbated.

**Treatment of hypertension who have had a recent myocardial infarction**

1. Initial therapy should include a b-blocker and an ACE inhibitor.

2. An ARB can be used if the patient is intolerant of an ACE inhibitor.

3. CCBs may be used in patients after myocardial infarctionwhen b-blockers are contraindicated or not effective. Nondihydropyridine CCBs should not be used when there is heart failure, evidenced by pulmonary congestion on examination or radiography

**Treatment of hypertension in association with heart failure**

1. In patients with systolic dysfunction (ejection fraction< 40%), ACE inhibitors and b-blockers are recommended for initial therapy. Aldosterone antagonists (mineralocorticoid receptor antagonists) may be added for patients with a recent cardiovascular hospitalization, acute myocardial infarction, elevated B-type natriuretic peptide or N-terminal pro-B-type natriuretic peptide level, or New York Heart Association class II-IV symptoms. Careful monitoring for hyperkalemia is recommended when adding an aldosterone antagonist to ACE inhibitor or ARB therapy. Other diuretics are recommended as additional therapy if needed. Beyond considerations of BP control, doses of ACE inhibitors or ARBs should be titrated to those reported to be effective in trials unless adverse effects become manifest.

2. An ARB is recommended if ACE inhibitors are not tolerated.

3. A combination of hydralazine and isosorbide dinitrate is recommended if ACE inhibitors and ARBs are contraindicated or not tolerated.

4. For hypertensive patients whose BP is not controlled, an ARB may be added to ACE inhibitor and other antihypertensive drug treatment. Careful monitoring should be used if combining an ACE inhibitor and an ARB because of potential adverse effects such as hypotension, hyperkalemia, and worsening renal function. Additional therapies may also include dihydropyridine CCBs.

**Treatment of hypertension in association with left ventricular hypertrophy**

1. Hypertensive patients with left ventricular hypertrophy should be treated with antihypertensive therapy to decrease the rate of subsequent cardiovascular events.

2. The choice of initial therapy can be influenced by the presence of left ventricular hypertrophy. Initial therapy can be drug treatment using ACE inhibitors, ARBs, long-acting CCBs, or thiazide/thiazide-like diuretics. Direct arterial vasodilators such as hydralazine or minoxidil should not be used.

**Treatment of hypertension in association with stroke**

A. BP management in acute stroke (onset to 72 hours)

1. For patients with ischemic stroke not eligible for thrombolytic therapy, treatment of hypertension in the setting of acute ischemic stroke or transient ischemic attack should not be routinely undertaken. Extreme BP increases (eg, SBP > 220 mm Hg or DBP > 120 mm Hg) may be treated to reduce the BP by approximately 15%, and not more than 25%, over the first 24 hours with gradual reduction thereafter. Avoid excessive lowering of BP because this might exacerbate existing ischemia or might induce ischemia, particularly in the setting of intracranial arterial occlusion or extracranial carotid or vertebral artery occlusion. Pharmacological agents and routes of administration should be chosen to avoid precipitous decreases in BP.

2. For patients with ischemic stroke eligible for thrombolytic therapy, very high BP (> 185/110 mm Hg) should be treated concurrently in patients who receive thrombolytic therapy for acute ischemic stroke to reduce the risk of secondary intracranial hemorrhage.

B. BP management after acute stroke

1. Strong consideration should be given to the initiation of antihypertensive therapy after the acute phase of a stroke or transient ischemic attack.

2. After the acute phase of a stroke, BP-lowering treatment is recommended to a target of consistently < 140/90 mm Hg.

3. Treatment with an ACE inhibitor and thiazide/ thiazide-like diuretic combination is preferred.

4. For patients with stroke, the combination of an ACE inhibitor and ARB is not recommended

**Treatment of hypertension in association with nondiabetic chronic kidney disease**

1. For patients with nondiabetic chronic kidney disease, target BP is < 140/90 mm Hg.

2. For patients with hypertension and proteinuric chronic kidney disease (urinary protein > 500 mg per 24 hours or albumin to creatinine ratio > 30 mg/mmol), initial therapy should be an ACE inhibitor or an ARB if there is intolerance to ACE inhibitors.

3. Thiazide/thiazide-like diuretics are recommended as additive antihypertensive therapy. For patients with chronic kidney disease and volume overload, loop diuretics are an alternative treatment.

4. In most cases, combination therapy with other antihypertensive agents might be needed to reach target BP levels.

5. The combination of an ACE inhibitor and ARB is not recommended for patients with nonproteinuric chronic kidney disease.

**Treatment of hypertension in association with renovascular disease**

1. Patients with hypertension attributable to atherosclerotic renal artery stenosis should be primarily medically managed because renal angioplasty and stenting offers no benefit over optimal medical therapy alone.

2. Renal artery angioplasty and stenting for atherosclerotic hemodynamically significant renal artery stenosis could be considered for patients with uncontrolled hypertension resistant to maximally tolerated pharmacotherapy, edema.

**Treatment of hypertension in association with diabetes mellitus**

1. Persons with diabetes mellitus should be treated to attain an SBP of < 140 mm Hg and DBP of < 90 mm Hg. Combination therapy using 2 first-line agents may also be considered as initial treatment of hypertension if SBP is 20 mm Hg greater than target or if DBP is 10 mm Hg greater than target. However, caution should be exercised in patients in whom a substantial decrease in BP is more likely or poorly tolerated (eg, elderly patients and patients with autonomic neuropathy).

2. For persons with cardiovascular or kidney disease, including microalbuminuria, or with cardiovascular risk factors in addition to diabetes and hypertension, an ACE inhibitor or an ARB is recommended as initial therapy.

3. For persons with diabetes and hypertension not included in other recommendations in this section, appropriate choices include (in alphabetical order): ACE inhibitors, ARBs, dihydropyridine CCBs, and thiazide/thiazide-like diuretics.

4. If target BP levels are not achieved with standard-dose monotherapy, additional antihypertensive therapy should be used. For persons in whom combination therapy with an ACE inhibitor is being considered, a dihydropyridine CCB is preferable to a thiazide/thiazide-like diuretic.

**Hypertension in Pregnancy**.

Drug treatment of severe hypertension in pregnancy (SBP >160 mmHg or DBP >110 mmHg) is recommended. Drug treatment may also be considered in pregnant women with persistent elevation of BP ≥150/95 mmHg, and in those with BP ≥140/90 mmHg in the presence of gestational hypertension, subclinical OD or symptoms. In women at high risk of pre-eclampsia, provided they are at low risk of gastrointestinal haemorrhage, treatment with low dose aspirin from 12 weeks until delivery may be considered. In women with child-bearing potential RAS blockers are not recommended and should be avoided.

Methyldopa, labetolol and nifedipine should be considered preferential antihypertensive drugs in pregnancy. Intravenous labetolol or infusion of nitroprusside should be considered in case of emergency (pre-eclampsia).

**Malignant hypertension**

Malignant hypertension is a hypertensive emergency, clinically defined as the presence of very high BP associated with ischaemic OD (retina, kidney, heart or brain). Although its frequency is very low, the absolute number of new cases has not changed much over the past 40 years. Current treatment is founded on agents that can be administered by intravenous infusion and titrated, and so can act promptly but gradually in order to avoid excessive hypotension and further ischaemic OD. Labetalol, sodium nitroprusside, nicardipine, nitrates and furosemide are among the intravenous agents most usually employed but in these severely ill patients, treatment should be individualized by the physician. When diuretics are insufficient to correct volume retention, ultrafiltration and temporary dialysis may help.

**Hypertensive emergencies and urgencies**

Hypertensive emergencies are defined as large elevations in SBP or DBP (180 mmHg or 120 mmHg, respectively) associated with impending or progressive OD, such as major neurological changes, hypertensive encephalopathy, cerebral infarction, intracranial haemorrhage, acute LV failure, acute pulmonary oedema, aortic dissection, renal failure, or eclampsia. Isolated large BP elevations without acute OD (hypertensive urgencies) often associated with treatment discontinuation or reduction as well as with anxiety should not be considered an emergency but treated by reinstitution or intensification of drug therapy and treatment of anxiety. Treat as malignant hypertension

**Resistant hypertension;**

Consider further diuretic therapy with low-dose spironolactone (25 mg once daily) if the blood potassium level is 4.5 mmol/l or lower. Use particular caution in people with a reduced estimated glomerular filtration rate because they have an increased risk of hyperkalaemia. Consider higher-dose thiazide-like diuretic treatment if the blood potassium level is higher than 4.5mmol/l. When using further diuretic therapy, monitor blood sodium and potassium and renal function within 1 month and repeat as required thereafter. If further diuretic therapy is not tolerated, or is contraindicated or ineffective, consider an alpha- or beta-blocker. If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained. Mineralocorticoid receptor antagonists, amiloride, and the alpha blocker doxazosin should be considered, if no contraindication exists. In case of ineffectiveness of drug treatment invasive procedures such as renal denervation and baroreceptor stimulation may be considered. Until more evidence is available on the long-term efficacy and safety of renal denervation and baroreceptor stimulation, it is recommended that these procedures remain in the hands of experienced operators and diagnosis and follow-up restricted to hypertension centers.?????

**Perioperative management of hypertension**

Presence of hypertension is one of the common reasons for postponing necessary surgery, the question of whether antihypertensive therapy should be maintained immediately before surgery is frequently debated. Sudden withdrawal of clonidine or beta-blockers should be avoided because of potential BP or heart rate rebounds. Both types of agent can be continued over surgery and, when patients are unable to take oral medications, beta-blockers can be given parenterally and clonidine transdermally. Diuretics should be avoided on the day of surgery because of potential adverse interaction with surgery-dependent fluid depletion. ACE inhibitors and ARBs may also be potentiated by surgery-dependent fluid depletion and it has been suggested that they should not be taken on the day of surgery and restarted after fluid repletion has been assured. Post-surgery BP elevation, when it occurs, is frequently caused by anxiety and pain after awakening, and disappears after treating anxiety and pain.

**Markers of organ damage(OD)**

LVH/ECG Low, LVH/echo Moderate, LVH/cardiac magnetic resonance, eGFR , Urinary protein excretion, Carotid wall thickness, Pulse wave velocity, Ankle/brachial index.

**Treatment of associated risk**

It is recommended to use statin therapy in hypertensive patients at moderate to high CV risk, targeting a low-density lipoprotein cholesterol or non HDL cholesterol. Antiplatelet therapy, in particular low-dose aspirin, is recommended in hypertensive patients with previous CV events. Aspirin should also be considered in hypertensive patients with reduced renal function or a high CV risk, provided that BP is well controlled. Aspirin is not recommended for CV prevention in low-moderate risk hypertensive patients, in whom absolute benefit and harm are equivalent. In hypertensive patients with diabetes, a HbA1c target of <6.5-7.0% is recommended in diabetics.

**Follow-up of hypertensive patients**

After the initiation of antihypertensive drug therapy, it is important to see the patient at 2- to 4-week intervals to evaluate the effects on BP and to assess possible side-effects. Some medications will have an effect within days or weeks but a continued delayed response may occur during the first 2 months. Once the target is reached, a visit interval of a few months is reasonable, and evidence has been obtained that no difference exists in BP control between 3- and 6-month intervals.

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