

Ministry of Healthcare of Ukraine
Poltava State Medical University

**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

Academic discipline

Module № 4

Content module

Study subject

Course

Faculty

Internal medicine

Current practice of internal medicine

Management of the patients with main 'symptoms and syndromes in cardiology clinic

Management of the patients with hypertension

VI

Of foreign students training

1.The aims of the training course:

To Know:

1. The principles of non-medicamentous and pharmacological treatment.
2. The first and second line treatment.
3. Existing standards of treatment.
4. Monotherapy and combination therapy.
5. Hypertensive crisis, medical tactics.
6. Primary and secondary prevention.
7. Weather and performance.

To be able to:

- Conduct surveys and patients examination with major cardiological syndromes
- To draft survey the patients with heart diseases, to justify the use of major invasive and non-invasive diagnostic techniques which are using in cardiology, to identify indications and contraindications for their conduction, possible complications
- Identify different options for the course and complications of heart disease
- Carry out differential diagnosis, justify and formulate diagnoses for major cardiac syndromes based on laboratory analysis and test tool
- Prescribe treatment, determine prognosis, to conduct primary and secondary prevention in heart disease
- Register and interpret the ECG in 12 assignments
- Measure and interpret blood pressure
- Diagnose and assist in syncope
- Diagnose and assist in hypertensive crisis
- Diagnose and assist with arterial hypotension
- Diagnose and assist in the paroxysmal disorders of cardiac rhythm
- Diagnose and assist syndrome Morhany-Edems-Stoks
- Conduct pulmonary heart reanimation
- Demonstrate knowledge of moral principles medical specialist and professional principles of subordination

The contents of topic:

Text

Merck Manual Professional Version <http://www.merckmanuals.com/professional>

Last full review/revision May 2014 by George L. Bakris, MD

Hypertension is sustained elevation of resting systolic BP (≥ 140 mm Hg), diastolic BP (≥ 90 mm Hg), or both. Hypertension with no known cause (primary; formerly, essential hypertension) is most common. Hypertension with an identified cause (secondary hypertension) is usually due to chronic kidney disease or primary aldosteronism. Usually, no symptoms develop unless hypertension is severe or long-standing. Diagnosis is by sphygmomanometry. Tests may be done to determine cause, assess damage, and identify other cardiovascular risk factors. Treatment involves lifestyle changes and drugs, including diuretics, β - blockers, ACE inhibitors, angiotensin II receptor blockers, and Ca channel blockers.

Etiology

Hypertension may be primary (85 to 95% of cases) or secondary.

Primary hypertension

Hemodynamics and physiologic components (eg, plasma volume, activity of the renin-angiotensin system) vary, indicating that primary hypertension is unlikely to have a single cause. Even if one factor is initially responsible, multiple factors are probably involved in sustaining elevated BP (the mosaic theory). In afferent systemic arterioles, malfunction of ion pumps on sarcolemmal membranes of smooth muscle cells may lead to chronically increased vascular tone. Heredity is a predisposing factor, but the exact mechanism is unclear. Environmental factors (eg, dietary Na, obesity, stress) seem to affect only genetically susceptible people at younger ages; however, in patients > 65, high Na intake is more likely to precipitate hypertension.

Secondary hypertension

Causes include primary aldosteronism (thought to be most common), renal parenchymal disease (eg, chronic glomerulonephritis or pyelonephritis, polycystic renal disease, connective tissue disorders, obstructive uropathy), renovascular disease, pheochromocytoma, Cushing syndrome, congenital adrenal hyperplasia, hyperthyroidism, myxedema, and coarctation of the aorta. Excessive alcohol intake and use of oral contraceptives are common causes of curable hypertension. Use of sympathomimetics, NSAIDs, corticosteroids, cocaine, or licorice commonly contributes to worsening of BP control.

Pathophysiology

Because BP equals cardiac output (CO) $\times$ total peripheral vascular resistance (TPR), pathogenic mechanisms must involve

- Increased CO
- Increased TPR
- Both

In most patients, CO is normal or slightly increased, and TPR is increased. This pattern is typical of primary hypertension and hypertension due to primary aldosteronism, pheochromocytoma, renovascular disease, and renal parenchymal disease.

In other patients, CO is increased (possibly because of venoconstriction in large veins), and TPR is inappropriately normal for the level of CO. Later in the disorder, TPR increases and CO returns to normal, probably because of autoregulation. Some disorders that increase CO (thyrotoxicosis, arteriovenous fistula, aortic regurgitation), particularly when stroke volume is increased, cause isolated systolic hypertension. Some elderly patients have isolated systolic hypertension with normal or low CO, probably due to inelasticity of the aorta and its major branches. Patients with high, fixed diastolic pressures often have decreased CO.

Plasma volume tends to decrease as BP increases; rarely, plasma volume remains normal or increases. Plasma volume tends to be high in hypertension due to primary aldosteronism or renal parenchymal disease and may be quite low in hypertension due to pheochromocytoma. Renal blood flow gradually decreases as diastolic BP increases and arteriolar sclerosis begins. GFR remains normal until late in the disorder; as a result, the filtration fraction is increased. Coronary, cerebral, and muscle blood flow is maintained unless severe atherosclerosis coexists in these vascular beds.

Abnormal Na transport

In many cases of hypertension, Na transport across the cell wall is abnormal, because the Na-K pump (Na^+ , K^+ -ATPase) is defective or inhibited or because permeability to Na^+ is increased. The result is increased intracellular Na, which makes the cell more sensitive to sympathetic stimulation. Ca follows Na, so accumulation of intracellular Ca may be responsible for the increased sensitivity. Because Na^+ , K^+ -ATPase may pump norepinephrine back into sympathetic neurons (thus inactivating this neurotransmitter), inhibition of this mechanism could also enhance the effect of norepinephrine, increasing BP. Defects in Na transport may occur in normotensive children of hypertensive parents.

Sympathetic nervous system

Sympathetic stimulation increases BP, usually more in patients with prehypertension (systolic BP 120 to 139 mm Hg, diastolic BP 80 to 89 mm Hg) or hypertension (systolic BP ≥ 140 mm Hg, diastolic BP ≥ 90 mm Hg, or both) than in normotensive patients. Whether this hyperresponsiveness resides in the sympathetic nervous system or in the myocardium and vascular smooth muscle is unknown. A high resting pulse rate, which may result from increased sympathetic nervous activity, is a well-known predictor of hypertension. In some hypertensive patients, circulating plasma catecholamine levels during rest are higher than normal.

Renin-angiotensin-aldosterone system

This system helps regulate blood volume and therefore BP. Renin, an enzyme formed in the juxtaglomerular apparatus, catalyzes conversion of angiotensinogen to angiotensin I. This inactive product is cleaved by ACE, mainly in the lungs but also in the kidneys and brain, to angiotensin II, a potent vasoconstrictor that also stimulates autonomic centers in the brain to increase sympathetic discharge and stimulates release of aldosterone and ADH. Aldosterone and ADH cause Na and water retention, elevating BP. Aldosterone also enhances K excretion; low plasma K (< 3.5 mEq/L) increases vasoconstriction through closure of K channels. Angiotensin III, present in the circulation, stimulates aldosterone release as actively as angiotensin II but has much less pressor activity. Because chymase enzymes also convert angiotensin I to angiotensin II, drugs that inhibit ACE do not fully suppress angiotensin II production.

Renin secretion is controlled by at least 4 mechanisms, which are not mutually exclusive: (1) A renal vascular receptor responds to changes in tension in the afferent arteriolar wall; (2) a macula densa receptor detects changes in the delivery rate or concentration of NaCl in the distal tubule; (3) circulating angiotensin has a negative feedback effect on renin secretion; and (4) via the renal nerve, the sympathetic nervous system stimulates renin secretion mediated by β -receptors.

Angiotensin is generally acknowledged to be responsible for renovascular hypertension, at least in the early phase, but the role of the renin-angiotensin-aldosterone system in primary hypertension is not established. However, in black and elderly patients with hypertension, renin levels tend to be low. The elderly also tend to have low angiotensin II levels.

Hypertension due to chronic renal parenchymal disease (renoprival hypertension) results from the combination of a renin-dependent mechanism and a volume-dependent mechanism. In most cases, increased renin activity is not evident in peripheral blood. Hypertension is typically moderate and sensitive to Na and water balance.

Vasodilator deficiency

Deficiency of a vasodilator (eg, bradykinin, nitric oxide) rather than excess of a vasoconstrictor (eg, angiotensin, norepinephrine) may cause hypertension. If the kidneys do not produce adequate amounts of vasodilators (because of renal parenchymal disease or bilateral nephrectomy), BP can increase. Vasodilators and vasoconstrictors (mainly endothelin) are also produced in endothelial cells. Therefore, endothelial dysfunction greatly affects BP.

Pathology and complications

No pathologic changes occur early in hypertension. Severe or prolonged hypertension damages target organs (primarily the cardiovascular system, brain, and kidneys), increasing risk of

- Coronary artery disease (CAD) and MI
- Heart failure
- Stroke (particularly hemorrhagic)
- Renal failure
- Death

The mechanism involves development of generalized arteriolosclerosis and acceleration of atherogenesis. Arteriolosclerosis is characterized by medial hypertrophy, hyperplasia, and hyalinization; it is particularly apparent in small arterioles, notably in the eyes and the kidneys. In the kidneys, the changes narrow the arteriolar lumen, increasing TPR; thus, hypertension leads to more hypertension. Furthermore, once arteries are narrowed, any slight additional shortening of already hypertrophied smooth muscle reduces the lumen to a greater extent than in normal-diameter arteries. These effects may explain why the longer hypertension has existed, the less

likely specific treatment (eg, renovascular surgery) for secondary causes is to restore BP to normal.

Because of increased afterload, the left ventricle gradually hypertrophies, causing diastolic dysfunction. The ventricle eventually dilates, causing dilated cardiomyopathy and heart failure (HF) due to systolic dysfunction often worsened by arteriosclerotic CAD. Thoracic aortic dissection is typically a consequence of hypertension; almost all patients with abdominal aortic aneurysms have hypertension.

Symptoms and Signs

Hypertension is usually asymptomatic until complications develop in target organs. Dizziness, flushed facies, headache, fatigue, epistaxis, and nervousness are not caused by uncomplicated hypertension. Severe hypertension can cause severe cardiovascular, neurologic, renal, and retinal symptoms (eg, symptomatic coronary atherosclerosis, HF, hypertensive encephalopathy, renal failure).

A 4th heart sound is one of the earliest signs of hypertensive heart disease.

Retinal changes may include arteriolar narrowing, hemorrhages, exudates, and, in patients with encephalopathy, papilledema. Changes are classified (according to the Keith, Wagener, and Barker classification) into 4 groups with increasingly worse prognosis: constriction of arterioles only (grade 1), constriction and sclerosis of arterioles (grade 2), hemorrhages and exudates in addition to vascular changes (grade 3), and papilledema (grade 4).

Diagnosis

- Multiple measurements of BP to confirm
- Urinalysis and urinary albumin:creatinine ratio; if abnormal, consider renal ultrasonography
- Blood tests: Fasting lipids, creatinine, K
- Renal ultrasonography if creatinine increased
- Evaluate for aldosteronism if K decreased
- ECG: If left ventricular hypertrophy, consider echocardiography
- Sometimes thyroid-stimulating hormone measurement
- Evaluate for pheochromocytoma or a sleep disorder if BP elevation sudden and labile or severe

Hypertension is diagnosed and classified by sphygmomanometry. History, physical examination, and other tests help identify etiology and determine whether target organs are damaged.

BP must be measured twice—first with the patient supine or seated, then after the patient has been standing for ≥ 2 min—on 3 separate days. The average of these measurements is used for diagnosis. BP is classified as normal, prehypertension, or stage 1 (mild) or stage 2 hypertension. Normal BP is much lower for infants and children.

Ideally, BP is measured after the patient rests > 5 min and at different times of day. A BP cuff is applied to the upper arm. An appropriately sized cuff covers two thirds of the biceps; the bladder is long enough to encircle $>80\%$ of the arm, and bladder width equals at least 40% of the arm's circumference. Thus, obese patients require large cuffs. The health care practitioner inflates the cuff above the expected systolic pressure and gradually releases the air while listening over the brachial artery. The pressure at which the first heartbeat is heard as the pressure falls is systolic BP. Total disappearance of the sound marks diastolic BP. The same principles are followed to measure BP in a forearm (radial artery) and thigh (popliteal artery). Sphygmomanometers that contain mercury are most accurate. Mechanical devices should be calibrated periodically; automated readers are often inaccurate.

BP is measured in both arms. BP > 15 mm Hg higher in one arm than the other is associated with higher mortality, and requires evaluation of the upper vasculature when this pattern of measurement is found. BP is also measured in a thigh (with a much larger cuff) to rule out coarctation of the aorta, particularly in patients with diminished or delayed femoral pulses; with coarctation, BP is significantly lower in the legs. If BP is in the low-hypertensive range or is markedly labile, more BP measurements are desirable. BP measurements may be sporadically high before hypertension becomes sustained; this phenomenon probably accounts for –white coat hypertension, in which BP is elevated when measured in the physician's office but normal when measured at home or by ambulatory BP monitoring. However, extreme BP elevation alternating with normal readings is unusual and possibly suggests pheochromocytoma, a sleep disorder such as sleep apnea, or unacknowledged drug use.

History

The history includes the known duration of hypertension and previously recorded levels; any history or symptoms of CAD, HF, or other relevant coexisting disorders (eg, stroke, renal dysfunction, peripheral arterial disease, dyslipidemia, diabetes, gout); and a family history of any of these disorders. Social history includes exercise levels and use of tobacco, alcohol, and stimulant drugs (prescribed and illicit). A dietary history focuses on intake of salt and stimulants (eg, tea, coffee, caffeine-containing sodas, energy drinks).

Physical examination

The physical examination includes measurement of height, weight, and waist circumference; funduscopic examination for retinopathy; auscultation for bruits in the neck and abdomen; and a full cardiac, respiratory, and neurologic examination. The abdomen is palpated for kidney

enlargement and abdominal masses. Peripheral arterial pulses are evaluated; diminished or delayed femoral pulses suggest aortic coarctation, particularly in patients < 30. A unilateral renal artery bruit may be heard in slim patients with renovascular hypertension.

Testing

The more severe the hypertension and the younger the patient, the more extensive is the evaluation. Generally, when hypertension is newly diagnosed, routine testing is done to

- Detect target-organ damage
- Identify cardiovascular risk factors

Tests include

- Urinalysis and spot urine albumin:creatinine ratio
- Blood tests (creatinine, K, Na, fasting plasma glucose, lipid profile, and often thyroid-stimulating hormone)
- ECG

Ambulatory BP monitoring, renal radionuclide imaging, chest x-ray, screening tests for pheochromocytoma, and renin-Na profiling are not routinely necessary. Peripheral plasma renin activity is not helpful in diagnosis or drug selection.

Depending on results of initial tests and examination, other tests may be needed. If urinalysis detects albuminuria (proteinuria), cylindruria, or microhematuria or if serum creatinine is elevated (≥ 1.4 mg/dL [$124 \mu\text{mol/L}$] in men; ≥ 1.2 mg/dL [$106 \mu\text{mol/L}$] in women), renal ultrasonography to evaluate kidney size may provide useful information. Patients with hypokalemia unrelated to diuretic use are evaluated for primary aldosteronism and high salt intake.

On ECG, a broad, notched P-wave indicates atrial hypertrophy and, although nonspecific, may be one of the earliest signs of hypertensive heart disease. Left ventricular hypertrophy, indicated by a sustained apical thrust and elevated QRS voltage with or without evidence of ischemia, may occur later. If either of these findings is present, echocardiography is often done. In patients with an abnormal lipid profile or symptoms of CAD, tests for other cardiovascular risk factors (eg, C-reactive protein) may be useful.

If coarctation of the aorta is suspected, chest x-ray, echocardiography, CT, or MRI helps confirm the diagnosis.

Patients with labile, significantly elevated BP and symptoms such as headache, palpitations, tachycardia, excessive perspiration, tremor, and pallor are screened for pheochromocytoma (eg, by measuring plasma free metanephrines). A sleep study should also be strongly considered.

Patients with symptoms suggesting Cushing syndrome, a connective tissue disorder, eclampsia, acute porphyria, hyperthyroidism, myxedema, acromegaly, or CNS disorders are evaluated.

Prognosis

The higher the BP and the more severe the retinal changes and other evidence of target-organ involvement, the worse is the prognosis. Systolic BP predicts fatal and nonfatal cardiovascular events better than diastolic BP. Without treatment, 1-yr survival is < 10% in patients with retinal sclerosis, cotton-wool exudates, arteriolar narrowing, and hemorrhage (grade 3 retinopathy), and < 5% in patients with the same changes plus papilledema (grade 4 retinopathy). CAD is the most common cause of death among treated hypertensive patients. Ischemic or hemorrhagic stroke is a common consequence of inadequately treated hypertension. However, effective control of hypertension prevents most complications and prolongs life.

General Treatment

- Weight loss and exercise
- Smoking cessation
- Diet: Increased fruits and vegetables, decreased salt, limited alcohol
- Drugs if BP is initially high (> 160/100 mm Hg) or unresponsive to lifestyle modifications

Primary hypertension has no cure, but some causes of secondary hypertension can be corrected. In all cases, control of BP can significantly limit adverse consequences. Despite the theoretical efficacy of treatment, BP is lowered to the desired level in only one third of hypertensive patients in the US.

JNC 8 recommends treatment targets:

- **For all patients**, including all those with a kidney disorder or diabetes, treatment aims to reduce BP to <140/90 mm Hg
- **For patients ≥ 60** , treatment aims to reduce BP to < 150/90 mm Hg

However, some clinicians believe that for patients ≥ 60 , the previous (JNC 7) target of 140/90 is still appropriate.

Even the elderly and frail elderly can tolerate a diastolic BP as low as 60 to 65 mm Hg well and without an increase in cardiovascular events. Ideally, patients or family members measure BP at

home, provided they have been trained to do so, they are closely monitored, and the sphygmomanometer is regularly calibrated. Treatment of hypertension during pregnancy requires special considerations because some antihypertensive drugs can harm the fetus.

Lifestyle modifications

Recommendations include regular aerobic physical activity at least 30 min/day most days of the week; weight loss to a body mass index of 18.5 to 24.9; smoking cessation; a diet rich in fruits, vegetables, and low-fat dairy products with reduced saturated and total fat content; dietary sodium [Na⁺] of < 2.4 g/day (< 6 g NaCl); and alcohol consumption of ≤ 1 oz/day [29.5 mL/day] in men and ≤ 0.5 oz/day [15 mL/day] in women. In stage 1 (mild) hypertension with no signs of target-organ damage, lifestyle changes may make drugs unnecessary. Patients with uncomplicated hypertension do not need to restrict their activities as long as BP is controlled. Dietary modifications can also help control diabetes, obesity, and dyslipidemias. Patients with prehypertension are encouraged to follow these lifestyle recommendations.

Drugs

If systolic BP remains > 140 mm Hg (> 150 mm Hg for patients ≥ 60) or diastolic BP remains > 90 mm Hg after 6 mo of lifestyle modifications, antihypertensive drugs are required. Unless hypertension is severe, drugs are usually started at low doses. Drugs are initiated simultaneously with lifestyle changes for all patients with hypertension plus diabetes, a kidney disorder, target-organ damage, or cardiovascular risk factors and for those with an initial BP of > 160/100 mm Hg. Signs of hypertensive emergencies require immediate BP reduction with parenteral antihypertensives.

For most hypertensive patients, one drug is given initially. For non-black patients, including those with diabetes, initial treatment should be with either an ACE inhibitor, angiotensin receptor blocker, Ca channel blocker, or a thiazide-like diuretic (chlorthalidone or indapamide). For black patients, including those with diabetes, a Ca channel blocker or a thiazide-like diuretic is recommended initially. Some antihypertensives are contraindicated in certain disorders (eg, β-blockers in asthma) or are indicated particularly for hypertensive patients with certain disorders (eg, Ca channel blockers for angina pectoris, ACE inhibitors or angiotensin II receptor blockers for diabetes with proteinuria).

If the target BP is not achieved in 1 mo, the dose of the initial drug can be increased or a second drug added (selected from the drugs recommended for initial treatment). Note that an ACE inhibitor and angiotensin receptor blocker should not be used together. Therapy is titrated frequently. If target BP cannot be achieved with 2 drugs, a third drug from the initial group is added. If such a third drug is not available (eg, for black patients) or tolerated, a drug from another class (eg, β-blocker, aldosterone antagonist) can be used. Patients with such difficulty to control BP may benefit from consultation with a hypertension specialist.

Devices and physical interventions

Percutaneous catheter-based radiofrequency ablation of the sympathetic nerves in the renal artery is approved in Europe and Australia for resistant hypertension.

Hypertension is defined as resistant when BP remains $> 160/100$ mm Hg despite use of 3 different antihypertensive drugs with complementary mechanisms of action (one of which being a diuretic). Although initial studies appeared promising, a recent large, double-blind study was done. This study for the first time incorporated a sham ablation procedure in the control arm and failed to show a benefit from radiofrequency ablation. Thus, sympathetic ablation should still be considered experimental and done only in European or Australian centers with extensive experience.

A second physical intervention involves stimulating the carotid baroreceptor with a device surgically implanted around the carotid body. A battery attached to the device, much like a pacemaker, is used to stimulate the baroreceptor and, in a dose-dependent manner, lower BP. This procedure has so far proven safe and effective, although experience is limited and trials are ongoing. This device is not yet approved for treatment of hypertension.

Key points:

- Only about three quarters of hypertensive patients in the US are being treated and only half are adequately controlled.
- Most hypertension is primary; only 5 to 15% is secondary to another disorder (eg, renal parenchymal or vascular disease, pheochromocytoma, Cushing syndrome, congenital adrenal hyperplasia, hyperthyroidism)
- Severe or prolonged hypertension damages the cardiovascular system, brain, and kidneys, increasing risk of MI, stroke, and renal failure.
- Hypertension is usually asymptomatic until complications develop in target organs.
- When hypertension is newly diagnosed, do urinalysis, spot urine albumin:creatinine ratio, blood tests (creatinine, K, Na, fasting plasma glucose, lipid profile, and often TSH), and ECG.
- Reduce BP to $< 140/90$ mm Hg for everyone < 60 , including those with a kidney disorder or diabetes.
- Reduce BP to $< 150/90$ mm Hg for everyone ≥ 60 .

- Treatment involves lifestyle changes, especially a low-sodium and higher potassium diet, methods to improve uninterrupted sleep duration, and drugs (including diuretics, ACE inhibitors, angiotensin II receptor blockers, and Ca channel blockers).

JNC 7 Classification of Blood Pressure in Adults

Classification	BP
Normal	< 120/80 mm Hg
Prehypertension	120–139/80–89 mm Hg
	140–159 mm Hg (systolic)
Stage 1	or
	90–99 mm Hg (diastolic)
	≥ 160 mm Hg (systolic)
Stage 2	or
	≥ 100 mm Hg (diastolic)

JNC= Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.

Initial Choice of Antihypertensive Drug Class

Drugs	Indications
	Old age
Thiazide- like diuretics* (chlorthalidone or indapamide)	Black race
	Heart failure
	Old age
	Black race
	Angina pectoris
Long-acting Ca channel blockers	Arrhythmias (eg, atrial fibrillation, paroxysmal supraventricular tachycardia)
	Isolated systolic hypertension in elderly patients (dihydropyridines)*
	High CAD risk (nondihydropyridines)*

Drugs

Indications

Youth

Left ventricular failure due to systolic dysfunction*

ACE inhibitors ‡

Type 1 diabetes with nephropathy*

Severe proteinuria in chronic renal disorders or diabetic glomerulosclerosis

Erectile dysfunction due to other drugs

Youth

Conditions for which ACE inhibitors are indicated but not tolerated because of cough

Angiotensin II receptor blockers ‡

Type 2 diabetes with nephropathy

Left ventricular failure with systolic dysfunction

Secondary stroke

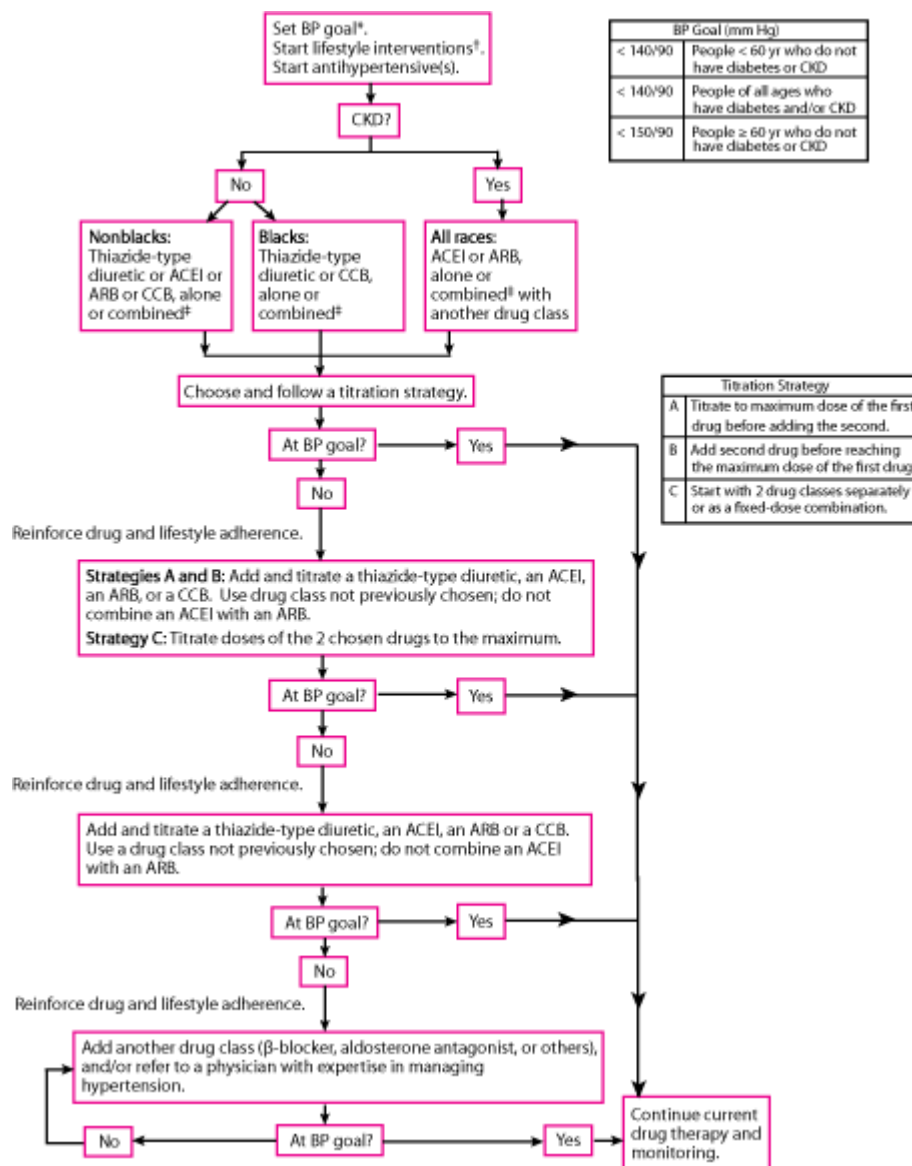
*Reduced morbidity and mortality rates in randomized studies.

† β -Blockers without intrinsic sympathomimetic activity.

‡ Contraindicated in pregnancy.

CAD = coronary artery disease.

Algorithm for treatment of hypertension in patients ≥ 18 yr.



*BP goal and drug therapy are based on age and the presence of diabetes and CKD.

Lifestyle interventions should be maintained throughout treatment.

ACEIs and ARBs should not be used together in the same patient.

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin II receptor blocker; CCB = calcium channel blocker; CKD = chronic kidney disease.

Data from James PA, Oparil S, Carter BL, et al: 2014 Evidence-based guideline for the management of high blood pressure in adults: Report from the panel members appointed to the Eighth Joint National committee (JNC 8). JAMA 311 (5): 507-520, 2014.

Antihypertensives for High-Risk Patients

Coexisting Condition

Drug Classes

ACE inhibitors

Heart failure

Angiotensin II receptor blockers

Coexisting Condition	Drug Classes
	β-Blockers
	K-sparing diuretics
Post-MI	Other diuretics*
	β-Blockers
	ACE inhibitors
Cardiovascular risk factors	Spirolactone, eplerenone
	β-Blockers
	ACE inhibitors
Diabetes	Diuretics
	Ca channel blockers
	Diuretics
Chronic kidney disorders	ACE inhibitors
	Angiotensin II receptor blockers
	Ca channel blockers
Risk of recurrent stroke	ACE inhibitors
	Angiotensin II receptor blockers
	ACE inhibitors, Angiotensin II receptor blockers
Risk of recurrent stroke	Ca channel blockers
	Diuretics

*Long-term diuretic use may increase mortality in patients with heart failure who do not have pulmonary congestion.

Last full review/revision May 2014 by George L. Bakris, MD

Self preparation at class:

Listen information;

Work with patients (with cardiac pathology);

Ask about the problems that have not been found in information given.

Self preparation at home:

Compose the plan of your answer;

Answer the questions to the topic; Do the test given above.

Question 1

A man aged 45 years with treated moderate hypertension left for a business trip. About 36 hours after leaving home he attended a casualty department complaining of headache, agitation, sweating and palpitations. The blood pressure was 220/145 mmHg. He was admitted and investigation showed a urinary catecholamine excretion of 15 μ mol/24 hours (274 mg/24 hours).

- (a) What was the differential diagnosis?
- (b) What hypotensive agent had he left at home ?
- (c) What is the treatment of this condition and with what drug ?

Question 2

A woman of 19 years was found to have a blood pressure of 180/125 mmHg. IVP normal; peripheral venous rennin 3490 pg ml⁻¹ h⁻¹; blood urea 12 mmol/l (72 mg/100ml); GFR 48 ml/min; urine microscopy no abnormality; proteinuria 1.9 g/day.

- (a) What was the diagnosis?

She was successfully treated with oral diazoxide and frusemide. One week later the peripheral venous renin was found to have approximately doubled in concentration and the GFR had fallen to 33 ml/min.

- (b) Comment upon the increase in the renin and the fall in GFR.
- (c) Comment upon the drug treatment.

Recommended literature:**I. Main:**

1. Internal Medicine: in 2 books. Book 1. Diseases of the Cardiovascular and Respiratory Systems: textbook / N.M. Seredyuk, I.P. Vakaliuk, R.I. Yatsyshyn et al. Київ, Медицина., 2019. - 664 + 48 кольор. вкл.).
2. Internal medicine: Part 1 (cardiology, rheumatology, haematology): textbook for English-speaking students of higher medical schools / edited by Professor M.A. Stanislavchuk and Professor V.A. Serkova. - Vinnytsia: Nova Knyha, 2019. - 392 p.
3. Медицина за Девідсоном: принципи і практика / Навчальний посібник: пер. 23-го англ. вид.: у 3 т. Т.3 С. Ралстона, Я. Пенмана, М. Стрекена, Р. Гобсона; К.: ВСВ «Медицина», 2021. – 642 с.
4. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)/
5. Побічна дія ліків – Side Effects of Medications: навчальний посібник 2 т. / заг. ред. В.М. Бобирьова, М.М. Потяженка. – Вінниця:
6. Cardiovascular diseases. Classification, standards of diagnosis and treatment / Edited by Academician Kovalenko V.M., Prof. Lutaia M.I., Prof. Sirenko Yu.M., Prof. Sychova O.S. – Kyiv. – 2020.
7. Perederii V.H., Tkach S.M. Principles of internal medicine. – Vol.2 / Textbook for students of higher educational institutions. – Vinnytsia: Nova knyha. – 2018.
8. Internal diseases. The textbook based on the principles of evidentiary medicine, 2018.

II. Additional literature:

1. Recommendations of the Association of Cardiologists of Ukraine for the diagnosis and treatment of chronic heart failure / Voronkov L.H. – moderator, working group of the Ukrainian Association of Heart Failure Specialists. – 2017.
2. Respiratory diseases / Ghanei M. - In Tech, 2012. - 242 p.
3. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
4. Principles and practice of interventional pulmonology / Ernst A., Herth F. -Springer, 2012. - 757 p.
5. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
6. Petrov Y. The chief symptoms and syndromes in patients with cardiovascular pathology : The practical handbook for medical students / Ye. Petrov, Yu. Goldenberg, N. Chekalina; UMSA. - Poltava : TexcepBic, 2010. - 143 .
7. Gastroenterology and Hepatology Board Review: Pearls of Wisdom, Third Edition (Pearls of Wisdom Medicine) by John K. DiBaise (May 11, 2012)
8. Clinical Pulmonology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Oct 30, 2011) - Kindle eBook
9. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Hematology: Clinical Principles and Applications, 4e by Bernadette F. Rodak MS MLS (Feb 18, 2017)
12. Rheumatology, 2-Volume Set: EXPERT CONSULT - ENHANCED ONLINE FEATURES AND PRINT, 5e by Marc C. Hochberg MD MPH, Alan J. Silman MD, Josef S. Smolen MD and Michael E. Weinblatt MD (Oct 19, 2019)
13. Endocrine Pathology: Differential Diagnosis and Molecular Advances by Ricardo V. Lloyd (Nov 5, 2018)
14. Clinical Endocrinology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2017) - Kindle eBook
15. Williams Textbook of Endocrinology: Expert Consult-Online and Print, 12e by Shlomo Melmed, Kenneth S. Polonsky MD, P. Reed MD Larsen and Henry M. Kronenberg MD (May 27, 2016)
16. Electrocardiography, 3e with Student CD (Booth, Electrocardiography for Health Care Personnel) by Kathryn A. Booth (Jan 27, 2017)
17. Echocardiography Review Guide: Companion to the Textbook of Clinical Echocardiography: Expert Consult: Online and Print, 2e (Expert Consult Title: Online + Print) by Catherine M. Otto (Mar 7, 2017).

Methodical recommendations consisted by

Kulishov S.K.