U2 Circulatory system disease module

U3 Infectious endocarditis

\*Reliable diagnostic criteria for infectious endocarditis

+positive blood culture

 fever

auscultatory picture

+detection of vegetation by echocardiography

 true A and D

# Reliable diagnostic criteria for infectious endocarditis

Lukin spots

heavy sweats

in. thromboembolic complications

accelerated ESR

+ d. reappeared valve regurgitation according to echocardiography

# Reliable diagnostic criteria for infectious endocarditis

 anemia

+ positive blood culture

focal glomerulonephritis

vasculitis

 growing symptoms of heart failure

#Reliable diagnostic criteria for infectious endocarditis

 detection of heart disease

leukocytosis

+ detection of vegetation on valves during ECHO KG study

Osler's nodules

 Spots of Janeway

\* Reliable diagnostic criteria for infectious endocarditis

fever duration

+ positive blood culture

. anemia

+ detection of vegetation on valves during ECHO KG study

 septic shock

\*Predisposing factors of infective endocarditis

+ transient bacteremia

+ hemodialysis

+ presence of artificial heart valves

infectious blood diseases

\*Infectious endocarditis may occur.

+ myocarditis

+ small vessel vasculitis

+ small vessel embolism with the development of abscesses

tamponade of the heart

hemorrhagic shock

\*Infectious endocarditis is possible

+. CNS damage

+ heart valve damage

+ embolism in various organs

+ renal failure

damage to the hematopoietic system

\*Infectious endocarditis is characterized by development

+ diffuse glomerulonephritis

+ heart attack kidney

+ focal nephritis

chronic pyelonephritis

\*With infectious endocarditis observed

 hepatitis

+ arthritis

 pleurisy

+ vasculitis

+ glomerulonephritis

\*With infectious endocarditis of the aortic valve, embolism is possible in

+ arteries of the kidneys

pulmonary arteries

+ spleen arteries

+ arteries of the brain

+ mesenteric arteries

\* Infectious endocarditis may cause

+ heart attack kidney

+ kidney abscess

+ diffuse glomerulonephritis

 nephropathy

D. nephrosclerosis

\*The cause of a negative result in blood culture in patients with infectious endocarditis may be

 gram negative cocci

+ wrong blood sampling technique

+ use of an insufficient set of environments

+ fungal nature of endocarditis

HACEK group bacteria

#The greatest probability of obtaining a positive blood culture result in a patient with infectious endocarditis is observed with blood sampling

+ 3-5 times in 24-48 hours after discontinuation of antibiotics

 8-10 times 12-24 hours after discontinuation of antibiotics

 during a period of fever, after discontinuation of antibiotics

repeatedly, amid rising temperatures

 once, before prescribing antibiotics

\*The prognosis in patients with infectious endocarditis is affected

+ sensitivity of microflora to antibiotics

localization of the pathological process

+ heart failure

+ antibiotic therapy start time

 hectic fever

#The tactics of antibiotic therapy for infectious endocarditis comes down to

+ immediate administration of large doses of antibiotics given the likely causative agent

prescribing antibiotics only after microflora research

 the use of reserve antibiotics

termination of penicillin in usual doses followed by an increase in dose

# Infectious endocarditis is recommended.

+ i/v administration of antibiotics

 v / m administration of antibiotics

 per os preparations

route of administration, the drug depends on the pathogen

the method of administration of the drug depends on the activity of the process

#Secondary infectious endocarditis develops in the background

pneumonia

bacteremia

embolism

+ valvular defect

 sepsis

# In patients with infectious endocarditis when receiving negative blood culture and primary culture

re-examination of blood is inappropriate

+ re-examination of blood within 48 hours is advisable

 before the second test, antibiotics are canceled for 2 days and blood is taken during an increase in body temperature

 repeated study on the background of antibiotic therapy

\*For patients with valvular prostheses, the prophylactic administration of antibiotics is advisable during

+ oral surgery

+ surgery on the heart and blood vessels

venous catheter manipulation

piercings and tattoos

#The cause of bacteremia with IE is most often

+ manipulations in the oral cavity (manipulations on the gum)

cystoscopy

gastroscopy

vein catheterization

conducting transesophageal echocardiography

# On the fundus of patients with infectious endocarditis are detected

Osler's nodules

+ mouth spots

Janeway spots

Lukin spots

#The most informative instrumental method of research in infectious endocarditis

+ transesophageal echocardiography

 ECG

ECHOKG

radionuclide ventriculography

Phonocardiography

# Vegetation for infectious endocarditis consists of

+ platelets, fibrin, bacteria, tissue detrin

fibrin, white blood cells, bacteria, microorganisms

ibactria, fibrin

 tissue detrin, fibrin, white blood cells

# With streptococcal infectious endocarditis with preserved sensitivity to penicillin prescribed

+ penicillin G

penicillin in combination with gentamicin

 ampicillin

Vancomycin

#When enterococcal infectious endocarditis is prescribed

+ amoxicillin + gentamicin

cephalosporins 2 and 3 generations

aminoglycosides

Macrolides

correct B and C

\*The main indications for the surgical treatment of infectious endocarditis

+ heart failure

+ repeated thromboembolism

+ the presence of abscesses of the fibrous ring

rhythm disturbance

staphylococcal nature of endocarditis

#With staphylococcal etiology of endocarditis, the most effective appointment

 ampicillin

oxacillin

 gentamicin and “protected” penicillins

+ vancomycin

Ceftriaxone

#Streptococcal etiology of endocarditis is suggested if symptoms of endocarditis occur after

wounds

 operations

+ oral manipulations

 obstetric manipulations

urogenital interventions

#Staphylococcal etiology of endocarditis is suggested if symptoms of endocarditis occur.

after wounds

after surgery

+ with furunculosis

drug addicts

\*Enterococcal etiology of endocarditis is suggested if symptoms of endocarditis occur after

wounds

with furunculosis

+ after urogenital interventions

+ after manipulation of the digestive tract

# For blood culture when suspected infectious endocarditis

 a single blood sample is usually sufficient

+ several blood samples taken during fever

getting a positive blood culture in at least two blood samples is enough for a diagnosis

 it is necessary to obtain a permanent blood culture in three blood samples

# Duration of antibiotic treatment for infectious endocarditis

2 weeks and less

 4-6 weeks

+ more than 6 weeks

more to 1 year

\*For the prevention of infectious endocarditis during manipulations in the oral cavity and on the upper respiratory tract, it is advisable to use

+ amoxicillin

 erythromycin

+ clindamycin

gentamicin

\*For the prevention of infectious endocarditis with urogenetic interventions, it is advisable to use

+ ampicillin

oxacillin

+ vancomycin

Gentamicin

# For the prevention of infectious endocarditis during manipulations accompanied by a risk of bacteremia, it is advisable to use

 oxacillin

erythromycin

ampicillin

+ g. the choice of antibiotic depends on the intended intervention, taking into account the possible microflora

#The cause of infectious endocarditis in drug addicts

+ staphylococci

streptococci

enterococci

fungi

Pseudomonas aeruginosa

# Treatments with a high risk of infectious endocarditis include

cystoscopy

gastroscopy

Colonoscopy

+ procedures on the root canal of the tooth

# High risk group for infectious endocarditis

  hypertrophic cardiomyopathy

+ surgically installed pulmonary shunt

implanted EX and / or CD

prior coronary artery bypass grafting

# High risk group for infectious endocarditis

surgically corrected atrial septal defect

 ventricular septal defect

prior rheumatic fever without valvular dysfunction

+ previous IE

congenital aortic stenosis

# Low risk group for infectious endocarditis

+ isolated atrial septal defect

mitral valve prolapse with regurgitation and (or) thickening of its cusps (myxomatous degeneration of the cusps of the mitral valve) congenital heart defects of the “blue type”

 acquired heart defects (including rheumatic)

# An isolated atrial septal defect belongs to the group of average risk of developing infectious endocarditis

+ mitral valve prolapse with regurgitation and (or) thickening of it

valves (myxomatous degeneration of the mitral valve cusps)

congenital heart defects "blue type"

acquired heart defects (including rheumatic)

# Prevention of Infectious endocarditis with antibiotics is not recommended

Patients with acquired heart defects (including rheumatic)

Infectious Endocarditis Patients

congenital heart defects "blue type"

+ atrial septal defect

# Infectious endocarditis in drug addicts is characterized by a defeat

           + intact tricuspid valve

             aortic clkpan

             pulmonary artery valves

              mitral valve

 \* Direct (large) echocardiographic criteria for infectious endocarditis are

             + vegetation

             + abscesses

            + the appearance of para-prosthetic fistulas

             stenosis of the left atrioventricular opening

# Small (additional) criteria for infectious endocarditis

+ previous heart disease or intravenous medication (including drugs)

repeated positive blood cultures

modified artificial valves according to echocardiography

the emergence of a new (increase in previous) regurgitation

# In acute IE, antibiotic chemotherapy should be prescribed

+ immediately

may be delayed for 24–48 hours

before receiving a positive blood culture

during the first day

# With the development of infectious endocarditis on the background of methicillin-resistant and vancomycin-resistant staphylococcus is prescribed

ciprofloxacin

rifampicin

+ daptomycin

Tetracycline

U2 Circulatory system disease module

U3 Symptomatic arterial hypertension

#WHAT IS IDENTIFIED OFTEN IN PATIENTS WITH ESSENTIAL HYPERTENSION AND NEUROGUMORAL DISORDERS:

increased uric acid content

hyperglycemia, hyperinsulinemia

decreased activity of sympathoadrenal system

 decreased activity of renin-aldosterone system

+ increased production of endothelin and decreased production of nitric monoxide.

# WHAT IS DETERMINED OFTEN IN PATIENTS WITH ESSENTIAL HYPERTENSION AND METABOLIC DISORDERS:

+increased content of uric acid, decreased activity of renin-aldosterone system

hyperglycemia, hyperinsulinemia

decreased activity of the sympathoadrenal system

increased production of endothelin and decreased production of nitric monoxide

# WHAT IS DETERMINED OFTEN IN PATIENTS WITH ESSENTIAL HYPERTENSION AND METABOLIC DISORDERS:

decreased level of natriuretic peptides in blood

+decreased activity of kallikreinkinin system

dyslipidemia

increased level of alpha cholesterol

#WHAT IS DETERMINED OFTEN IN PATIENTS WITH ESSENTIAL HYPERTENSION AND METABOLIC DISORDERS:

decreased level of natriuretic peptides in blood

decreased activity of kallikreinkinin system

+dyslipidemia

increased level of alpha cholesterol

#OPTIMAL LEVEL OF ARTERIAL PRESSURE (MMHG) ACCORDING TO CLASSIFICATION OF WORLD HEALTH ORGANIZATION IS:

SBP below 140, diastolic - below 90

SBP below 120, diastolic - below 85

+SBP below 120, diastolic - below 80

SBP 140-150, diastolic - 94-100

SBP 160-180, diastolic - 94-100

#GRADE 1 AH (MMHG) ACCORDING TO CLASSIFICATION OF WORLD HEALTH ORGANIZATION IS:

SBP below 140, diastolic - below 90

SBP below 120, diastolic - below 85

SBP below 120, diastolic - below 80

SBP 140-150, diastolic - 94-100

+SBP 140-159, diastolic - below 90-99

 # GRADE 2 AH (MMHG) ACCORDING TO CLASSIFICATION OF WORLD HEALTH ORGANIZATION IS:

SBP below 140, diastolic - below 90

SBP below 120, diastolic - below 85;

SBP below 120, diastolic - below 80

+SBP 160-179, diastolic - 100-109

SBP 160-180, diastolic - 94-100

#NORMAL LEVEL OF ARTERIAL PRESSURE (MMHG) ACCORDING TO CLASSIFICATION OF WORLD HEALTH ORGANIZATION IS:

SBP below 140, DBP below 90

+SBP below 130, diastolic - below 85

SBP above 200, diastolic - above 110

SBP above 180, DBP above 110

#GRADE 3 AH (MMHG) ACCORDING TO CLASSIFICATION OF WORLD HEALTH ORGANIZATION IS:

SBP below 140, diastolic - below 90

SBP below 130, diastolic - below 85

SBP above 200, diastolic - above 110

+SBP above 180, diastolic - above 110

SBP above 160, diastolic - below 80

#ISOLATED AH ACCORDING TO CLASSIFICATION OF WORLD HEALTH ORGANIZATION IS:

SBP below 140, diastolic - below 90

SBP below 130, diastolic - below 85

SBP above 200, diastolic - above 110

SBP above 180, diastolic - above 110

+SBP above 140, diastolic - below 90

#SIGNS INDICATING HEART DISEASE IN AH:

+diameter of cavity of left atrium - 4.8 cm

thickness of interventricular septum -10 mm

height of R-wave in V1 - 30 mm

ratio of arteries and veins diameter of retina 1: 1

narrowing of lumen of carotid arteries by 30%

#SIGNS INDICATING DAMAGE OF ARTERIES IN AH:

paroxysms of atrial fibrillation;

+narrowing of left carotid artery by 30%

albuminuria 100 mg per day

proteinuria 400 mg per day.

#SIGNS INDICATING KIDNEY DAMAGE IN AH:

dysuric disorders

polydipsia, polyuria

hypokalemia

+albuminuria 100 mg per day

#FACTORS Predisposing to onset of hypertensive disease:

+age

amount of sodium chloride consumed

amount of sodium chloride consumed

psychosocial stress

alcohol abuse

#Potassium-sparing diuretics include:

ethacrylic acid

chlortalidone

furosemide

+4) spironolactone

Triamterenum

#LOOP DIURETICS:

+act in upward section of Henle loop

increase excretion of the body mainly K, Cl

severity of diuretic effect depends on blood aldosterone

inhibit carbonic anhydrase

increase renal blood flow

#FACTORS INCREASING ARTERIAL PRESSURE: A. AGE; B. GENETIC FACTORS; C. OBESITY; D. GROWTH; E. SODIUM CHLORIDE CONSUMPTION; F. MAGNESIUM AND IRON CONSUMPTION; G. PSYCHOSOCIAL OVERLOADS; H. ALCOHOL ABUSE:

true A, C, D, E

+true C, E, F, H

true A, B, D

true A, B, C

#BASIC HEMODYNAMIC FACTORS DETERMINING LEVEL OF ARTERIAL PRESSURE: A. FREQUENCY OF HEART RATE; B. cardiac output; c. VASCULAR RESISTANCE; D. atrial natriuretic peptides; E. CORTICOSTEROIDS; F. ENDOTELIN; g. prostacyclin, BRADIKININ; H. NITRIC OXIDE; I. Catecholamines, angiotensin:

+true A, B, C

) true H, G

true A, B, C, D

true A, B, D

#FACTORS RISING LEVEL OF ARTERIAL PRESSURE:

atrial natriuretic peptides

+endothelin

prostacyclin

bradykinin

nitric oxide

#BASIC HEMODYNAMIC FACTORS DETERMINING DIASTOLIC ARTERIAL PRESSURE LEVEL:

heart rate

cardiac output

+state of vascular tone

circulating blood volume

#FACTORS RISING LEVEL OF ARTERIAL PRESSURE:

+endothelin

prostacyclin

bradykinin

nitric oxide

#FACTORS REDUCING LEVEL OF ARTERIAL PRESSURE:

+atrial natriuretic peptides

corticosteroids

endothelin

catecholamines,

angiotensin

HUMORAL FACTORS DETERMINING TONUS OF VASCULAR WALL:

adenosine triphosphate

endothelin

nitrogen monoxide

+angiotensin

#FACTORS DETERMINING TONUS OF VASCULAR WALL

adenosine triphosphate

adrenaline, norepinephrine

+bradykinin

natriuretic peptides

#MAIN FACTORS WITH NEGATIVE INFLUENCE ON HEART (MYOCARDIAL HYPERTROPHY) IN PATIENTS WITH HYPERTENSION DISEASE:

organ damage is more correlated with SBP

+organ damage is more correlated with DBP.

angiotensin I

level of bradykiniN

#MAIN FACTORS WITH NEGATIVE INFLUENCE ON VESSELS (REMODELING) IN PATIENTS WITH HYPERTENSION DISEASE:

+endothelin

angiotensin I

absence of decrease in BP at night time during daily monitoring

ctivity of sympathoadrenal system

27. VASCULAR COMPLICATIONS, ASSOCIATED WITH AH IN PATIENTS:

ventricular tachycardia

sudden cardiac death

+acute encephalopathy

pulmonary embolism

#VASCULAR COMPLICATIONS IN PATIENTS WITH AH RELATED TO ARTERY ATHEROSCLEROSIS:

acute encephalopathy

hemorrhagic stroke

+ventricular tachycardia, sudden cardiac death

pulmonary edema in patient with left ventricular ejection function of 50%

#VASCULAR COMPLICATIONS, ASSOCIATED WITH AH IN PATIENTS:

atrial fibrillation

myocardial infarction

ischemic stroke

+nephrosclerosis

intermittent claudication

#VASCULAR COMPLICATIONS IN PATIENTS WITH ARTERIAL HYPERTONIA ASSOCIATED WITH ARTERY ATHEROSCLEROSIS: A. Atrial fibrillation; B. Myocardial infarction; C. ischemic stroke; D. nephrosclerosis; E. intermittent claudication:

+true A, B, C, E

true A, E

true C, D

true A, B, C, D, E

#REASONS FOR ISOLATED SYSTOLIC HYPERTENSION AS INDEPENDENT DISEASE:

aortic valve insufficiency

arteriovenous fistula

Paget's disease

systolic hypertension in young

+systolic hypertension in elderly

#REASONS FOR ISOLATED SYSTOLIC HYPERTENSION AS SYMPTOM OF ANOTHER DISEASE:

+aortic valve insufficiency;

aortic valve stenosis

subaortic stenosis

pulmonary artery stenosis

#INDICATIONS FOR HOSPITALIZATION OF PATIENTS WITH AH:

AH detected in patient under 20 years old

+refractory hypertension to combinated therapy

isolated systolic hypertension

masked hypertension

WHAT RISK FACTORS INFLUENCING FORECAST SHOULD BE TAKEN INTO ACCOUNT FOR RISK STRATIFICATION IN PATIENTS WITH AH:

+grade of increase in BP (1-3 grade)

women in menopause

stroke, transient ishemic attack

painless depression of ST segment detected on ECG Holter

# WHAT END-ORGAN DAMAGE INFLUENCING FORECAST SHOULD BE TAKEN INTO ACCOUNT FOR RISK STRATIFICATION IN PATIENTS WITH AH:

grade of increase in BP (1-3 grade)

men - over 55 years old, women - over 65 years old

overweight;

+narrowing of arteries of retina

#WHAT END-ORGAN DAMAGE INFLUENCING FORECAST SHOULD BE TAKEN INTO ACCOUNT FOR RISK STRATIFICATION IN PATIENTS WITH AH:

content of total cholesterol in blood is more than 6.5 mmol

content of total cholesterol in blood is more than 5.2 mmol

+left ventricular hypertrophy;

congestive heart failure

# REDUCING BP LEVEL TO NORMAL LEVEL IN PATIENTS WITH ITS ASYMMETRY ON RIGHT AND LEFT HANDS IS DANGEROUS BECAUSE OF:

development of coronary thrombosis

development of acute aneurysm of thoracic aorta

impaired renal function

+development of ischemic stroke and myocardial infarction

38. ABSOLUTE DIAGNOSTIC CRITERION OF AH AT PHEOCHROMOCYTOMA IS:

+presence of signs of adrenal tumor and overproduction of catecholamines

increased plasma concentration of aldosterone

high level in urine of 5-hydroxyindoleacetic acid

low levels of catecholamines in blood flowing through renal veins, and its concentration in urine

#HORMONE WITH HIGH PRESSIVE ACTIVITY IS:

calcitonin

+adrenaline

insulin

aldosterone

prolactin

#ENDOGENOUS REASON FOR DEVELOPMENT OF HYPERTENSION CRISIS IS:

acute cerebral ischemia with sharp decrease in BP

sudden withdrawal of antihypertensive drugs

resuscitation during and after operations

excess salt intake

+disorders of urodynamics in prostate adenoma

#EXOGENOUS FACTOR CONTRIBUTING TO DEVELOPMENT OF HYPERTENSIVE CRISIS: IS

exacerbation of coronary heart disease

sharp violation of renal hemodynamics

+psycho-emotional stress

sickle cell crisis

sharp violation of renal hemodynamics

 #HYPERTONIC CRISES COMPLICATION IS:

 chronic coronary syndrome

 acute right ventricular failure

  +stroke

 thromboembolic syndrome

 aortic aneurysm

#HOSPITALIZATION IS required AT:

+newly detected uncomplicated hypertonic crisis

increased SBP> 180 mmHg

nausea, repeated vomiting

dizziness

#THE MOST QUICK REDUCTION OF BP IS NECESSARY TO CARRY OUT IN PATIENT WITH HYPETONIC CRISIS, COMPLICATED BY:

hemorrhagic stroke

+dissecting aortic aneurysm

myocardial infarction

atrial tachycardia

ischemic stroke

#OPTIMAL TIME TO ACHIEVE TARGET BP IN HYPERTENSIVE CRISIS, COMPLICATED BY ACUTE LEFT VENTRICULAR INSUFFICIENCY IS:

+no more than 20 minutes

within 2 hours

within 1 hour

do not reduce BP <160 | 90

within 12-24 hours

#REFRACTORy (RESISTANT) AH IS CONDITION WHEN TREATMENT USING THREE DRUGS

including peripheral α-blocker, does not allow to reduce SBP and DBP less than 140 and 90 mmHg respectively

+including diuretic, does not allow to reduce SBP and DBP less than 140 and 90 mmHg respectively

including central α-adrenergic agonist, does not allow to reduce SBP and DBP less than 140 and 90 mmHg respectively

including diuretic, does not allow to reduce SBP and DBP less than 130 and 80 mmHg respectively

#ADDITIONAL RECOMMENDATION FOR PATIENTS WITH REFRACTORy AH IS:

direct renin inhibitor

surgical treatment

imidazoline receptor blocker

peripheral α-blocker

+spironolactone

#SURGICAL TREATMENT OF AH APPLICABLE IN RUSSIA IS:

stimulation of carotid sinus baroreceptors

formation of ileofemoral arteriovenous fistula

+radiofrequency renal artery denervation

denervation of carotid sinus

#DRUG OF CHOICE IN ACUTE HYPERTENSIVE ENCEPHALOPATHY IS:

diuretic (furosemide)

alpha-blocker (urapidil).

+sodium nitroprusside

nitroglycerin

antipsychotics (droperidol)

#DRUG OF CHOICE IN ACUTE LEFT VENTRICULAR INSUFFICIENCY ON THE BACKGROUND OF HYPERTENSIVE CRISIS IS:

+nitroglycerin

β-blocker (metoprolol, esmolol)

alpha-blocker (urapidil)

ACE inhibitors (enalaprilat)

antipsychotics (droperidol)

#WHAT IT IS PREFERRED FOR AORTIC DISSECTION AS COMPLICATION OF HYPERTENSIVE CRISIS:

nitroglycerin

+β-blockers

sodium nitroprusside

alpha blockers

diuretics

#WHAT IT IS PREFERRED FOR ACUTE CORONARY SYNDROM AS COMPLICATION OF HYPERTENSIVE CRISIS:

sodium nitroprusside

alpha-blockers

diuretics

+β-blockers (metoprolol, esmolol)

#In uncomplicated hypertensive crises speed reduction of BP:

should not exceed 25% for first 6 hours

should achieve target BP within 2 hours

should achieve target BP within 20-30 minutes

+should not exceed 25% in first 2 hours

#GESTATIONAL AH IS:

a condition caused by pregnancy and manifested by increase level of BP ≥120/80 mmHg after 20th week of pregnancy

increase BP ≥140/90 mmHg determined before pregnancy

+condition caused by pregnancy and manifested by increase BP level ≥140/90 mmHg after 20th week of pregnancy, disappearing within 6-12 weeks after childbirth

increase BP ≥140/90 mmHg determined before pregnancy and does not disappear within 6-12 weeks after childbirth

#PREECLAPSY IS CHARACTERIZED BY DAILY PROTEINURIA:

30 mg/l

+300 mg/l

300 mg/dl

200 mg/dl

CRITERIA FOR SEVERE PRE-CLAMPSIA ARE:

 BP≥170/110 mmHg

proteinuria 5.0 g/l, impaired renal function

+BP ≥160/110 mmHg, proteinuria 5.0 g/l, impaired function of kidney and liver

thrombocytopenia, hemolysis

#DRUG OF CHOICE FOR AH IN PREGNANCY IS:

+methyldopa

direct renin inhibitors

angiotensin converting enzyme inhibitors

angiotensin receptor blocker

non-dihydropyridine calcium antagonists

#WHAT TACTICK IS REQUIRED FOR PATIENTS WITH ACUTE ISCHEMIC STROKE AND BP > 220/120 MMHG WITHOUT THROMBOLITIC THERAPY

+it is recommended to reduce BP by 15% during first 24 hours

after onset of stroke

reducing BP is not recommended

it is recommended to reduce BP by 25% during first 24 hours

after onset of stroke

it is recommended to reduce BP to target level within first 24 hours after onset of stroke

immediate AHT is recommended

#FOR PATIENTS WITH AH AND TRANSITOR-ISCHEMIC ATTACK

it is recommended to reduce BP by 15% during first 24 hours

after onset of stroke

reducing BP is not recommended

it is recommended to reduce BP by 25% during first 24 hours

after onset of stroke

it is recommended to reduce BP to target level within first 24 hours after onset of stroke

+immediate AHT is recommended

#WHAT TACTICK IS REQUIRED FOR PATIENTS WITH ACUTE ISCHEMIC STROKE AND AH USING THROMBOLITIC THERAPY

it is recommended to reduce BP by 15% during first 24 hours

after thrombolysis

it is recommended to reduce BP by 25% during first 24 hours

after thrombolysis

reducing BP is not recommended

+it is recommended to reduce and maintain BP less than 180/105 mmhHg during first 24 hours after thrombolysis

immediate AHT is recommended

#IN PATIENTS WITH ACUTE INTRACEREBRAL HAEMORRHAGE WITH SBP <220 MMHG:

it is recommended to reduce BP by 15% during first 24 hours

+immediate decrease BP is not recommended

it is recommended to reduce BP by 25% during first 24 hours

it is recommended to reduce and maintain BP less than 180/105 mmHg during the first 24 hours

immediate AHT is recommended

#IN PATIENTS WITH ACUTE INTRACEREBRAL HAEMORRHAGE WITH SBP <220 MMHG:

it is recommended to reduce BP by 15% during the first 24 hours

+it is recommended to lower BP to less than 180 mmHg in 4.5 hours

it is recommended to reduce BP by 25% during the first 24 hours

immediate decrease BP is not recommended

immediate antihypertensive therapy is recommended

#IN ALL PATIENTS WITH AH FOR STROKE PREVENTION … ARE RECOMMENDED:

+1) blocker of RAS, CCBs or thiazide/thiazide-like diuretic

blocker of renin-angiotensin-system, ACE inhibitor or thiazide/thiazide-like diuretic

β-blocker, calcium channel blockers or thiazide/thiazide-like diuretic

an imidazoline receptor blocker, CCBs or thiazide/thiazide-like diuretic

imidazoline receptor blocker, ACE inhibitor or thiazide/thiazide-like diuretic

#IF THERE ARE SYMPTOMS OF INCREASED SYMPATHOADRENAL ACTIVITY AND HYPERTENSIVE CRISIS, THE DRUG OF CHOICE IS:

thiazide/thiazide-like diuretic

calcium channel blocker

+imidazoline receptor blocker

ACE inhibitors

blocker of renin-angiotensin-system

#IN ABSENCE OF INCREASED SYMPATHOADRENAL ACTIVITY IN HYPERTENSIVE CRISIS, THE DRUG OF CHOICE IS:

thiazide/thiazide-like diuretic

calcium channel blocker

imidazoline receptor blocker

+ACE inhibitors

blocker of renin-angiotensin-system

#IN PATIENTS WITH AH AND PERIPHERAL ATHEROSCLEROSIS, IT IS RECOMMENDED

 combination of RAS blocker with calcium channel blocker

+combination of RAS blocker with ACE inhibitor

β-blocker

imidazoline receptor blocker

clonidine

**U2 Circulatory system disease module**

**U3 Symptomatic arterial hypertension**