Ryazan State Medical University
named after academician I.P. Pavlov

Department of faculty therapy

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FACULTY THERAPY
IN SCHEMES AND TABLES

manual
for students of general medicine faculty

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The manual is intended for 4th year students of general medicine faculty, contains information on classifications, etiopathogenesis, clinical picture and algorithms for the treatment of major diseases, studied in the discipline "Faculty therapy". The material is presented in the form of schemes and tables that will help in the study of educational material.

Fig. 74; Tab. 42

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О.М. Урясьев, А.В. Соловьева

ФАКУЛЬТЕТСКАЯ ТЕРАПИЯ В СХЕМАХ И ТАБЛИЦАХ

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У 739 Факультетская терапия в схемах и таблицах: учебное пособие обучающихся по специальности Лечебное дело / О.М. Урясьев, А.В. Соловьева; ФГБОУ ВО РязГМУ Минздрава России. – Рязань: ОТСиОП, 2020. – 117 с.

Учебное пособие предназначено для студентов 4 курса, обучающихся по специальности 31.05.01 «Лечебное дело», содержит информацию по классификациям, этиопатогенезу, клинической картине и алгоритмам лечения основных заболеваний, изучаемым по дисциплине "Факультетская терапия". Материал представлен в виде схем и таблиц, что поможет в изучении учебного материала.

Илл. 74; табл. 42

УДК 616(075.8)
ББК 54.1

CONTENT

INTRODUCTION

The manual "Faculty therapy in schemes and tables" is intended for 4th year students of general medical faculty. Presentation of educational material in the form of schemes, tables and figures will help to assimilate the etiology, pathogenesis, clinical symptoms, algorithms for diagnosis and treatment of the main nosological forms studied in the discipline "Faculty therapy". The textbook is based on modern Clinical recommendations of cardiological, pulmonological and gastroenterological communities. The clinical tasks presented at the end of the manual demonstrate typical clinical cases of therapeutic diseases and contribute to the consolidation of the educational material.

ABBREVIATIONS

AAT - Alpha antitrypsin
Ab - Antibody
ACC - American College of Cardiology
ACE - Angiotensin-converting enzyme
ACO - Asthma-COPD overlap
AHA - American Heart Association
ALT - Alanine aminotransferase
ANA - Antinuclear antibodies
ANCA - Anti-neutrophil cytoplasmic antibodies
ARF - Acute rheumatic fever
ASG - Antistreptohyaluronidase
ASK - Antistreptokinase
ASO - Antistreptolysin-O
AST - Aspartate aminotransferase
BB - Beta-blockers
BD – Bronchodilator
BMI - Body mass index
BNP - B-type natriuretic peptide
BP - Blood pressure
BPM - Beats per minute
BR – Breathing rate
BSA - Body surface area
CABG - Coronary artery bypass graft
CAD - Coronary artery disease
CB1R antagonist – Cannabinoid 1 receptor antagonist
CBC - Complite blood count
CHD - Congenital heart disease
CK - Creatine kinase
CKD - Chronic kidney disease
COPD - Chronic obstructive pulmonary disease
CRP - C-reactive protein
CT - Computed tomography
CTGF – Connective tissue growth factor
CV – Cardiovascular
CVD - Cardiovascular diseases
DBP - Diastolic blood pressure

DDI - Drug–drug interactions
DHP-CCB - Dihydropyridine calcium channel blockers
DM - Diabetes mellitus
ECG - Electrocardiogram
EchoCG - Echocardiography
EF - Ejection fraction
ESC - European Society of Cardiology
ESR - Erythrocyte sedimentation rate
ET-1 antagonist – Endothelin-1 antagonist
FDG - Fluorodeoxyglucose
FEV_1 - Forced expiratory volume in one second
FVC - Forced vital capacity
FXR agonist - Farnesoid X receptor agonist
GAS - Group A streptococcal infection
GERD - Gastroesophageal reflux disease
GFR- estimated glomerular filtration rate
GOLD - Global Initiative for Obstructive Lung Disease
H. paraphrophilus, H. influenzae, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae, and K. denitrificans;
HACEK - Haemophilus parainfluenzae, H. aphrophilus,
Hb - Hemoglobin
HbAlc – Glycated hemoglobin
HBV – Hepatitis B virus
HCl - Hydrochloric acid
HCV – Hepatitis C virus
HDL - High density lipoproteins
HDL-C - HDL cholesterol
HF - Heart failure
HFpEF - heart failure with preserved ejection fraction
HIV - Human immunodeficiency virus
HMOD - hypertension-mediated organ damage
HSC – Hematopoietic stem cell
HT - Hypertension
ICD - International Classification of Diseases
ICS – Inhaled corticosteroids
ICU - Intensive care unit
IE - Infective endocarditis

Ig - Immunoglobulin
IL - Interleukin
INR - International nomalized relation
iv - intravenous
LA- Left atrium
LABA – long-acting beta agonists
LAD – Left anterior descending artery
LAMA - Long-acting muscarinic antagonist
LAN - Long-acting nitrates
LCX – HLft circumflex artery
LDH - Lactate dehydrogenase
LDL - Low density lipoproteins
LMWH - Low Molcular Weight Heparins
LOXL2 mAb – Lysyl Oxidase Homolog 2 Antibody
LTRA – Leukotriene receptor antagonist
LV- Left ventricle
LVH - Left ventricular hypertrophy
MI - Myocardial infarction
MRCP - Magnetic resonance cholangiopancreatography
MRSA - Methicillin-resistant Staphylococcus aureus
NAFLD - Non-alcoholic fatty liver disease
NASH - Non-alcoholic steatohepatitis
NF-KB – Nuclear factor kappa-light-chain-enhancer of activated B-cells
NK cell – Natural killer cell
Non DHP-CCB – non Dihydropyridine calcium channel blockers
NSAIDs - Nonsteroidal anti-inflammatory drugs
NT-proBNP - N-terminal pro-B type natriuretic peptide
OCS – Oral corticosteroids
OGTT - Oral Glucose Tolerance Test
PCI - Percutaneous coronary intervention
PDE4 - phosphodiesterase type 4
PET - Positron emission tomography
PPAR-γ agonist - Peroxisome proliferator-activated receptor-γ agonists
PPI - Proton pump inhibitor
PPI - Proton pump inhibitors
PWV - pulse wave velocity

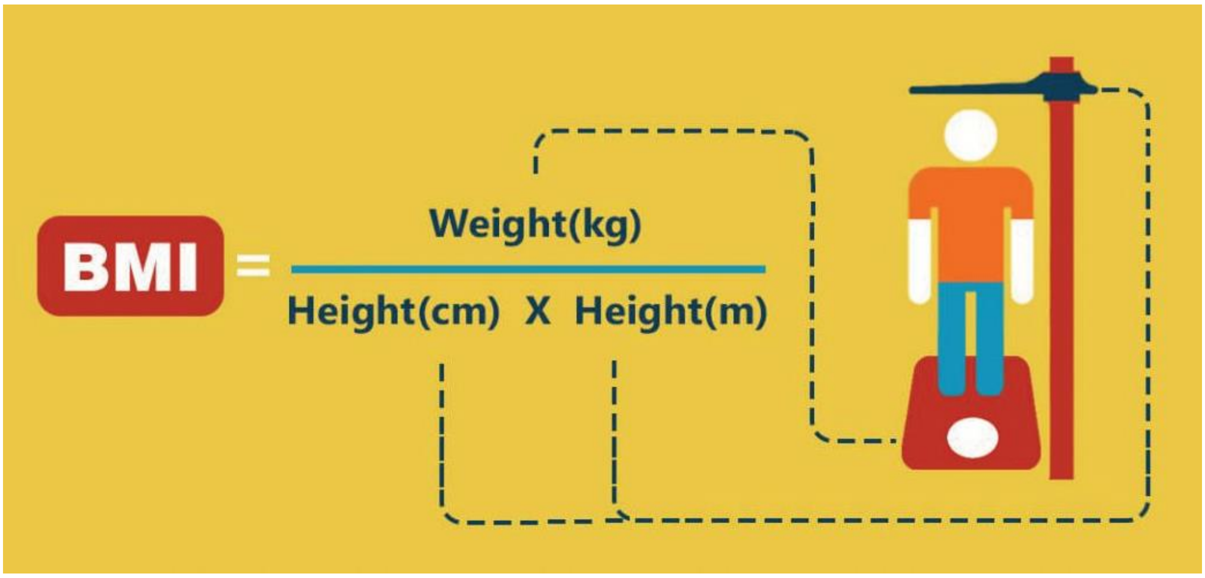


Fig. 2. Body mass index (BMI) calculation

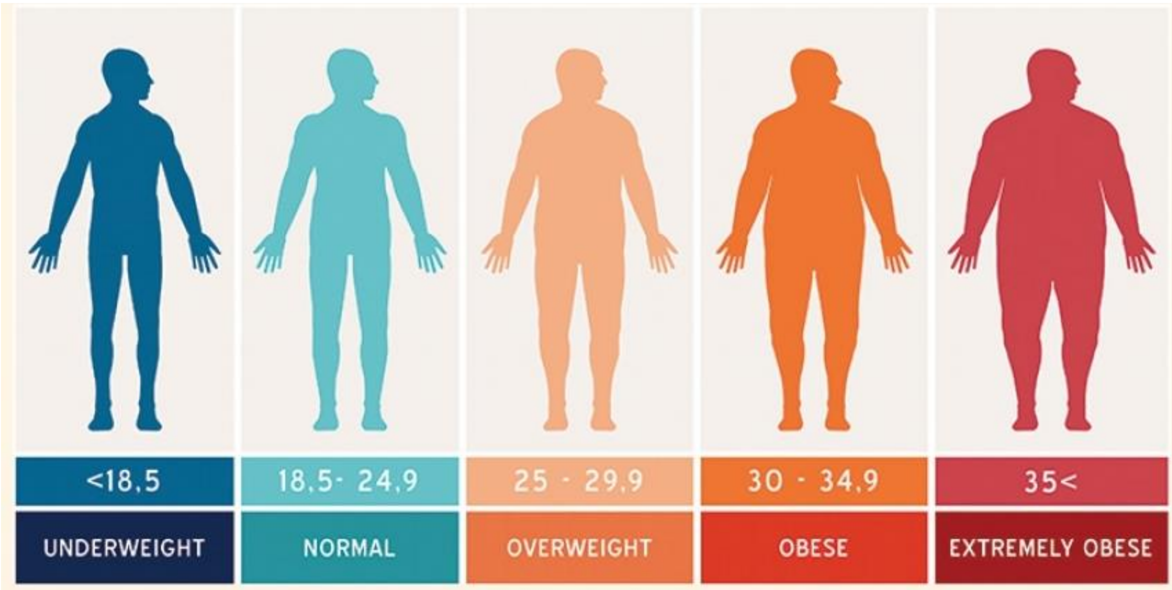


Fig. 3. Degrees of obesity according to BMI

Fig. 4. Scale SCORE (Systematic COronary Risk Evaluation)

Table 2.

Ten years cardiovascular risk categories

Very high risk	People with any of the following:
	Documented CVD, either clinical or unequivocal on imaging. • Clinical CVD includes myocardial infarction, acute coronary syndrome, coronary or other arterial revascularization, stroke, TIA, aortic aneurysm, and PAD • Unequivocal documented CVD on imaging includes significant plaque (i.e. ≥50% stenosis) on angiography or ultrasound; it does not include increase in carotid intima-media thickness • Diabetes mellitus with target organ damage (proteinuria) or with a major risk factor such as grade 3 hypertension or hypercholesterolaemia • Severe CKD (eGFR <30 mL/min/1.73 m^2) • 10 year SCORE of ≥10%
High risk	People with any of the following: • Marked elevation of a single risk factor, particularly cholesterol >8 mmol/L (familial hypercholesterolaemia) or grade 3 hypertension • Most other people with DM
	Hypertensive LVH
	Moderate CKD (eGFR 30-59 mL/min/1.73 m2)
	10 year SCORE of 5-10%
Moderate risk	People with: • A calculated 10 year SCORE of 1 to <5% • Grade 2 hypertension • Many middle-aged people belong to this category
Low risk	People with: • 10 year SCORE of <1%

eGFR - estimated glomerular filtration rate; LVH - left ventricular hypertrophy; TIA - transient ischaemic attack; PAD - peripheral artery disease; CKD - chronic kidney disease; CVD - cardiovascular disease;

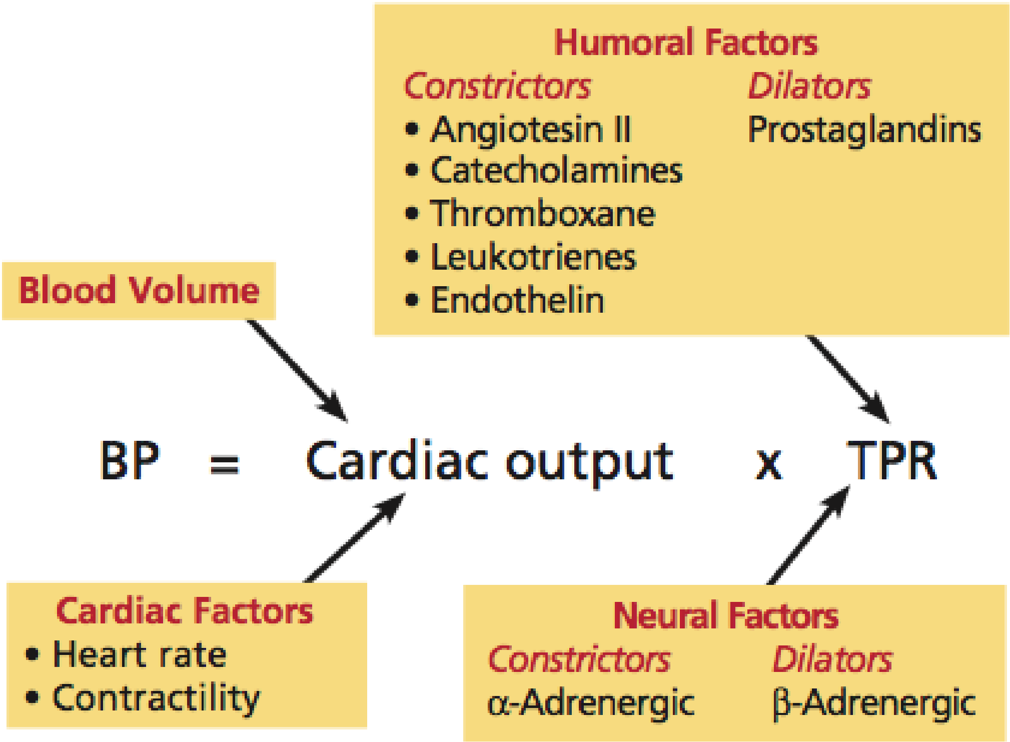


Fig. 5. Pathophysiology of hypertention.
TPR – total peripheral resistance.

Table 3.

Definitions and classification of office blood pressure (mmHg)

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension	≥140	and	<90

Table 4.

Classification of hypertension stages

Hypertension disease staging	Other risk factors, HMOD, or disease	BP (mmHg) grading			
		High normal SBP 130–139 DBP 85–89	Grade 1 SBP 140–159 DBP 90–99	Grade 2 SBP 160–179 DBP 100–109	Grade 3 SBP 180 or DBP 110
Stage 1	No other risk factors	Low risk	Low risk	Moderate risk	High risk
	1 or 2 risk factors	Low risk	Moderate risk	Moderate to high risk	High risk
	3 risk factors	Low to Moderate risk	Moderate to high risk	High Risk	High risk
Stage 2	HMOD, CKD grade 3, or DM without organ damage	Moderate to high risk	High risk	High risk	High to very high risk
Stage 3	Established CVD, CKD grade 4, or DM with organ damage	Very high risk	Very high risk	Very high risk	Very high risk

Table 5.

Blood pressure thresholds for the definition of hypertension with different types of BP measurement

Category	Systolic BP (mmHg)		Diastolic BP (mmHg)
Office BP	≥140	and/or	≥90
≥80 years of age	≥160	and/or	≥90
Ambulatory BP			
Daytime (or awake)	≥135	and/or	≥85
Night-time (or asleep)	≥120	and/or	≥70
24 h	≥130	and/or	≥80
Home BP	≥135	and/or	≥85

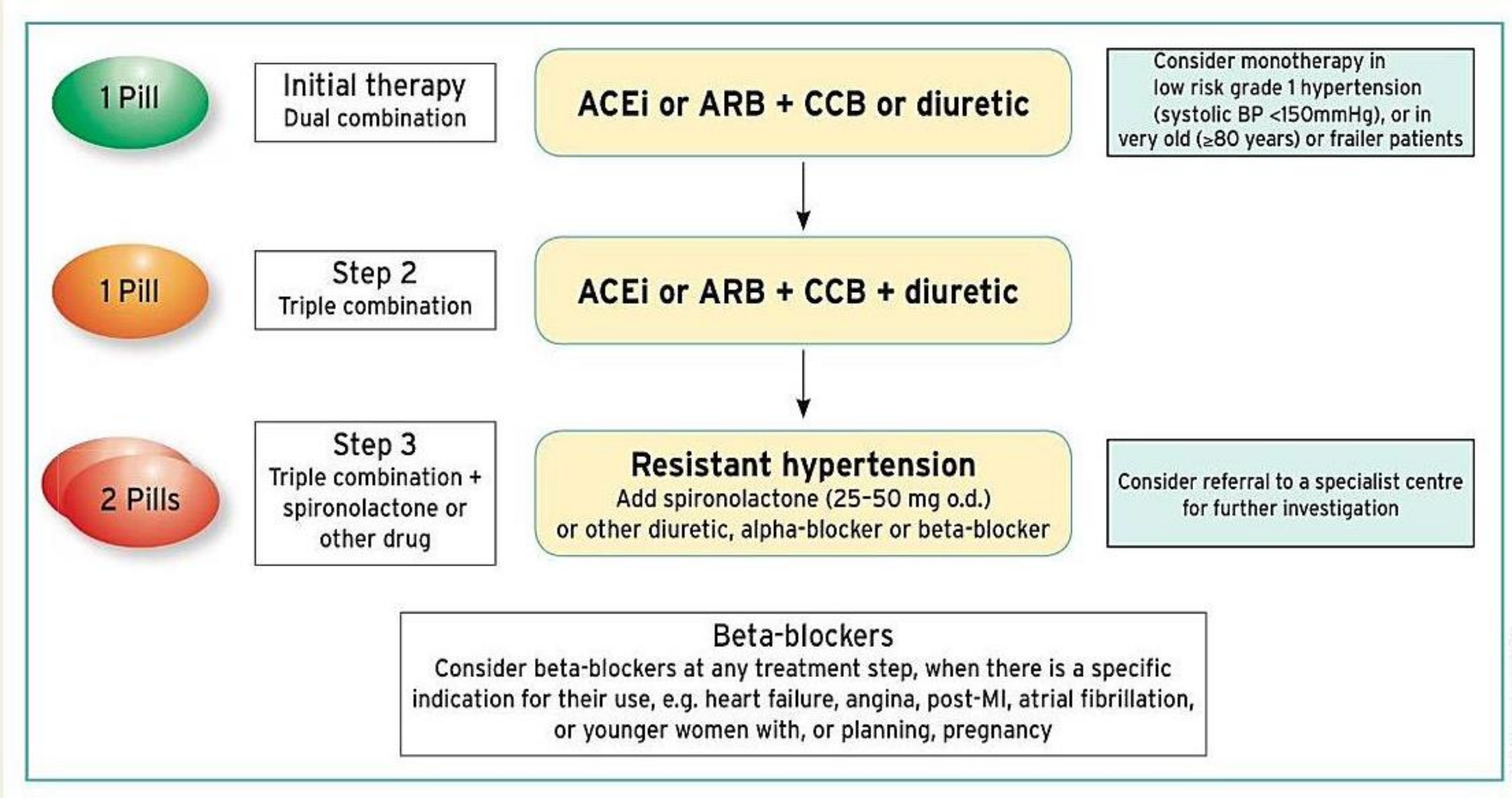


Fig. 6. Pharmacological treatment strategy for hypertension

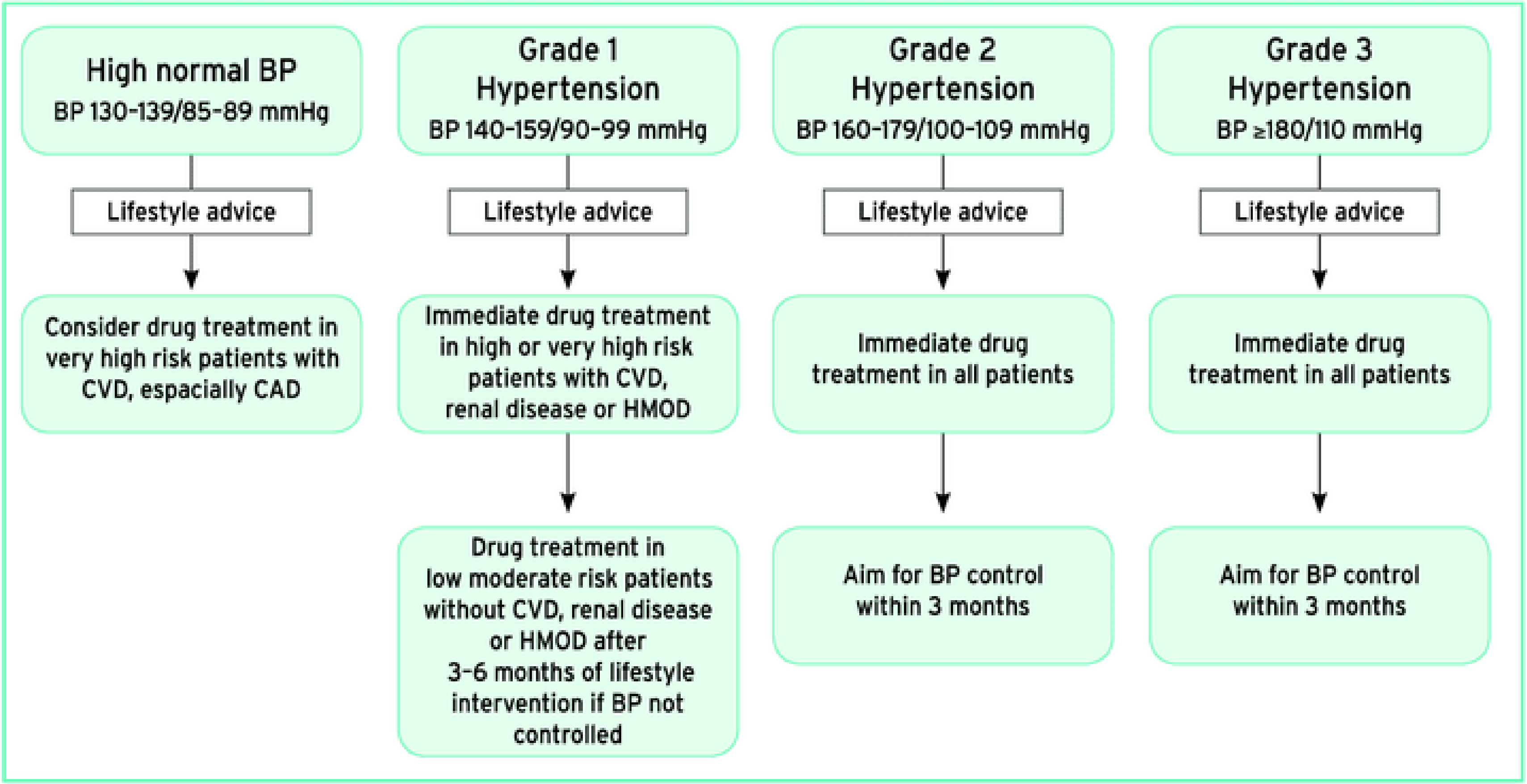


Fig. 7. Initiation of antihypertensive treatment at different initial office blood pressure levels

CORONARY HEART DISEASE

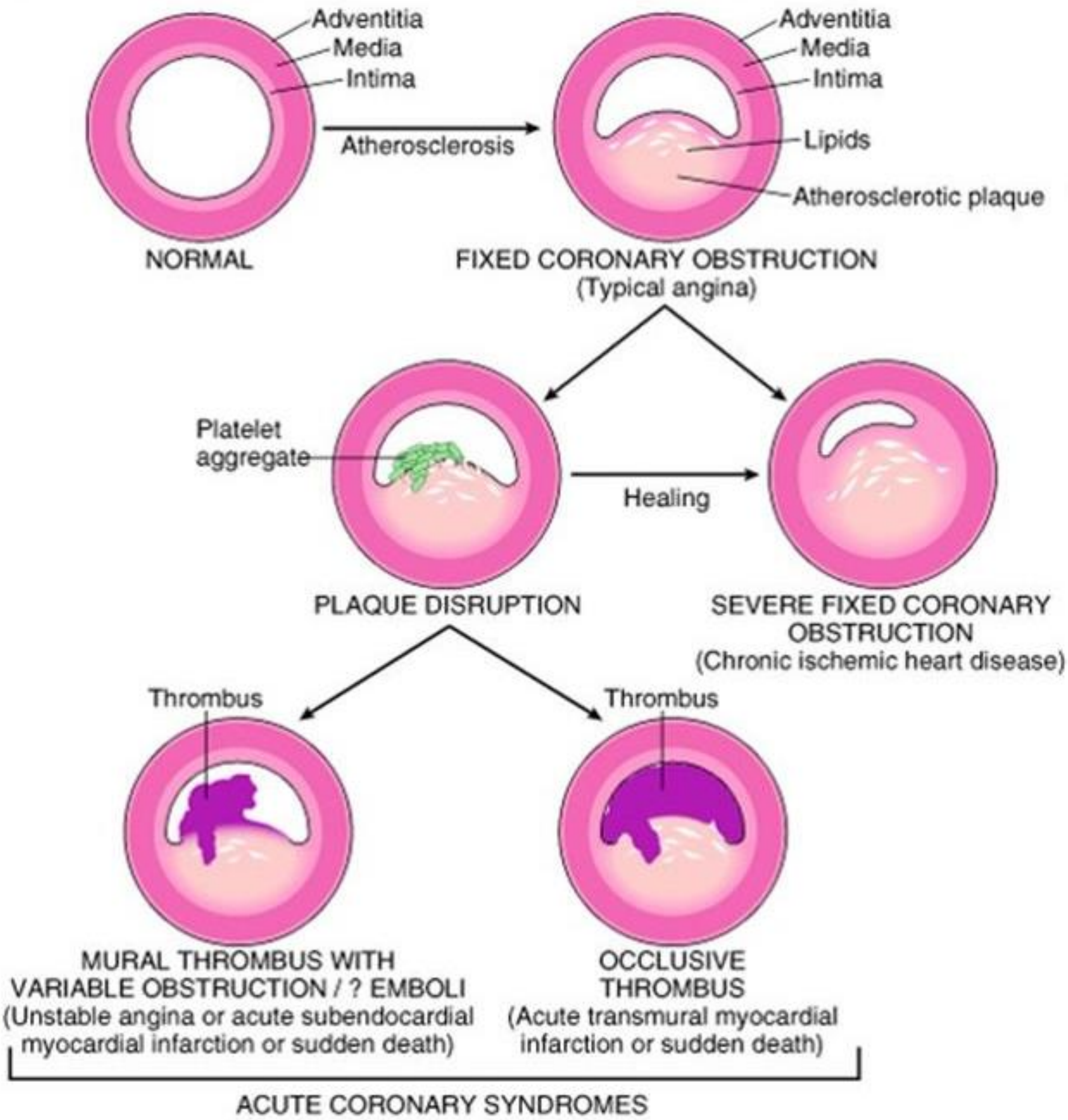


Fig. 8. Pathogenesis of coronary heart disease

STABLE ANGINA

Table 6.

Definitions of typical and atypical angina

Typical angina (definite)	Meets three of the following characteristics • Substernal chest discomfort of characteristic quality and duration • Provoked by exertion or emotional stress • Relieved by rest and/or GTN
Atypical angina (probable)	Meets two of these characteristics
Non-cardiac chest pain	Meets one or none of the characteristics

Table 7.

Classification of angina severity according to the Canadian Cardiovascular Society

Class I	Ordinary activity does not cause angina (walking and climbing stairs). Angina with rapid or prolonged exertion at work or recreation.
Class II	Slight limitation of usual activity. Angina on walking or climbing stairs rapidly, walking or stair climbing after meals, or in cold, wind or under emotional stress, or only than two blocks on the level and climbing more than one conditions.
Class III	Marked limitation of ordinary physical activity. Angina on walking one to two blocks* on the level of one flight of stairs in normal conditions and at a normal pace.
Class IV	Inability to carry on any physical activity without discomfort' – angina syndrome may be present at rest'.

*Equivalent to 100 – 200 m.

Fig. 9. Diagnostic algorithm in chest pain

Table 8.

Biochemical tests, recommended in stable CAD

Measurements of troponin to rule out myocardial necrosis associated with ACS
Complete blood count
Screening for potential T2DM (HbA1c, fasting plasma glucose, an OGTT)
Creatinine measurement
A fasting lipid profile (including LDL)
If indicated by clinical suspicion of thyroid disorder - assessment of thyroid function
Liver function tests (after beginning statinotherapy)
Creatine kinase measurement - in suspection of statin induced myopathy
BNP/NT-proBNP measurements - in patients with suspected heart failure

Table 9.

Fredrickson classification of dyslipidemia

Type	**Synonyms**	**Lipoprotein Elevation**
I (rare)	"Primary hyperlipoproteinemia", or "Familial hyperchylomicronemia"	Chylomicrons
IIa	"Polygenic or Familial hypercholesterolemia"	LDL
IIb	"Combined hyperlipidemia"	LDL+VLDL
III (rare)	"Familial dysbetalipoproteinemia"	Chylomicrons+ IDL
IV	"Familial hyperlipemia"	VLDL
V (rare)	"Endogenous hypertriglyceridemia"	VLDL+ Chylomicrons

Table 10.

Pharmacological treatments in stable CAD patients

General considerations
Optimal medical treatment indicates at least one drug for angina/ischaemia relief plus drugs for event prevention.
It is recommended to educate patients about the disease, risk factors and treatment strategy.
It is indicated to review the patient's response soon after starting therapy.
Angina/ischaemia relief
Short-acting nitrates are recommended.
First-line treatment - ß-blockers and/or calcium channel blockers to control heart rate and symptoms.
For second-line treatment - to add long-acting nitrates or ivabradine or nicorandil or ranolazine, according to heart rate, blood pressure and tolerance.
For second-line treatment – trimetazidine.
According to comordidities/tolerance it is indicated to use second line therapies as first-line treatment in selected patients.
In asymptomatic patients with large areas of ischaemia (>10%) ß-blockers should be considered.
In patients with vasospastic angina, calcium channel blockers and nitrates should be considered and beta-blockers avoided.
Event prevention
Low-dose aspirin daily is recommended in all SCAD patients.
Clopidogrel is indicated as an alternative in case of aspirin intolerance.
Statins are recommended in all SCAD patients.
ACE inhibitors (or ARBs) are recommended if presence of other conditions (e.g. heart failure, hypertension or diabetes).

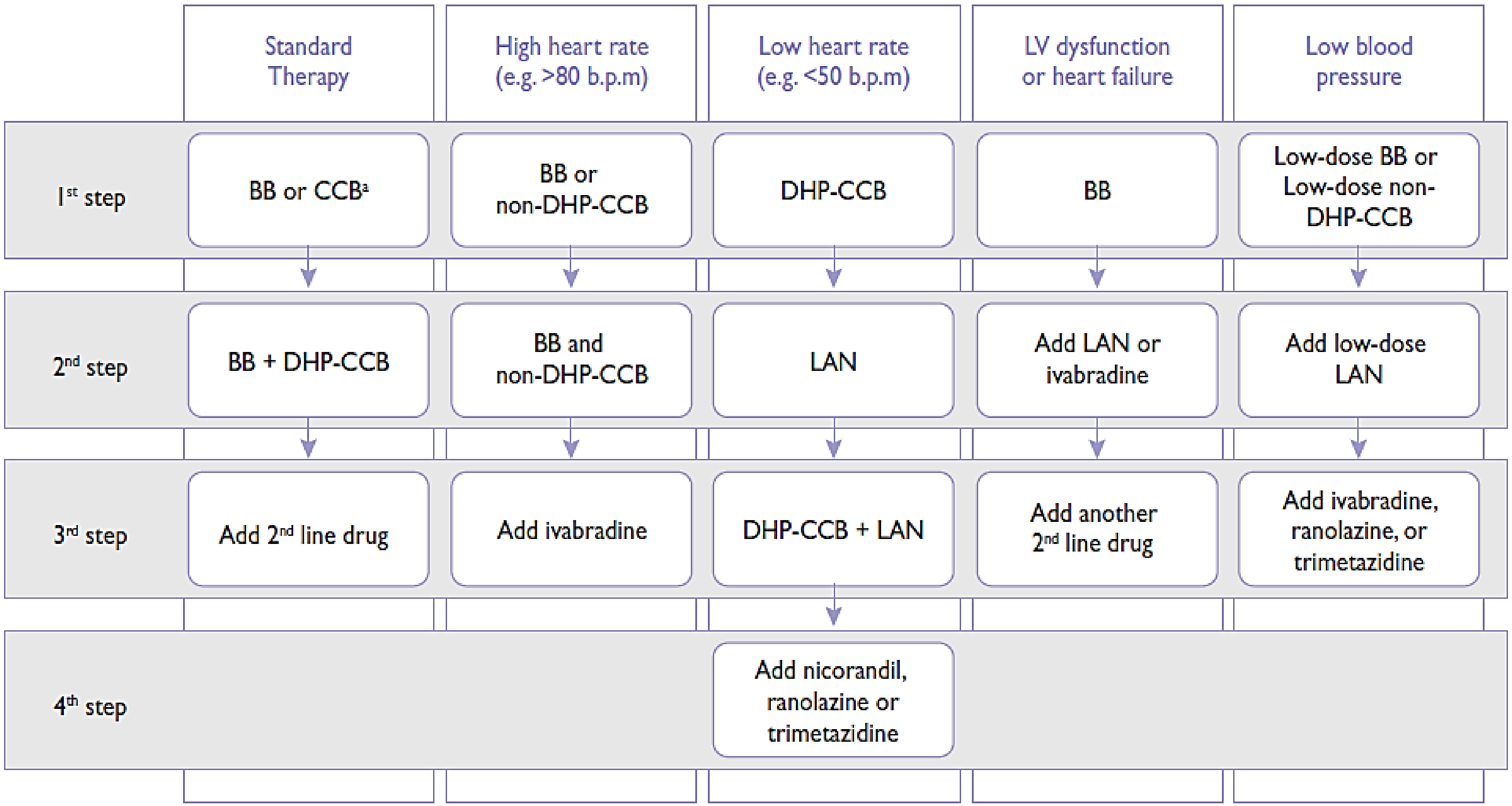


Fig. 10. Medical management of patients with stable coronary artery disease
BB - Beta-blockers,
DHP-CCB - Dihydropyridine calcium channel blockers,
LAN - Long-acting nitrates

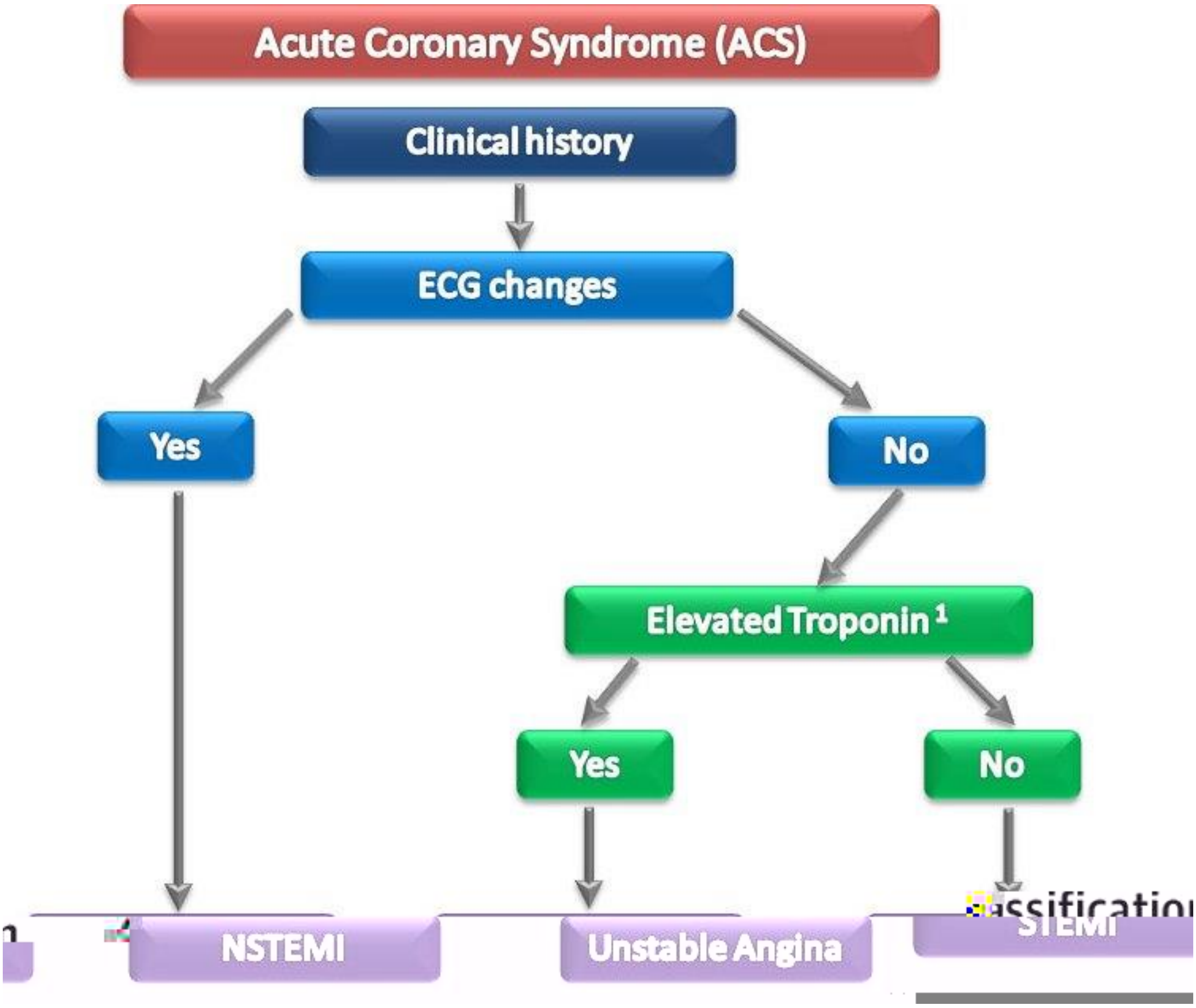


Fig. 11. Acute coronary syndrome

MIOCARDIAL INFARCTION

Acute myocardial infarction (MI) defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischaemia. A combination of criteria is required to meet the diagnosis of acute MI, namely the detection of an increase and/or decrease of a cardiac biomarker, preferably high-sensitivity cardiac troponin, with at least one value above the 99th percentile of the upper reference limit and at least one of the following:

- ❖ Symptoms of ischaemia.
- ❖ New or presumed new significant ST-T wave changes or left bundle branch block on 12-lead ECG.
- ❖ Development of pathological Q waves on ECG.
- ❖ Imaging evidence of new or presumed new loss of viable myocardium or regional wall motion abnormality.
- ❖ Intracoronary thrombus detected on angiography or autopsy.

Table 11.

Universal definition of miocardial infarction

Type	Classification	Clinical and Diagnostic Criteria
1	Spontaneous MI	Plaque rupture, ulceration, fissuring, erosion, or dissection resulting in coronary thrombosis
2	Supply/demand mismatch	Mismatch between myocardial oxygen supply and demand driven by a secondary process other than coronary artery disease
3	Suspected MI-related death	Cardiac death in a setting suggestive of ischemic process without definitive cardiac biomarker evidence of MI
4a	PCI-related MI	Rise in cardiac biomarkers accompanied by symptoms, electrocardiographic, angiographic, or imaging evidence of ischemia after PCI
4b	Stent thrombosis	Confirmed stent thrombosis in context of ischemia and dynamic cardiac biomarker changes
5	CABG-related MI	Rise in cardiac biomarkers accompanied by electrocardiographic, angiographic, or imaging evidence of ischemia after CABG

PCI- percutaneous intervention, CABG- coronary artery bypass graft

Table 12.

Conditions other than myocardial infarction type 1 associated with cardiac troponin elevation

Tachyarrhythmias
Heart failure
Hypertensive emergencies
Critical illness (e.g. shock/ sepsis/ burns)
Myocarditisa
Tako-Tsubo cardiomyopathy
Structural heart disease
Aortic dissection
Pulmonary embolism, pulmonary hypertension
Renal dysfunction and associated cardiac disease
Coronary spasm
Acute neurological event (e.g. stroke or subarachnoid haemorrhage)
Cardiac contusion or cardiac procedures (CABG, PCI, cardioversion, ablation, pacing or endomyocardial biopsy)
Hypo- and hyperthyroidism
Infiltrative disease (haemochromatosis, amiloidosis, sarcoidosis, scleroderma)
Miocardial drug toxicity or poisoning (5-fluorouracil, Herceptin, doxorubicin, snake venoms)
Extreme endurance efforts
Rhabdomyolysis

CABG - coronary artery bypass graft;
PCI - percutaneous coronary intervention

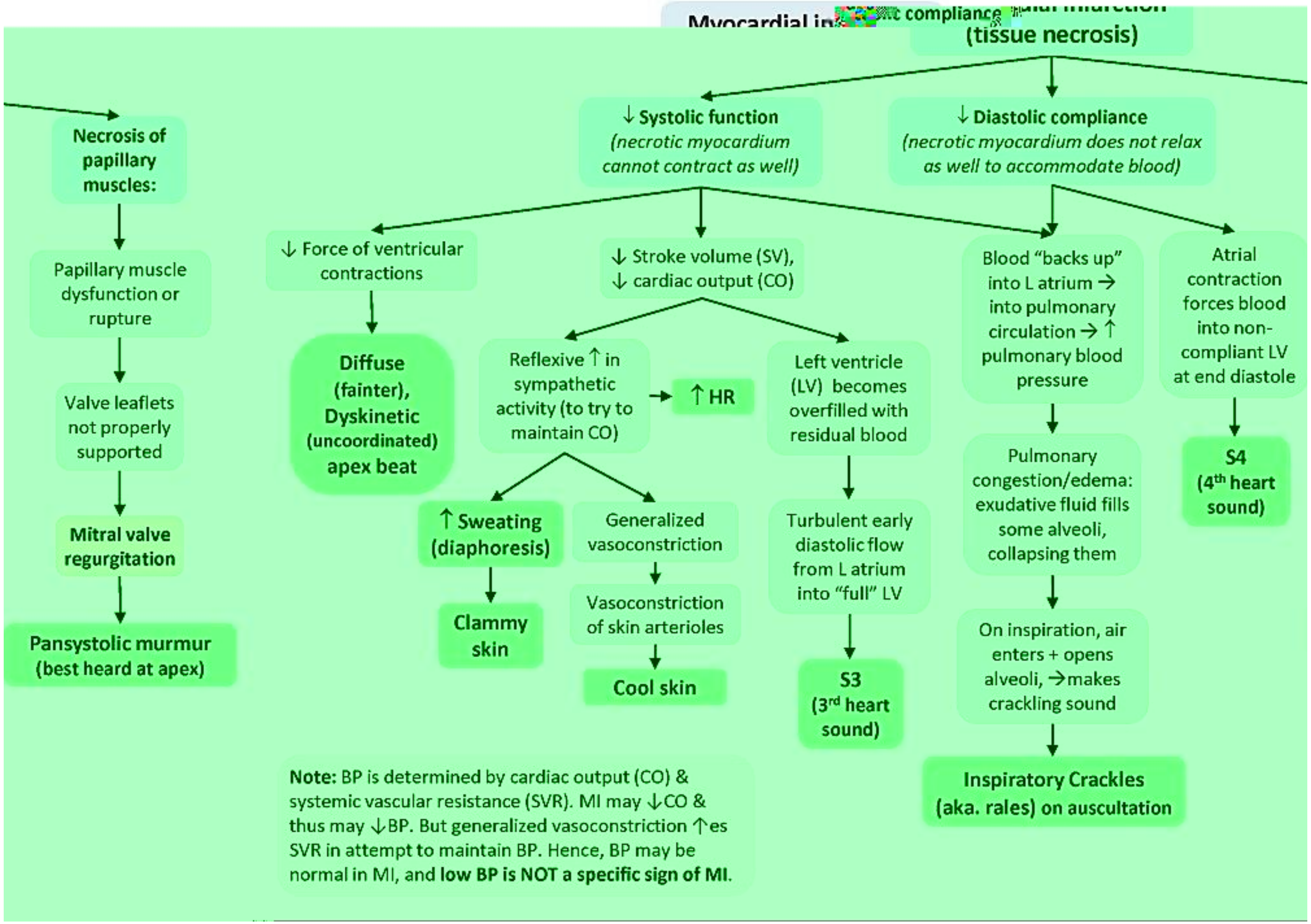


Fig. 12. Findings on physical exam in myocardial infarction

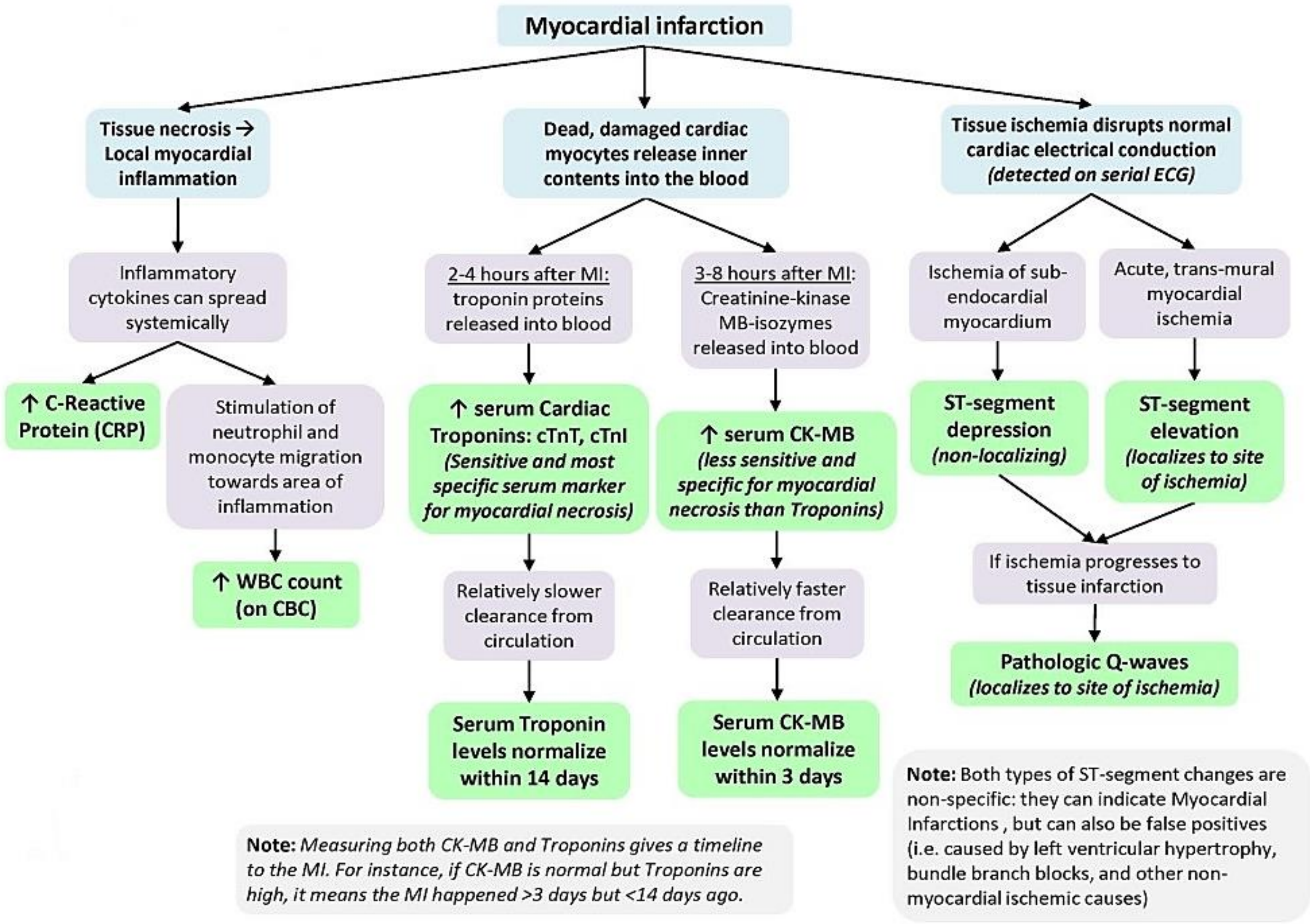


Fig. 13. Findings on laboratory and instrumental investigations in MI

Table 13.

Markers of myocardial necrosis

Marker	Initial rise	Peak	Return to normal	Notes
Troponin I/T	2–4 h	10–24 h	5–10 d	Troponin I and T are sensitive and specific markers (unlike troponin C). Their blood level clearly relates to risk and thus they represent a powerful tool for risk stratification
Creatine kinase*	3–4 h	10–24 h	2–4 d	CK-MB is the main cardiac isoenzyme
Lactate [illegible]genase	10 h	24–72 h	14 d	Myocardium mainly has LDH1
[illegible]in	1–2 h	4–8 h	24 h	Convenient early marker due to availability of assay, but not cardiac specific
[illegible]ty-acid [illegible]proteins	1.5 h	5–10 h	24 h	Undergoing evaluation

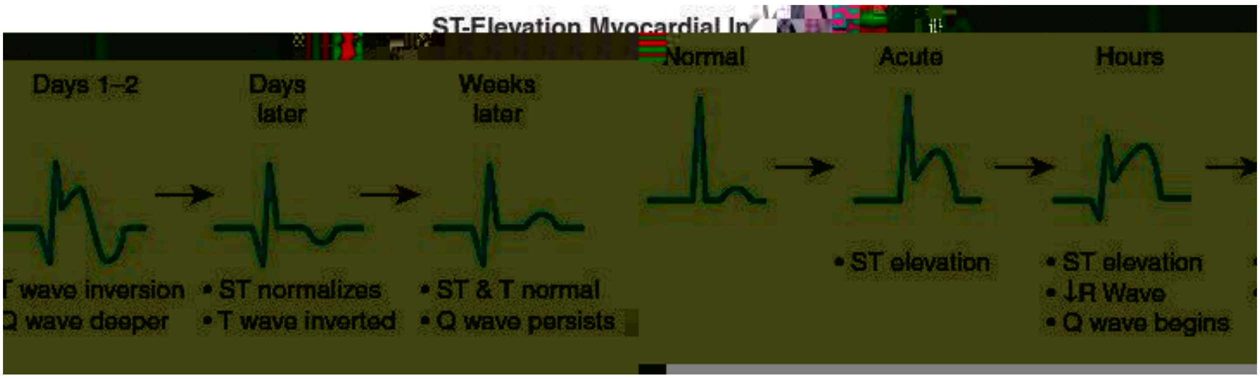


Fig. 14. ECG evolution during ST-elevation MI (Q-wave MI)

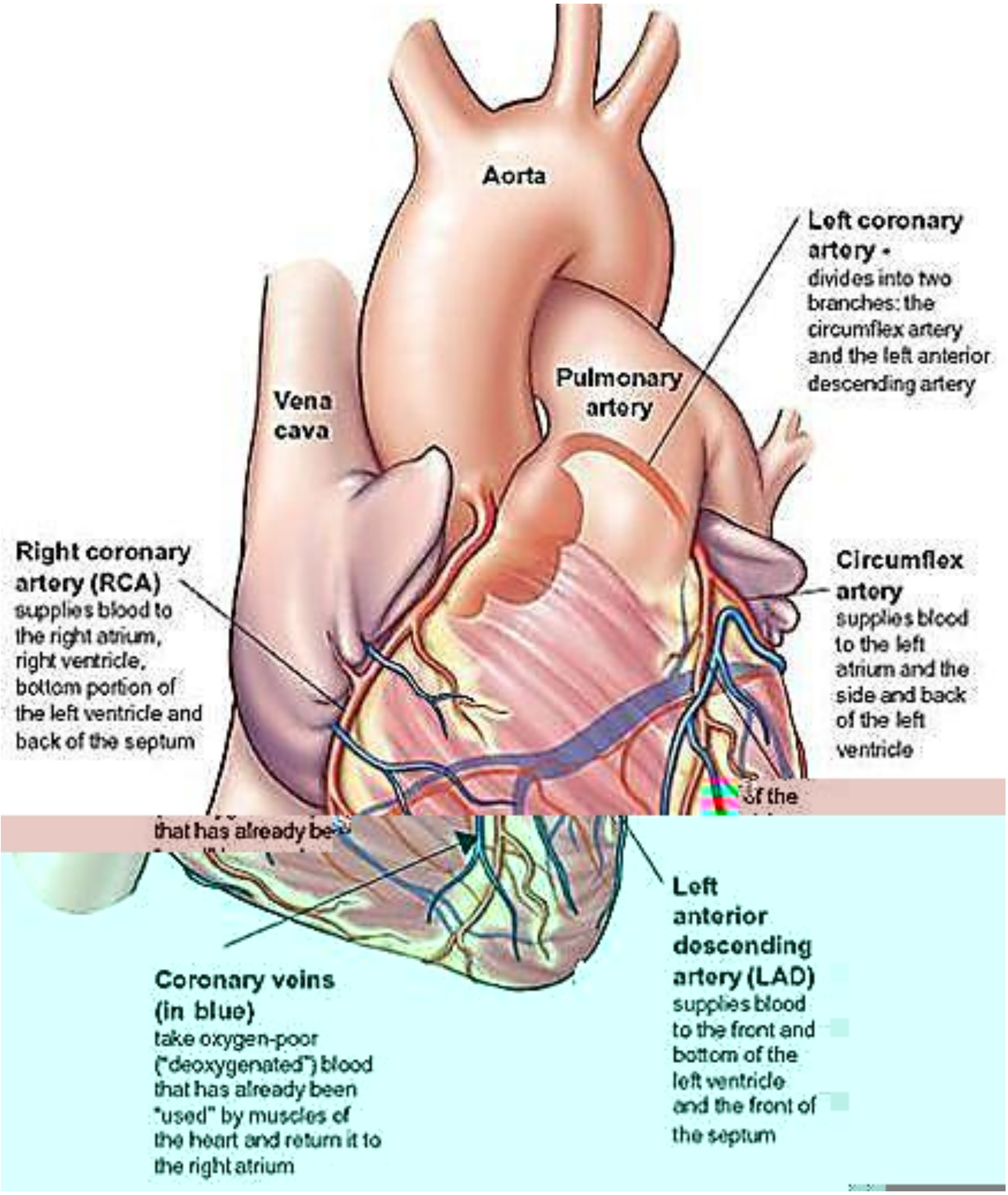


Fig. 15. Coronary arteries anatomy

LIMB LEADS		PRECORDIAL LEADS	
I Lateral left ventricle	aVR square root of squat	V1 Septal	V4 Anterior
II Inferior portion of the left ventricle	aVL Lateral left ventricle	V2 Antero-Septal	V5 Lateral left ventricle
III Inferior portion of the left ventricle	aVF Inferior portion of the left ventricle	V3 Anterio-Septal	V6 Lateral left ventricle

Localization of the Myocardial Infarct with the ECG

Localization	ST elevation	Reciprocal ST depression	Coronary Artery
Anterior MI	V1-V6	None	LAD
Septal MI	V1-V4 disappearance of septum Q in leads V5-V6	None	LAD
Laterla MI	I, aVL, V5-V6	II, III, aVF (Inf.leads)	LCX
Inferior MI	II, III, aVF	[illegible]	[illegible]
[illegible]	V7-V8,V9	High R in V1-V3 with ST depression V1-V3 > 2 mm (mirror vew)	RCA or LCX
ht Ventricle MI	V1-V4R	I, aVL	RCA
ial MI	PTa in I, V5, V6	PTa in I, II or III	RCA

Fig. 16. Localization of myocardial infarction
LAD – left anterior descending artery
RCA – right coronary artery
LCX – left circumflex artery

Table 14.

Pharmacological therapy in MI

Nitrates:	are useful for pain management in acute ischemia and should initially be given sublingually or orally. If pain becomes worse, nitrates are given intravenously.
Morphine Sulfate:	relieves pain not only directly but also indirectly by diminishing the sympathetic nervous system's drive and catecholamine secretion, which increases myocardial oxygen demand.
Beta-blockers and calcium-channel blockers:	should be started early in all patients, who do not have contraindications; otherwise calcium channel blockers (CCBs), such as diltiazem is given. Dihydropyridine calcium antagonists, such as nifedipine, should not be given.
ACE-Inhibitors:	should be initiated early in all patients as its benefit goes beyond blood pressure control and may relate to plaque stabilization.
Antiplatelet:	Therapy with **aspirin**, **clopidogrel** (or ticlopidine) and **glycoprotein IIb/IIIa antagonist** are used. All patients should take aspirin, 300mg and clopidogrel, 300mg stat, followed with 75 daily of aspirin indefinitely as a maintenance dose and clopidogrel, 75mg for up to one year.
Anticoagulants:	These agents include Heparin, Low Molecular Weight Heparins (LMWH), and Direct Thrombin Inhibitors.
Thrombolytic (Fibrinolytic) Therapy:	Patients without ST-segment elevation do not benefit from immediate aggressive thrombolytic therapy. Thrombolytic therapy is recommended for STEMI patients.

COMPLICATIONS OF MYOCARDIAL INFARCTION

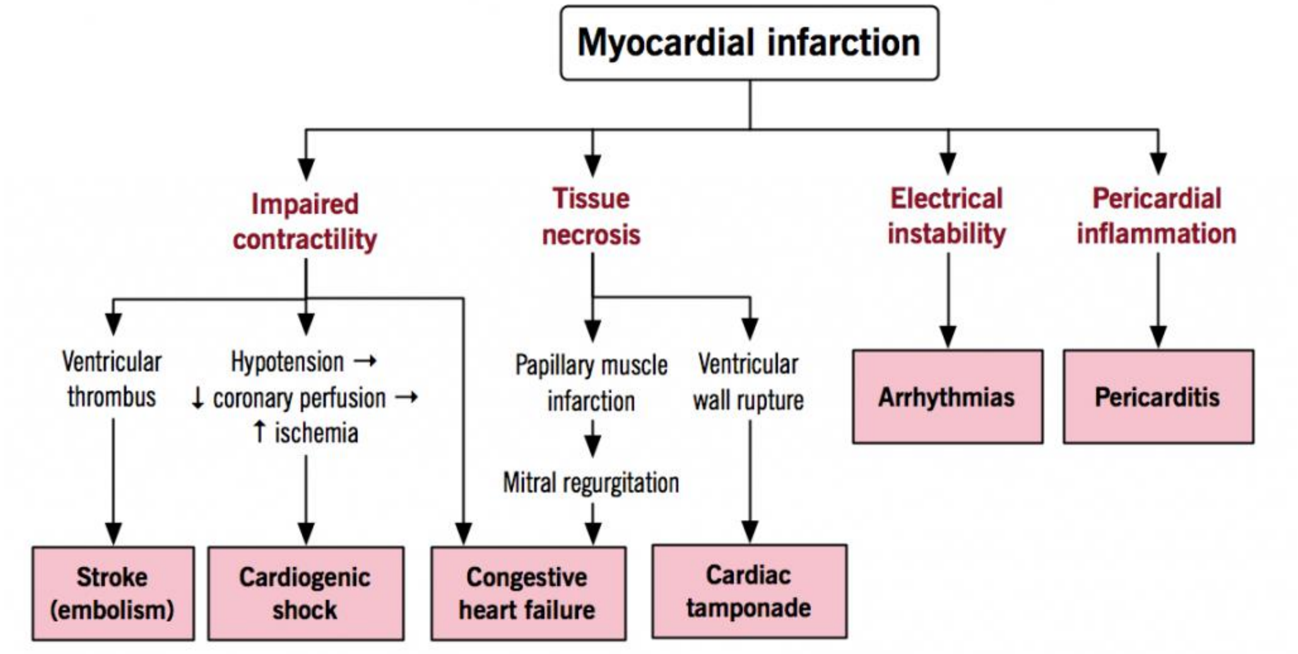


Fig. 17. Complications of MI

INFECTIVE ENDOCARDITIS

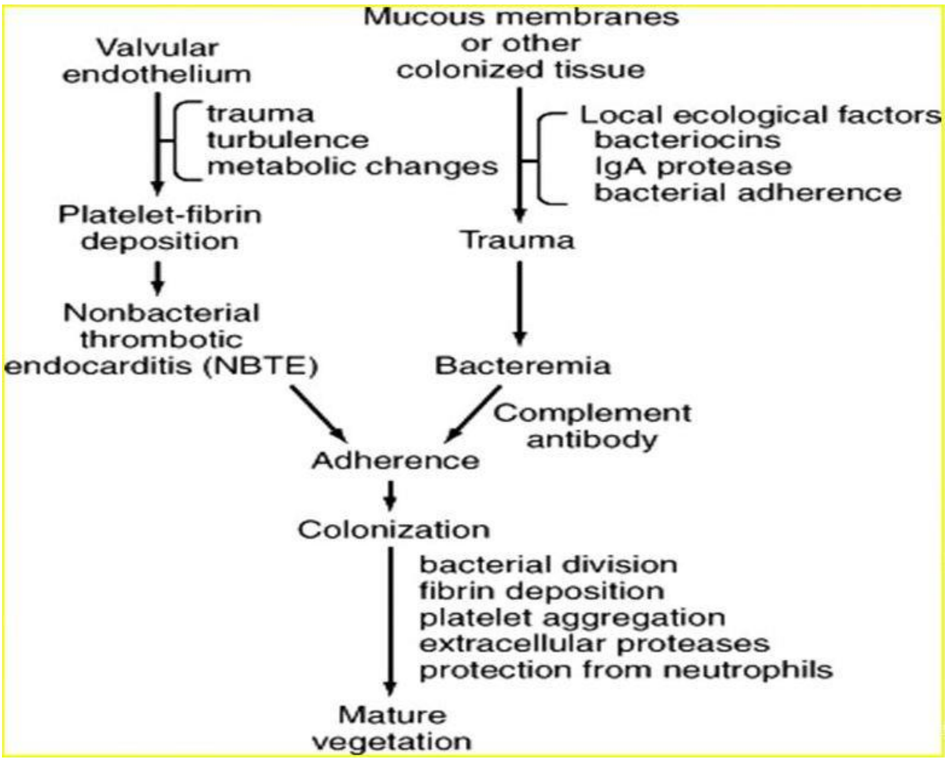


Fig. 18. Pathogenesis of Infective endocarditis

Table 15.

Definition of infective endocarditis according to the modified Duke criteria

Definite IE
Pathological criteria • Microorganisms demonstrated by culture or on histological examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen; or • Pathological lesions; vegetation or intracardiac abscess by histological examination showing active endocarditis Clinical criteria • 2 major criteria; or • 1 major criterion and 3 minor criteria; or • 5 minor criteria
Possible IE
• 1 major criterion and 1 minor criterion; or • 3 minor criteria
Rejected IE
• Firm alternate diagnosis; or • Resolution of symptoms suggesting IE with antibiotic therapy for ≤4 days; or • No pathological evidence of IE at surgery or autopsy, with antibiotic therapy for ≤4 days; or • Does not meet criteria for possible IE, as above

Table 16.

Modified criteria for the diagnosis of infective endocarditis

Major criteria
1. Blood cultures positive for IE • Typical microorganisms consistent with IE from 2 separate blood cultures: ✓ Viridans streptococci, Streptococcus gallolyticus (Streptococcus bovis), HACEK group, Staphylococcus aureus; or ✓ Community-acquired enterococci, in the absence of a primary focus; or • Microorganisms consistent with IE from persistently positive blood cultures: ✓ ≥2 positive blood cultures of blood samples drawn >12 h apart; or ✓ All of 3 or a majority of ≥4 separate cultures of blood (with and last samples drawn ≥1 h apart); or • Single positive blood culture for Coxiella burnetii or phase I IgG antibody titre >1:800
2.Imaging positive for IE • Echocardiogram positive for IE: ✓ Vegetation; ✓ Abscess, pseudoaneurysm, intracardiac ✓ Valvular perforation or aneurysm; ✓ New partial dehiscence of prosthetic valve. • Abnormal activity around the site of prosthetic valve implantation detected by 18F-FDG PET/CT (only if the prosthesis was implanted for >3 months) or radiolabelled leukocytes SPECT/CT. • Defenite paravalvular lesions by cardiac CT.
Minor criteria
1. Predisposition such as predisposing heart condition, or injection drug use. 2. Fever as temperature >38°C. 3. Vascular phenomena (including those detected by imaging only): major arterial emboli, septic pulmonary infarcts, infectious (mycotic) aneurysm, intracranial haemorrhage, conjunctival haemorrhages, and Janeway’s lesions. 4. Immunological phenomena: glomerulonephritis, Osler’s nodes, Roth’s spots, and rheumatoid factor. 5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE.

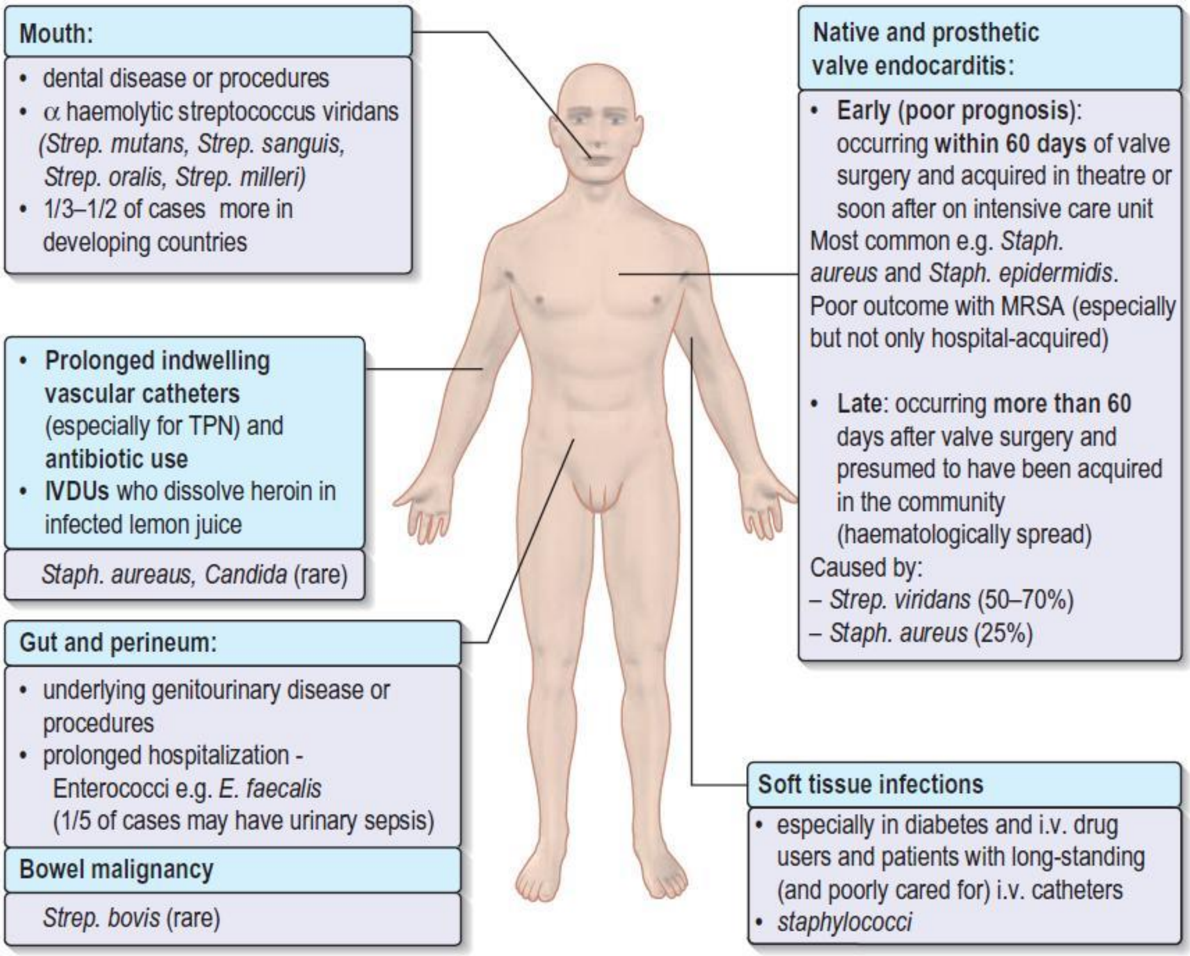


Fig. 19. Etiology of Infective Endocarditis (from Kumar and Clark`s Clinical Medicine, 7th Edition)

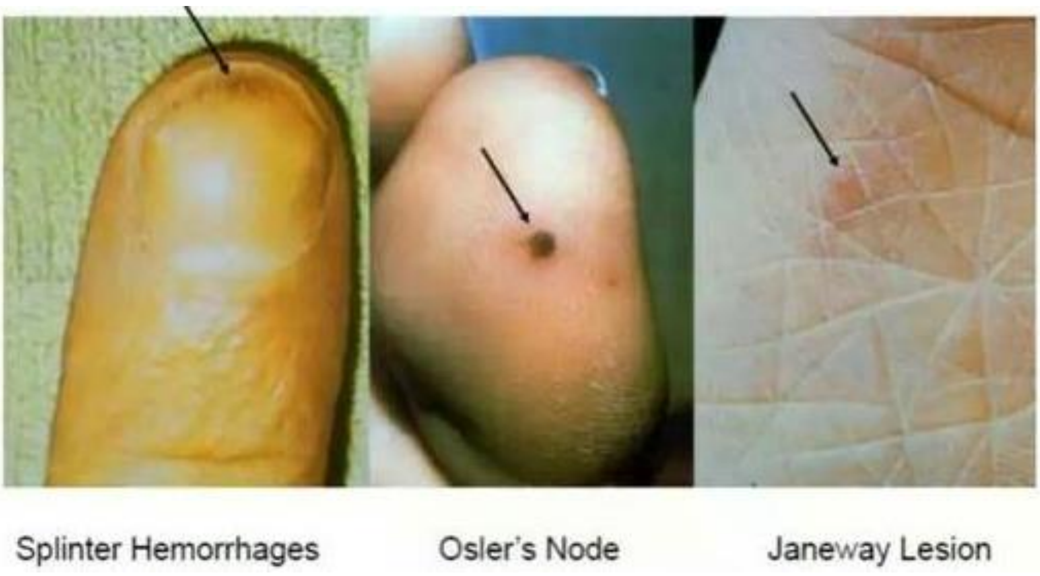


Fig. 20. Cutaneous manifestations of endocarditis

Table 17.

Echocardiographic findings in infective endocarditis

Vegetation	Oscillating or non- oscillating intracardiac mass on valve or other endocardial structures, or on implanted intracardiac material.
Abscess	Thickened, non- homogeneous perivalvular area with echodense or echolucent appearance.
Pseudoaneurysm	Pulsatile perivalvular echo-free space.
Perforation	Interruption of endocardial tissue continuity
Fistula	Colour-Doppler communication between two neighbouring cavities through a perforation.
Valve aneurysm	Saccular bulging of valvular tissue.
Dehiscence of a prosthetic valve	Paravalvular regurgitation, with or without rocking motion of the prosthesis.

Table 18.

Predictors of poor outcome in patients with infective endocarditis

Patient characteristics • Older age • Prosthetic valve IE • Diabetes mellitus • Comorbidity (e.g., frailty, immunosuppression, renal or pulmonary disease)
Clinical complications of IE • Heart failure, Renal failure • >Moderate area of ischaemic stroke • Brain haemorrhage • Septic shock
Microorganism • Staphylococcus aureus • Fungi • Non-HACEK Gram-negative bacilli
Echocardiographic findings • Periannular complications • Severe left-sided valve regurgitation • Low left ventricular ejection fraction • Pulmonary hypertension • Large vegetations • Severe prosthetic valve dysfunction • Premature mitral valve closure and other signs of elevated DBP

CHRONIC HEART FAILURE

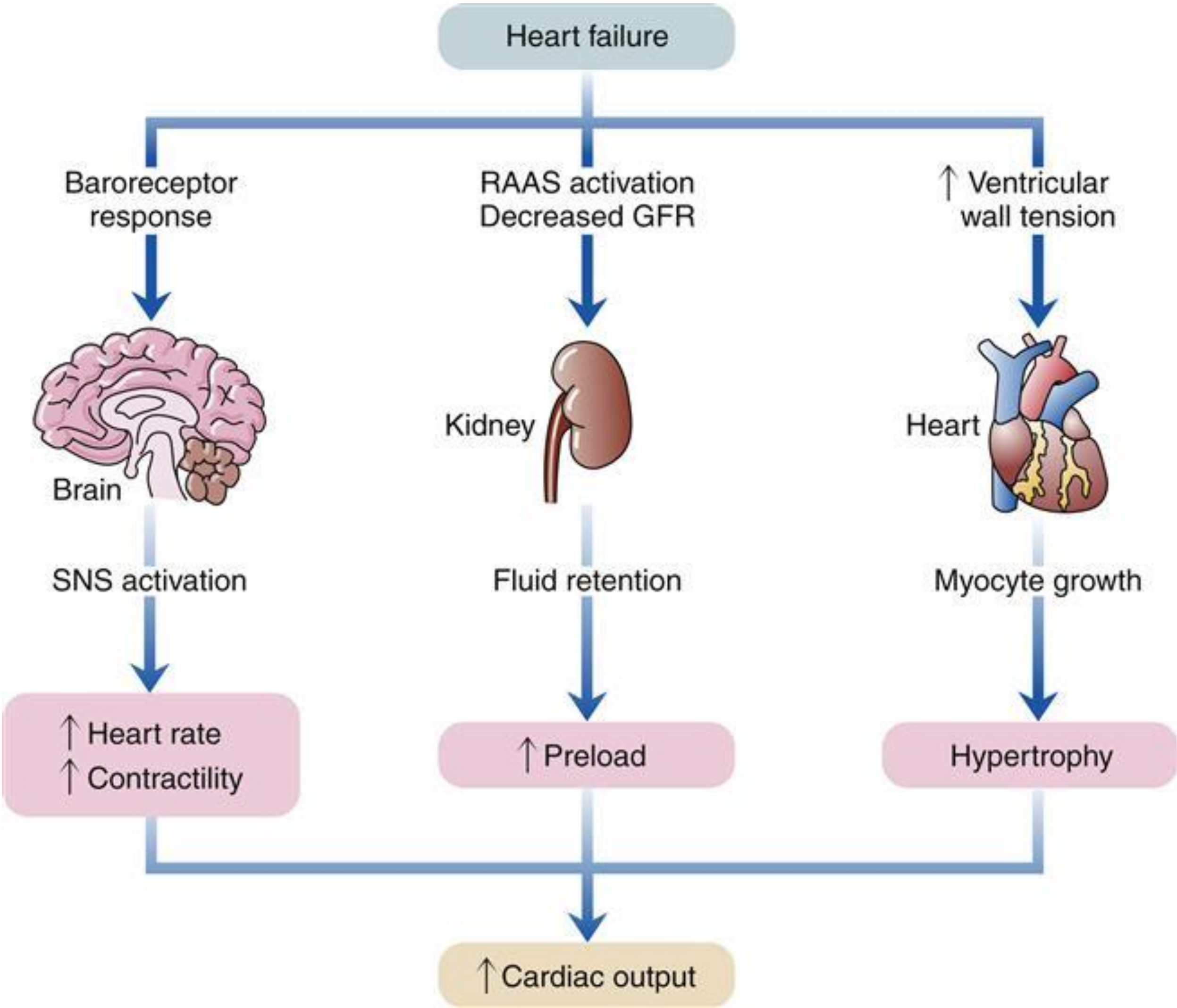


Fig. 21. Main compensatory mechanisms in heart failure

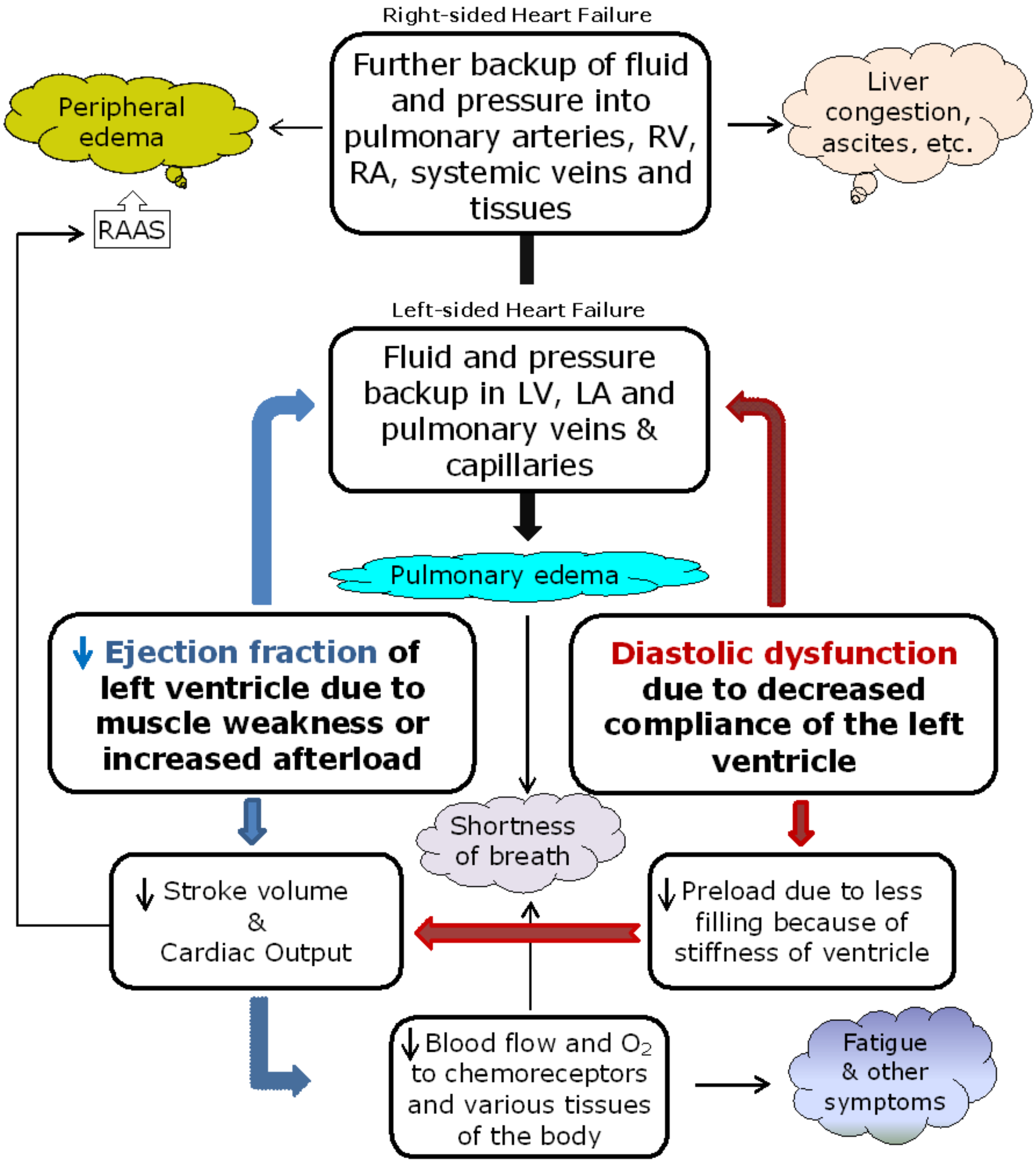


Fig. 22. Pathophysiology of congestive heart failure (from Prohealthinsight.com – Feb.22, 2019)

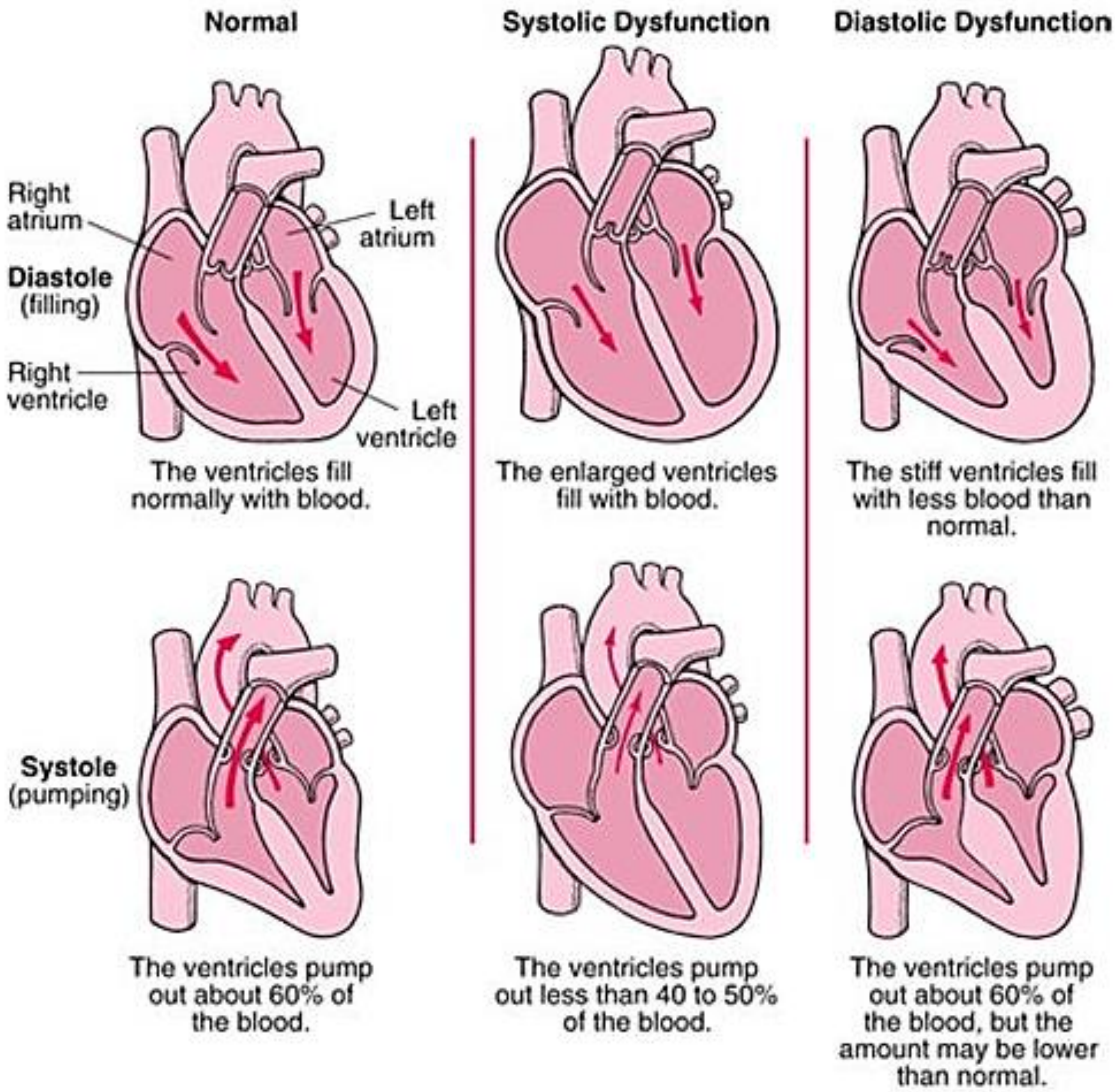


Fig. 23. Types of heart failure

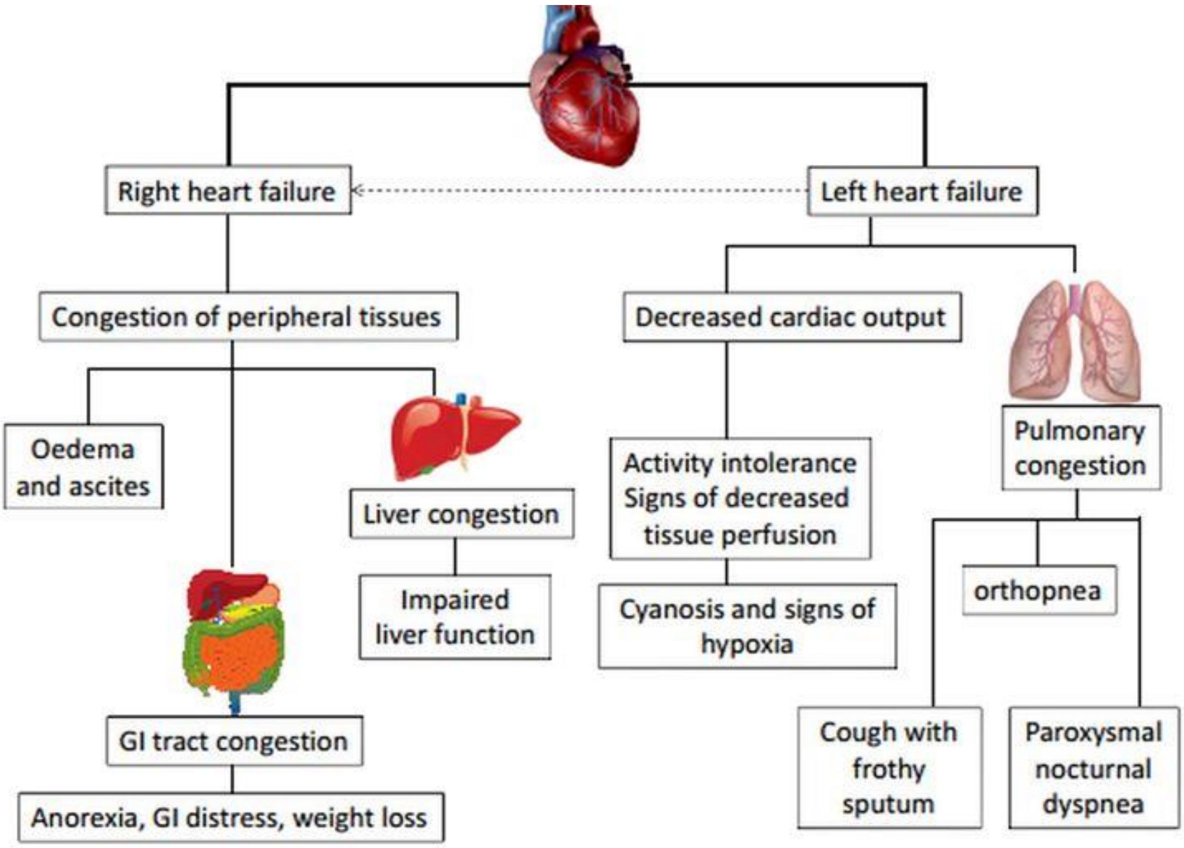


Fig. 24. Symptoms of left- and right heart failure

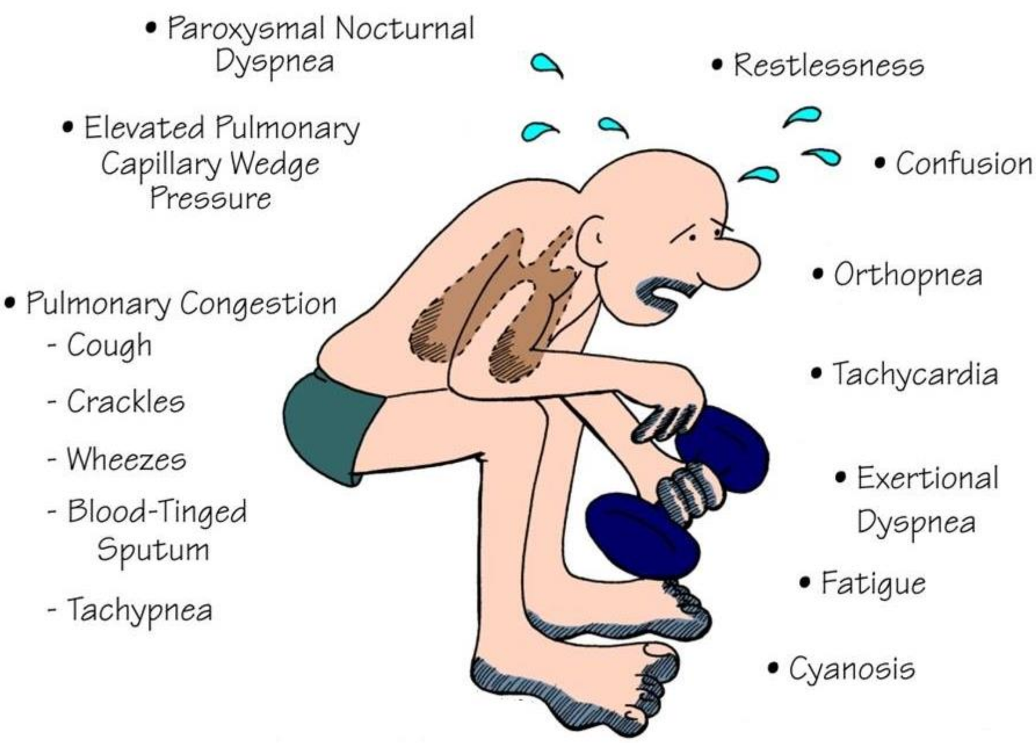


Fig. 25. Left-sided heart failure symptoms

Table 19.

Symptoms and signs of heart failure (HF)

Symptoms	**Signs**
Typical	**More specific**
Breathlessness Orthopnoea Paroxysmal nocturnal dyspnoea Ankle swelling Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise	Elevated jugular venous pressure Hepatojugular reflux Third heart sound (gallop rhythm) Laterally displaced apical impulse
Less typical	**Less specific**
Nocturnal cough Wheezing Bloated feeling Palpitations Dizziness Syncope Bendopnea Loss of appetite Confusion (especially in the elderly) Depression	Weight gain (>2 kg/week) Weight loss (in advanced HF) Tissue wasting (cachexia) Cardiac murmur Peripheral oedema (ankle, sacral, scrotal) Pulmonary crepitations Reduced air entry and dullness to percussion at lung bases (pleural effusion) Narrow pulse pressure Tachycardia Irregular pulse Tachypnoea Cheyne Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria

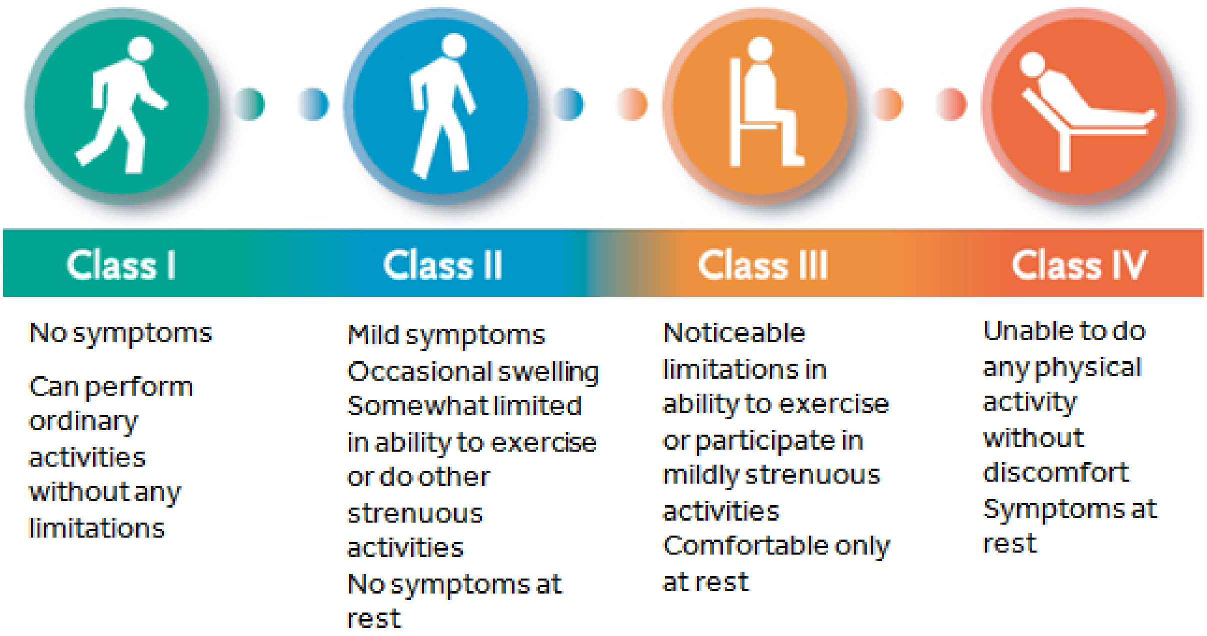


Fig. 26. NYHA functional classification of CHF

Table 20.

Classification of CHF according N.D.Strazhesko, V.H.Vasilenko

I stage		Initial stage, latent. There are symptoms during physical exercises: dyspnea, palpitation. These symptoms subside at rest.
II stage	**A**	There are features of congestion in a large or small circle of blood circulation.
	B	The features of congestion in both: a large and small circles of blood circulation. Patients are fully disabled. At rest pronounced cyanosis, swollen jugular veins, edema, and ascites are revealed.
III stage		Final, dystrophic stage with marked congestion in large and small circles of blood circulation, circulation hemodynamic disorders, irreversible morphological changes of all organs, functional and metabolic disorders.

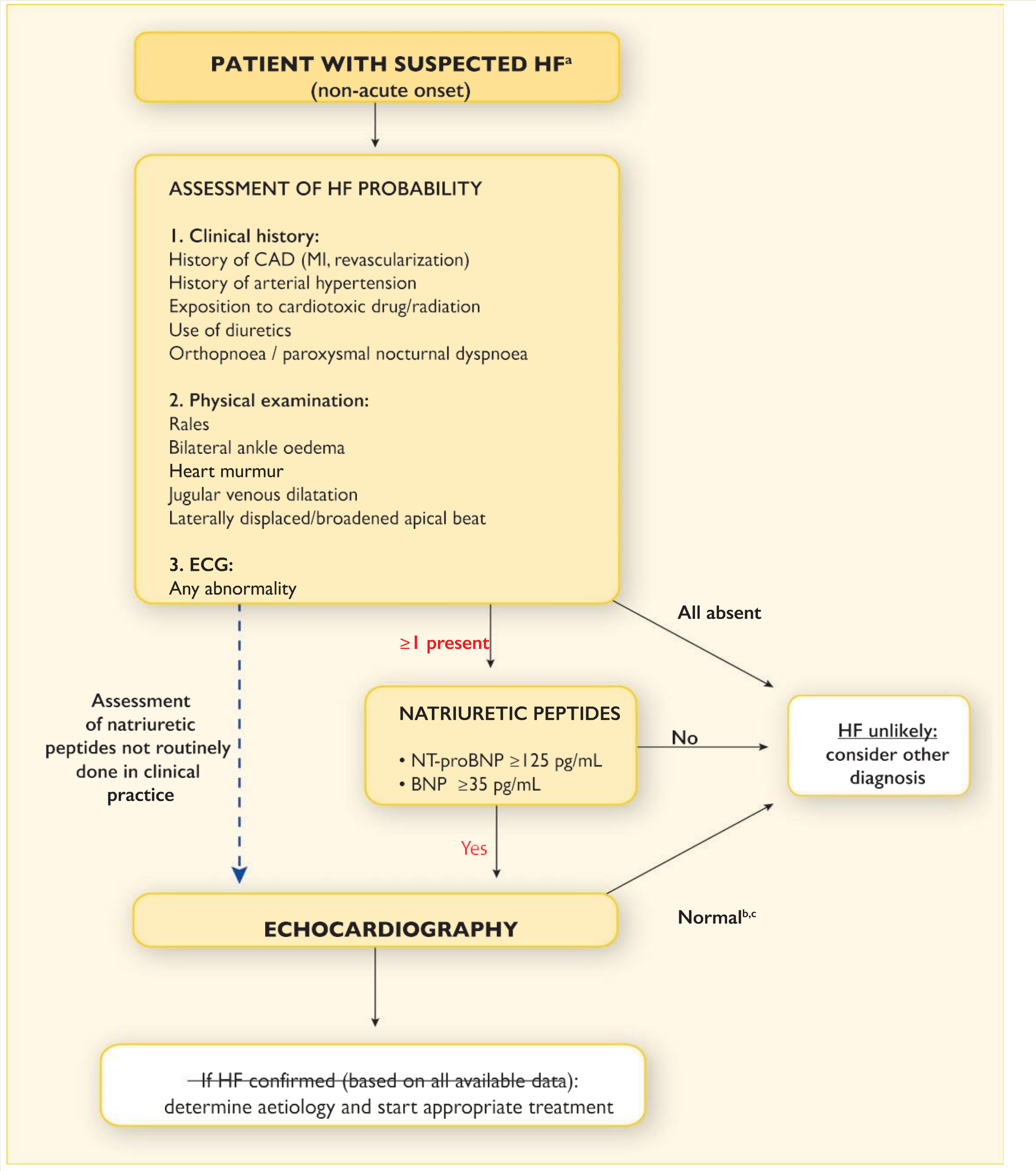


Fig. 27. Diagnostic algorithm for a diagnosis of heart failure of non-acute onset

BNP - B-type natriuretic peptide; CAD - coronary artery disease;
HF - heart failure; MI - myocardial infarction;
NT-proBNP - N-terminal pro-B type natriuretic peptide

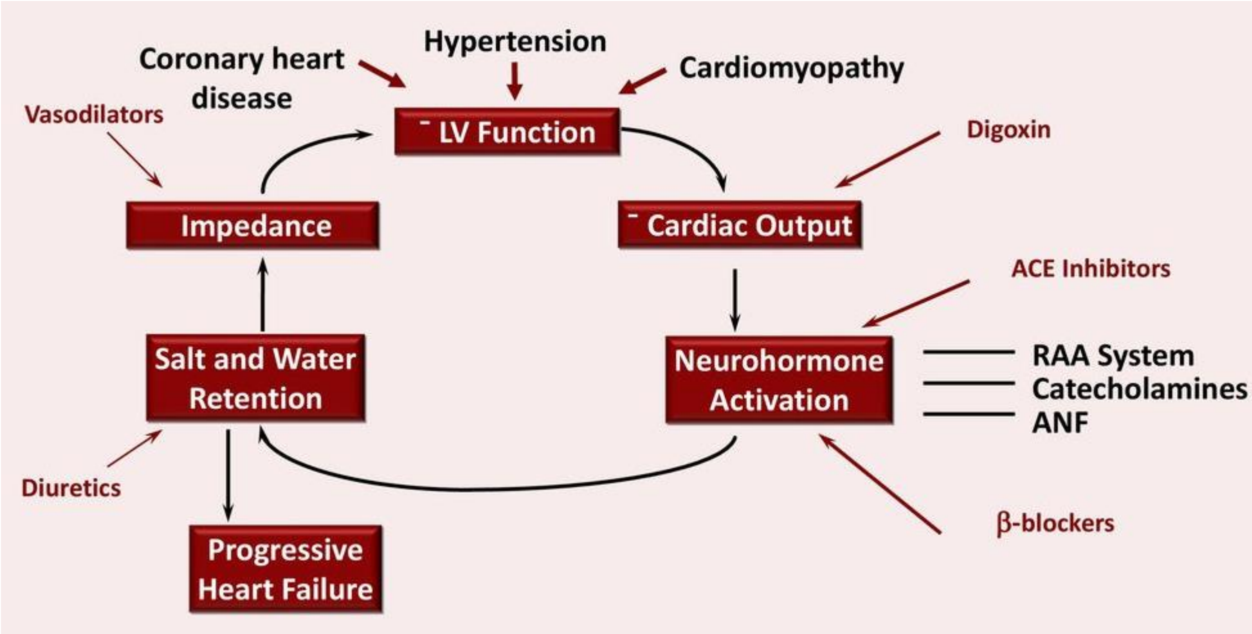
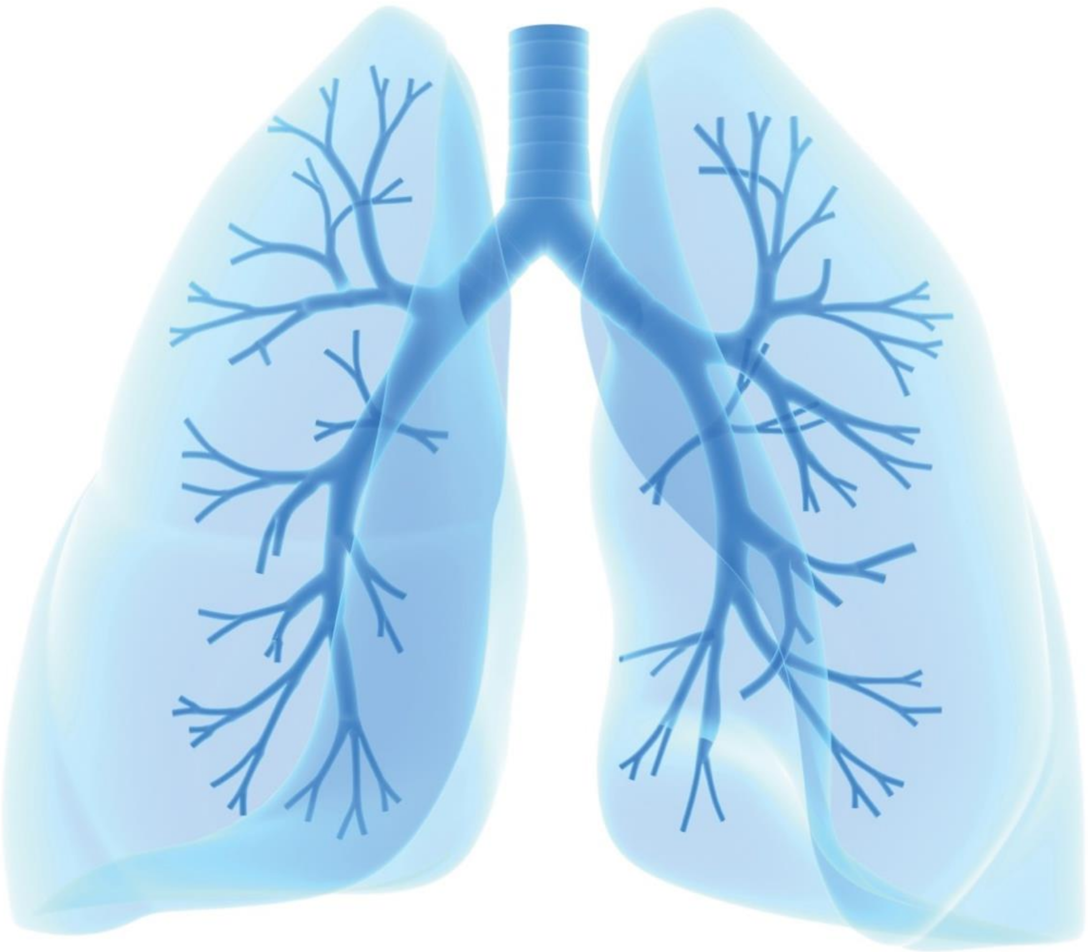


Fig. 28. Mechanisms of action of medications for heart failure

PULMONOLOGY



PNEUMONIA

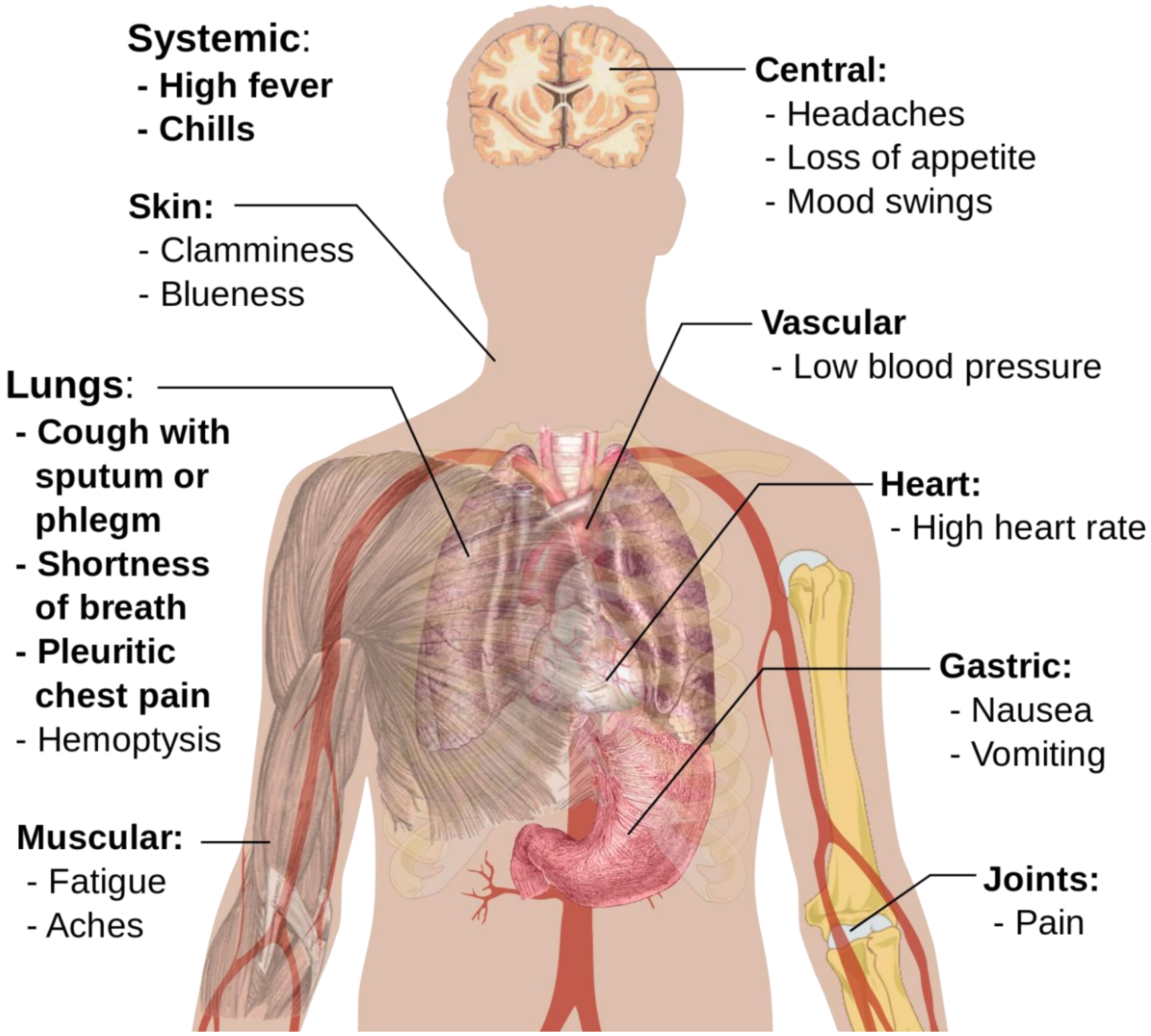


Fig. 29. Main symptoms of pneumonia

Table 21.

Features of typical and atypical pneumonias

Features	Typical Pneumonia	Atypical Pneumonia
Onset	Rapid	Slow
Organisms	• Strep. pneumoniae (most common cause of pneumonia) •H. infulenza	•Mycoplasma pneumoniae •Legionella pneumoniae •Chlamydia pneumoniae
C/F	Fever a/w chills, and predominant pulmonary features such as productive cough with mucopurulent sputum, pleuritic chest pain ; signs of consolidation such as decreased chest movement, dullness, increased vocal fremitus, egophony, bronchial breath sounds and crepitations.	Non-pulmonary features are predominant such as gradual onset of fever and dry cough; myalgia, arthralgia, headache, sore throat, nausea, vomiting and diarrhea. Respiratory signs like chest pain and productive cough, are less prominent.
Labs	Leucocytosis	WBC count may be WNL or marginally raised
Chest X-ray	Patchy or lobar infiltrates (opacity)	Patchy non-lobar infiltrates present bilaterally

WBC - white blood cells, WNL - Within normal limits.

- **C**onfusion (disorientation to person, place, or time)
- **U**rea (blood urea nitrogen) >7 mmol/L (20 mg/dL)
- **R**espiratory rate >30 breaths/minute
- **B**lood pressure (systolic <90 or diastolic <60)
- Age **>65** years

Score	Mortality	Treatment
0-1	0.7-2.1	Outpatient
2	9.2	Inpatient
≥3	≥14.5	ICU

Fig. 30. CURB-65 score.
(ICU - intensive care unit)

Table 22.

Complications of pneumonia

Pulmonary	**Extrapulmonary**
• Pleurisy • Empyema • Pulmonary destruction • Pulmonary abscess • Pneumothorax • Acute respiratory insufficiency	• Infectious-toxic shock • Disseminated intravascular coagulation syndrome • Cardio-vascular insufficiency • Respiratory distress syndrome • Sepsis • Infective endocarditis

Table 23.

Empiric antibacterial treatment of community-acquired pneumonia

Clinical case	Antibiotics of choice
Outpatient treatment	
Patients without concomitant diseases, antibiotics not taking the last 3 months	Amoxicillin Or Macrolide (per os)
Patients with concomitant diseases, antibiotics not taking the last 3 months	Amoxicillin / clavulonate Macrolide or New fluoroquinolone (per os)
Inpatient treatment	
General Department	β-lactam + macrolide (iv) or New fluoroquinolone (iv)
Intensive Care Unit	β-lactam + macrolide (iv) or New fluoroquinolone (iv) + cephalosporin III

Table 24.

Empirical antibiotic therapy of nosocomial pneumonia

Nosocomial pneumonia	Probable pathogens	Recommended Antibiotics
Early (<5 days) of any severity in patients without risk factors	*S. pneumoniae* *H. influenzae* *S. aureus* *E. Coli* *K. Pneumoniae* *Enterobacter spp.* *Proteus spp.* *Serratia marcescens*	Cephalosporins without anti-Pseudomonas activity (ceftriaxone, cefotaxime) Inhibitor-protected aminopenicillins Fluoroquinolones (levofloxacin, moxifloxacin, ciprofloxacin) Carbapenems without anti-Pseudomonas activity (ertapenems)
Late (> 5 days) of any severity in patients with risk factors	P.aeruginosa K. Pneumoniae , Acinetobacter spp. Methicillin resistant S. aureus	1. Cephalosporins with anti-Pseudomonas activity (cefipime, ceftazidime) 2. Carbapenems with anti-Pseudomonas activity (imipenem, meropenem) 3. β-lactam with anti-Pseudomonas activity (cefoperazone / sulbactam, piperacillin / tazobactam, ticarcillin / clavulanate) + 1. Fluoroquinolones with anti-Pseudomonas activity (levofloxacin, ciprofloxacin) 2. Amikacin + Linezolid or vancomycin

BRONCHIAL ASTHMA

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation.

Table 25.

Classification of bronchial asthma

J45.0 Predominantly allergic asthma

Allergic:

- bronchitis NOS
- rhinitis with asthma

Atopic asthma

Extrinsic allergic asthma

Hay fever with asthma

J45.1 Nonallergic asthma

Idiosyncratic asthma

Intrinsic nonallergic asthma

J45.8 Mixed asthma

Combination of conditions listed in J45.0 and J45.1

J45.9 Asthma, unspecified

Asthmatic bronchitis NOS

Late-onset asthma

J46 Status asthmaticus

Incl.:

Acute severe asthma

NOS - Not Otherwise Specified - is a subcategory in systems of disease/disorder classification such as ICD-9, ICD-10. It is generally used to note the presence of an illness where the symptoms presented were sufficient to make a general diagnosis, but where a specific diagnosis was not made.

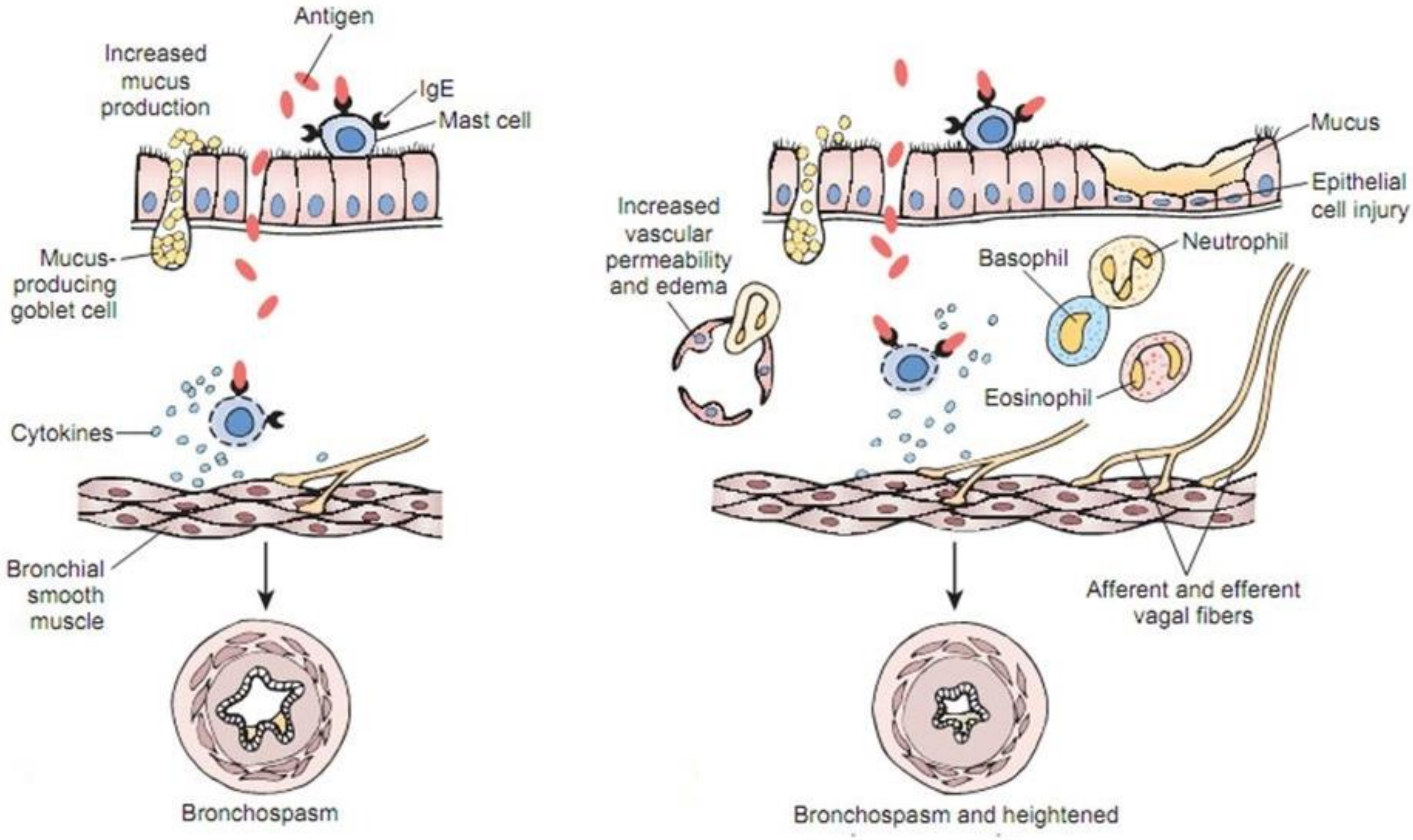


Fig. 31. Causes of bronchial asthma

Table 26.

Asthma triggers (agents and events)

Respiratory infection	RSV, rhinovirus, influenza, parainfluenza, *Mycoplasma pneumonia*
Allergens	Airborne pollens (grass, trees, weeds), house-dust mites, animal danders, cockroaches, fungal spores
Environment	Cold air, fog, ozone, sulfur dioxide, nitrogen dioxide, tobacco smoke, wood smoke
Emotions	Anxiety, stress, laughter
Exercise	Particularly in cold, dry environments
Drugs / preservatives	Aspirin, NSAIDs (cyclooxygenase inhibitors), sulfites, benzalkonium chloride, nonselective β-blockers
Occupational stimuli	Bakers (flour dust); farmers (hay mold); spice and enzyme workers; printers (arabic gum); chemical workers (azo dyes, anthraquinone, ethylenediamine, toluene diisocyanates, polyvinyl chloride); plastics, rubber, and wood workers (formaldehyde, western cedar, dimethylethanolamine, anhydrides)

Table 27.

Asthma severity classification

Clinical course, severity	Daytime asthma symptoms	Nighttime awakenings	FEV1, PEFR
Intermittent	< 1 /week	2 and < /month	>80% predicted. Daily variability < 20%
Mild persistent	≥ 1 /week but not daily	> 2 /month	>80% predicted. Daily variability – 20-30%
Moderate persistent	Daily	> 1 /week	60-80% predicted. Variability>30%.
Severe persistent	Persistent, which limit normal activity	Daily	<60% predicted. Variability > 30%.

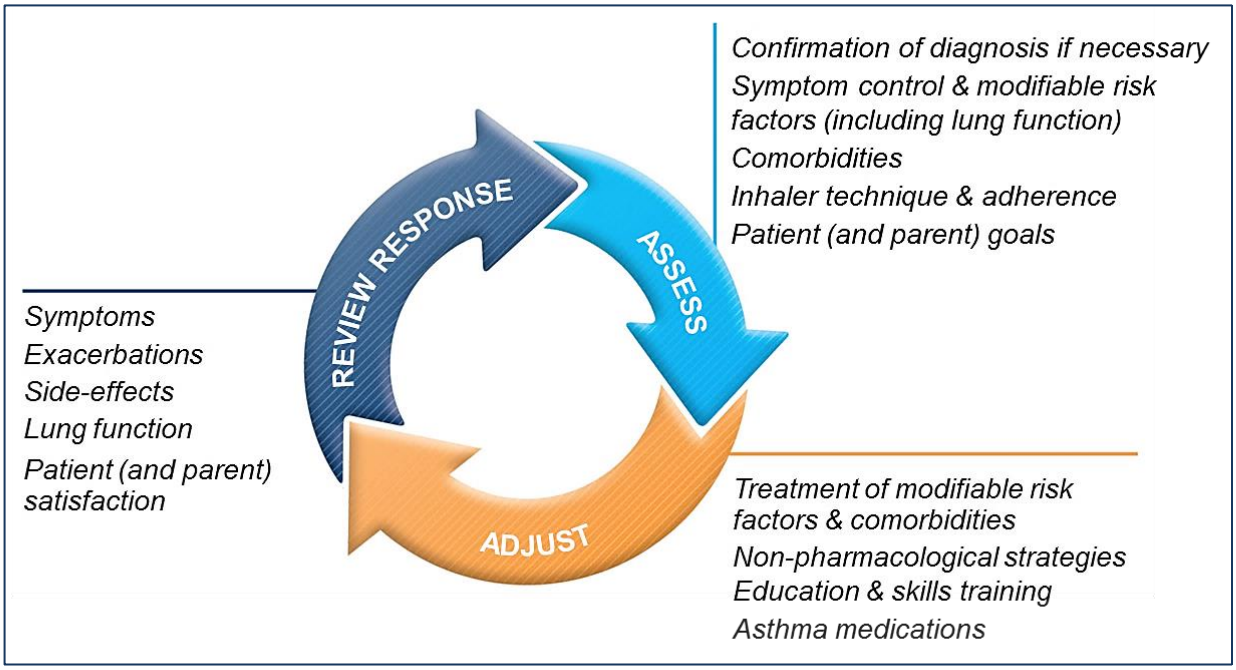


Fig. 32. The control-based asthma management cycle

Table 28.

Categories of asthma medications

Controller medications	these are used to reduce airway inflammation, control symptoms, and reduce risks of exacerbations and decline in lung function. In patients with mild asthma, controller treatment may be delivered through as-needed low dose ICS-formoterol
Reliever (rescue) medications	these are provided to all patients for as-needed relief of breakthrough symptoms, including during worsening asthma or exacerbations. They are also recommended for short-term prevention of exercise-induced bronchoobstruction. Reducing and eliminating the need for reliever treatment is both an important goal in asthma management
Add-on therapies for patients with severe asthma	these may be considered when patients have persistent symptoms and/or exacerbations despite optimized treatment with high dose controller medications (a high dose ICS and a LABA) and treatment of modifiable risk factors

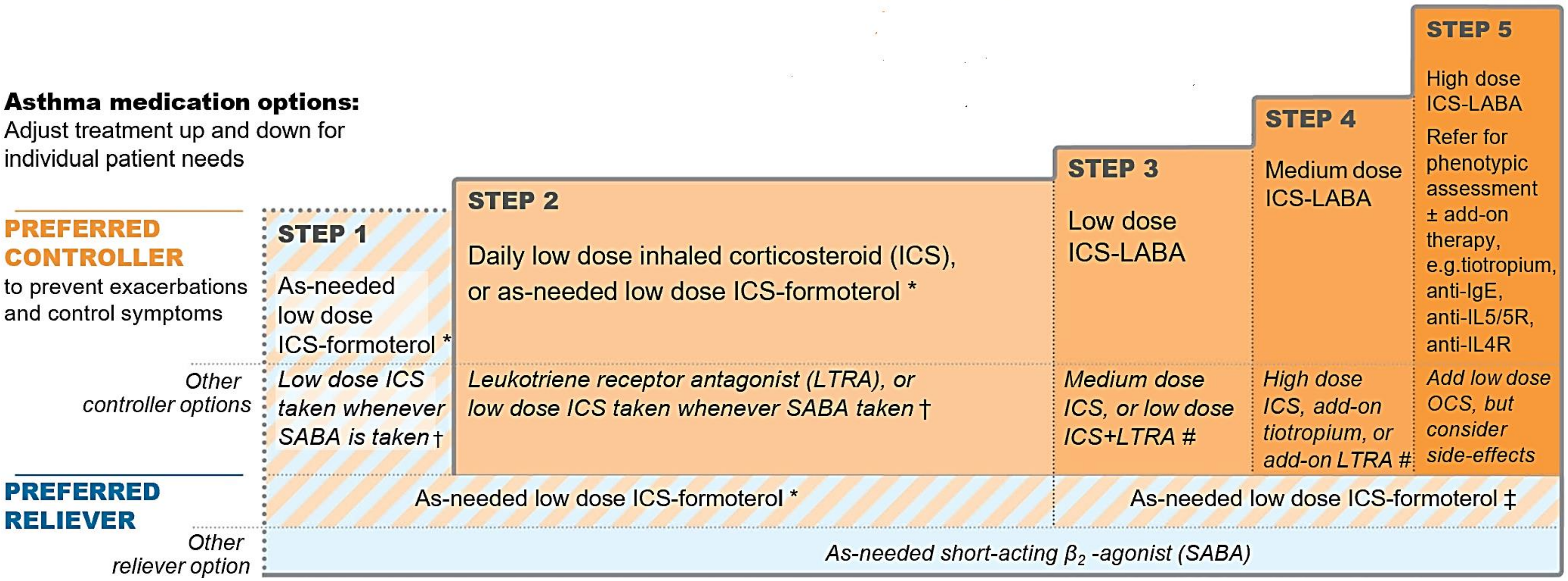


Fig. 33. Personalized management for adults and adolescents to control symptoms and minimize future risk

ICS – inhaled corticosteroids
OCS – oral corticosteroids
LABA – long-acting beta agonists
SABA – short-acting beta agonists
LTRA – Leukotriene receptor antagonist

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

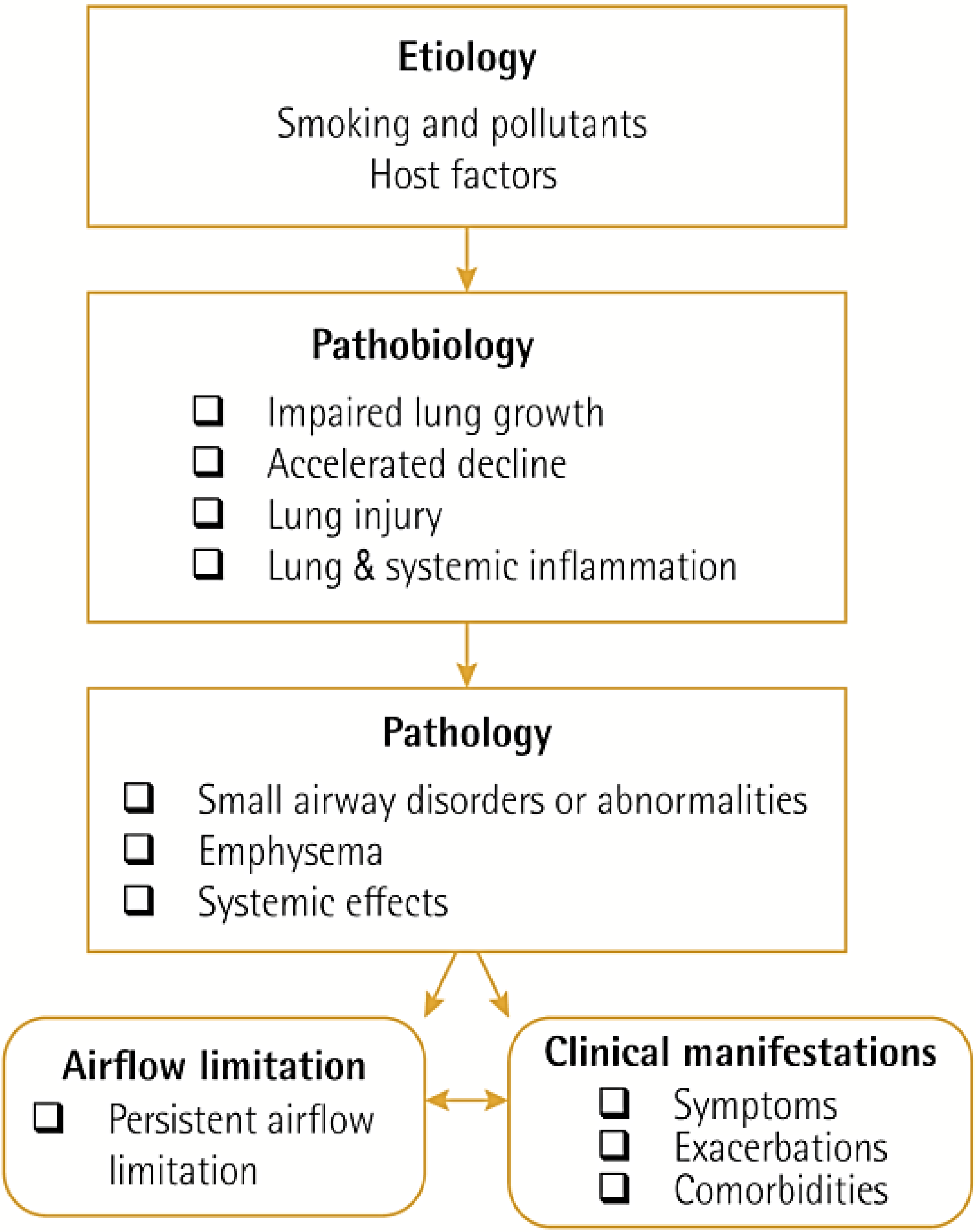


Fig. 34. Etiology, pathobiology and pathology of COPD leading to airflow limitation and clinical manifestations

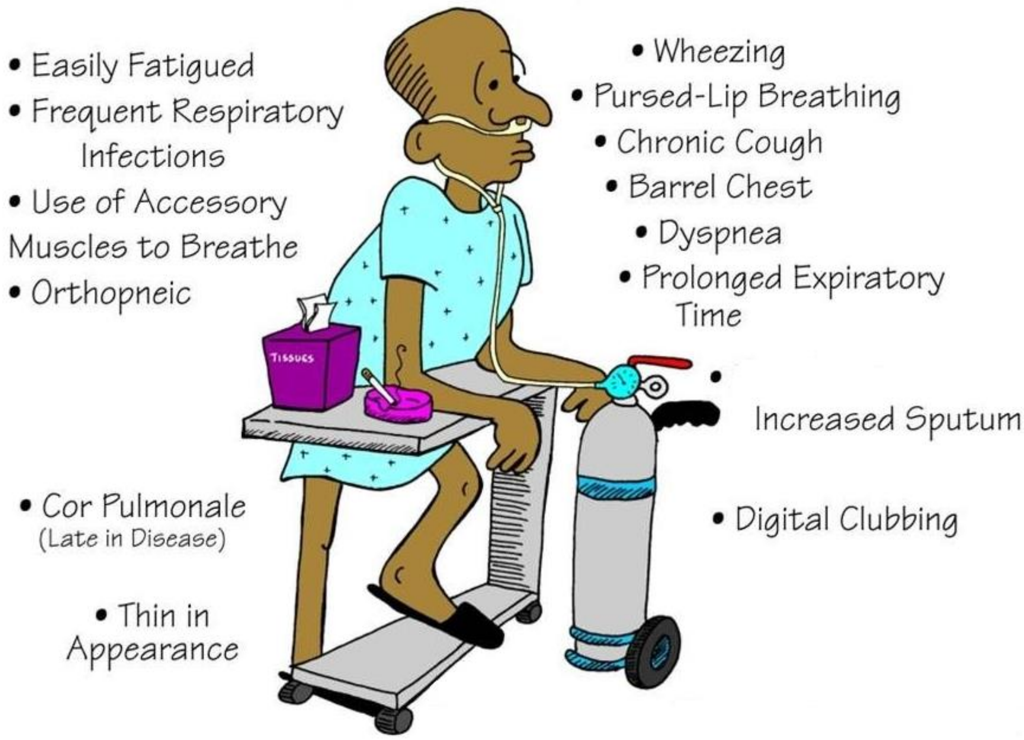


Fig. 35. Clinical symptoms of COPD

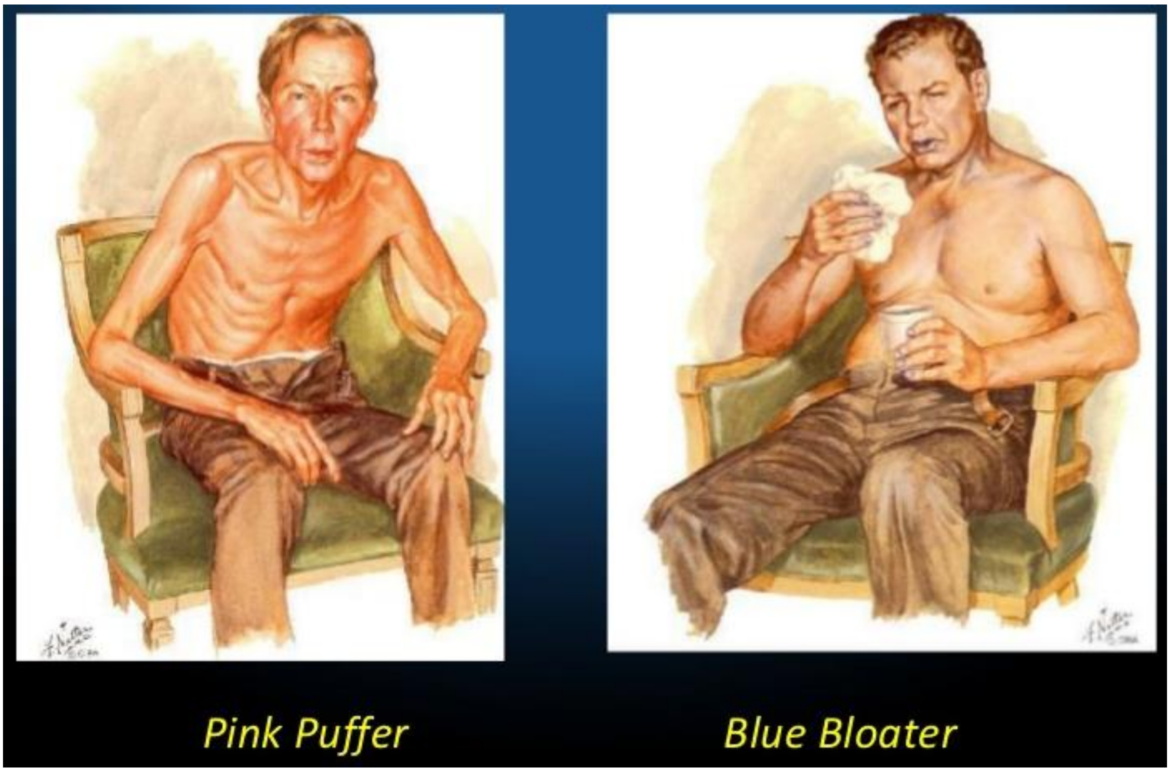


Fig. 36. Main phenotypes of COPD

Table 29.

Key indicators for considering a diagnosis of COPD

Consider COPD, and perform spirometry, if any of these indicators are present in an individual over age 40. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD. Spirometry is required to establish a diagnosis of COPD.	
Dyspnea that is:	Progressive over time. Characteristically worse with exercise. Persistent.
Chronic cough:	May be intermittent and may be unproductive. Recurrent wheeze.
Chronic sputum production:	Any pattern of chronic sputum production may indicate COPD.
Recurrent lower respiratory tract infections	
History of risk factors:	Host factors (such as genetic factors, congenital/developmental abnormalities etc.). Tobacco smoke (including popular local preparations). Smoke from home cooking and heating fuels. Occupational dusts, vapors, fumes, gases and other chemicals.
Family history of COPD and/or childhood factors:	For example low birthweight, childhood respiratory infections etc.

Table 30.

Values for FEV_1, FVC, FEV_1/FVC ratio indicating different spirometry patterns

Condition	Normal	Obstruction (Please refer to the note below ref severity)	Restriction	Combined
FVC	Above 80% predicted	Above 80% predicted	Below 80% predicted	Below 80% predicted
FEV_1	Above 80% predicted	Below 80% predicted	Below 80% predicted	Below 80% predicted
FEV_1/FVC Ratio	Above 70%	Below 70%	Above 70%	Below 70%

Table 35.

Classification of airflow limitation severity in COPD

In patients with $FEV_1/FVC < 0.70$:		
GOLD 1:	Mild	$FEV_1 \geq 80\%$ predicted
GOLD 2:	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3:	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4:	Very Severe	$FEV_1 < 30\%$ predicted

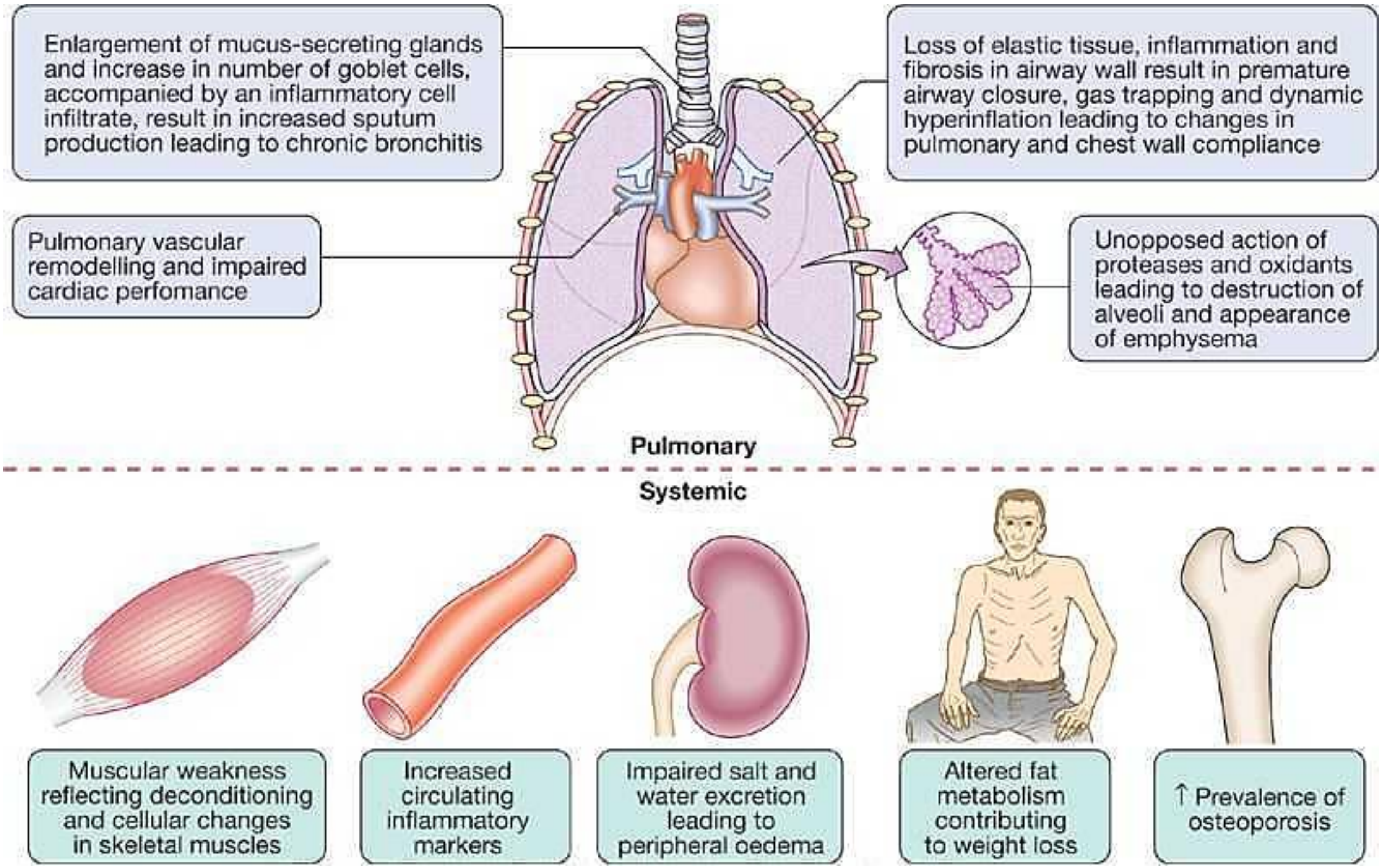


Fig. 37. Pulmonary and systemic presentations of COPD

For each item below, place a mark (X) in the box that best describes you currently. Be sure to only select one response for each question.

Example: I am very happy 0 ~~1~~ 2 3 4 5 I am very sad

			SCORE
I never cough	0 1 2 3 4 5	I cough all the time	
I have no phlegm (mucus) in my chest at all	0 1 2 3 4 5	My chest is completely full of phlegm (mucus)	
My chest does not feel tight at all	0 1 2 3 4 5	My chest feels very tight	
When I walk up a hill or one flight of stairs I am not breathless	0 1 2 3 4 5	When I walk up a hill or one flight of stairs I am very breathless	
I am not limited doing any activities at home	0 1 2 3 4 5	I am very limited doing activities at home	
I am confident leaving my home despite my lung condition	0 1 2 3 4 5	I am not at all confident leaving my home because of my lung condition	
I sleep soundly	0 1 2 3 4 5	I don't sleep soundly because of my lung condition	
I have lots of energy	0 1 2 3 4 5	I have no energy at all	
		TOTAL SCORE	

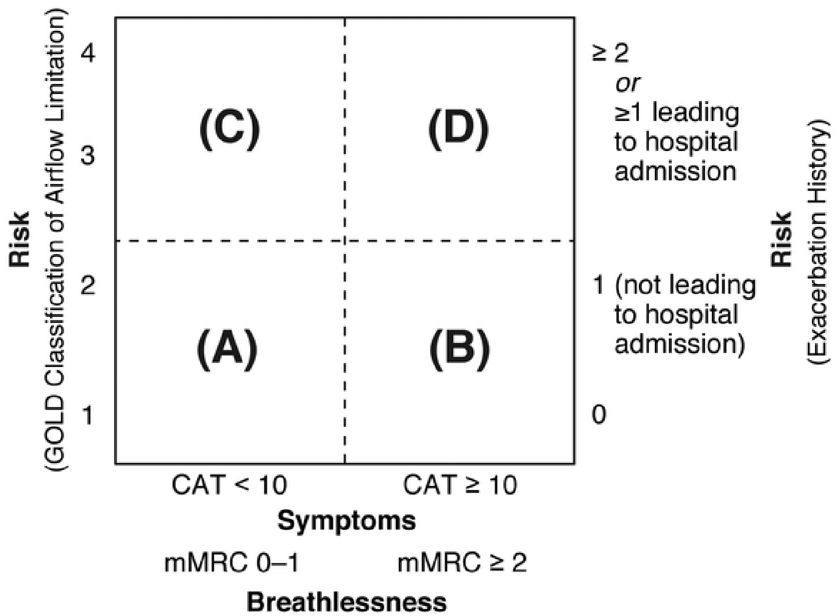
Fig. 38. COPD Assessment Test (CAT)

Table 31.

Modified MRC dyspnea scale

Grade of dyspnoea	Description
0	Not troubled by breathlessness except on strenuous exercise
1	Shortness of breath when hurrying on the level *or* walking up a slight hill
2	Walks slower than people of the same age on the level because of breathlessness *or* has to stop for breath when walking at own pace on the level
3	Stops for breath after walking about 100 m *or* after a few minutes on the level
4	Too breathless to leave the house *or* breathless when dressing or undressing

When assessing risk, choose the highest risk according to GOLD grade or exacerbation history. (One or more hospitalizations for COPD exacerbations should be considered high risk.)



Patient Category	Characteristics	Spirometric Classification	Exacerbations per Year	CAT	mMRC
A	Low Risk, Less Symptoms	GOLD 1–2	≤ 1	< 10	0–1
B	Low Risk, More Symptoms	GOLD 1–2	≤ 1	≥ 10	≥ 2
C	High Risk, Less Symptoms	GOLD 3–4	≥ 2	< 10	0–1
D	High Risk, More Symptoms	GOLD 3–4	≥ 2	≥ 10	≥ 2

Fig. 39. The refined ABCD assessment tool

Table 32.

Bronchodilators in stable COPD

- Inhaled bronchodilators in COPD are central to symptom management and commonly given on a regular basis to prevent or reduce symptoms **(Evidence A)**.
- Regular and as-needed use of SABA or SAMA improves FEV_1 and symptoms **(Evidence A)**.
- Combinations of SABA and SAMA are superior compared to either medication alone in improving FEV_1 and symptoms **(Evidence A)**.
- LABAs and LAMAs significantly improve lung function, dyspnea, health status, and reduce exacerbation rates **(Evidence A)**.
- LAMAs have a greater effect on exacerbation reduction compared with LABAs **(Evidence A)** and decrease hospitalizations **(Evidence B)**.
- Combination treatment with a LABA and LAMA increases FEV_1 and reduces symptoms compared to monotherapy **(Evidence A)**.
- Combination treatment with a LABA and LAMA reduces exacerbations compared to monotherapy **(Evidence B)** or ICS/LABA **(Evidence B)**.
- Tiotropium improves the effectiveness of pulmonary rehabilitation in increasing exercise performance **(Evidence B)**.
- Theophylline exerts a small bronchodilator effect in stable COPD **(Evidence A)** and that is associated with modest symptomatic benefits **(Evidence B)**.

Table 33.

Anti-inflammatory therapy in stable COPD

Inhaled corticosteroids
• An ICS combined with a LABA is more effective than the individual components in improving lung function and health status and reducing exacerbations in patients with exacerbations and moderate to very severe COPD **(Evidence A)**.
• Regular treatment with ICS increases the risk of pneumonia especially in those with severe disease **(Evidence A)**.
• Triple inhaled therapy of ICS/LAMA/LABA improves lung function, symptoms and health status **(Evidence A)** and reduces exacerbations **(Evidence B)** compared to ICS/LABA or LAMA monotherapy.
Oral glucocorticoids
• Long-term use of oral glucocorticoids has numerous side effects **(Evidence A)** with no evidence of benefits **(Evidence C)**.
PDE4 inhibitors
• In patients with chronic bronchitis, severe to very severe COPD and a history of exacerbations: » A PDE4 inhibitor improves lung function and reduces moderate and severe exacerbations **(Evidence A)**. » A PDE4 inhibitor improves lung function and decreases exacerbations in patients who are on fixed-dose LABA/ICS combinations **(Evidence B)**.
Antibiotics
• Long-term azithromycin and erythromycin therapy reduces exacerbations over one year **(Evidence A)**.
• Treatment with azithromycin is associated with an increased incidence of bacterial resistance **(Evidence A)** and hearing test impairments **(Evidence B)**.
Mucolytics/antioxidants
• Regular use of NAC and carbocysteine reduces the risk of exacerbations in select populations **(Evidence B)**.
Other anti-inflammatory agents
• Simvastatin does not prevent exacerbations in COPD patients at increased risk of exacerbations and without indications for statin therapy **(Evidence A)**. However, observational studies suggest that statins may have positive effects on some outcomes in patients with COPD who receive them for cardiovascular and metabolic indications **(Evidence C)**.
• Leukotriene modifiers have not been tested adequately in COPD patients.

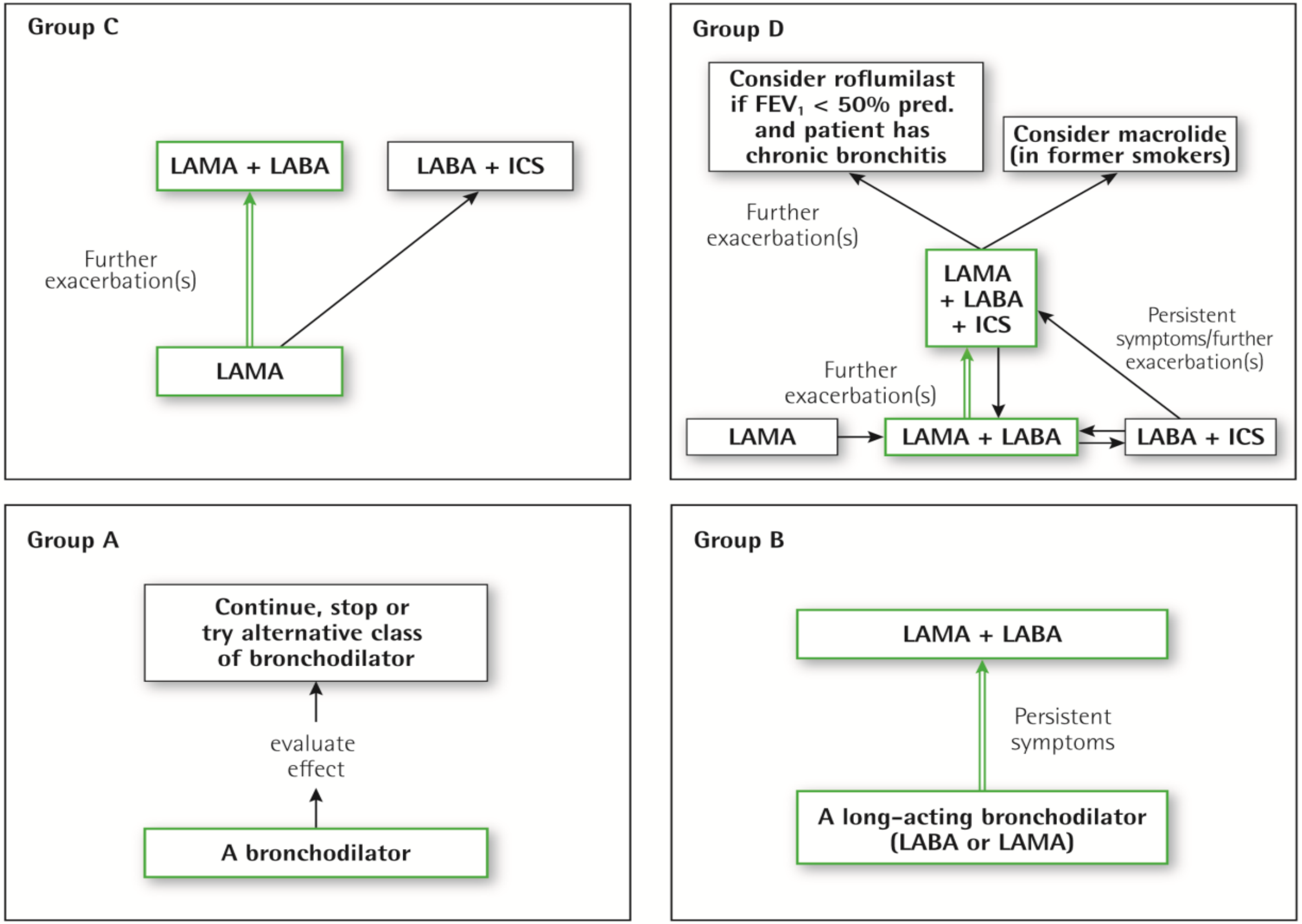


Fig. 40. Pharmacological treatment algorithm by GOLD grade. ICS – inhaled corticosteroids, LABA – long-acting beta agonist, LAMA - Long-acting muscarinic antagonist, LTRA – Leukotriene receptor antagonist

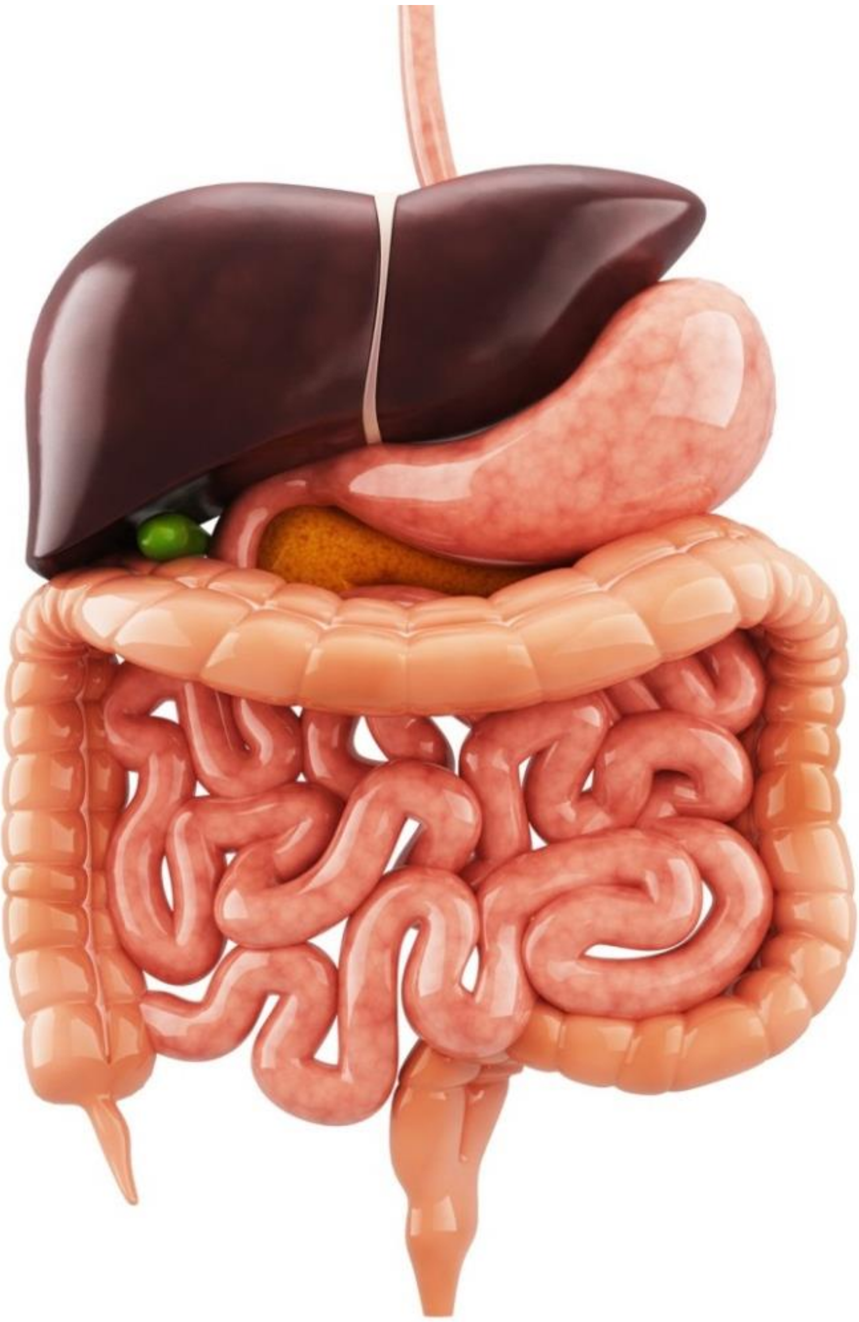
Table 34.

Spirometric measures in asthma, COPD and asthma-COPD overlap

Spirometric variable	Asthma	COPD	Asthma-COPD overlap
Normal FEV_1/FVC pre- or post BD	Compatible with diagnosis	Not compatible with diagnosis	Not compatible with diagnosis
Post-BD FEV_1/FVC <0.7	Indicates airflow limitation but may improve spontaneously or on treatment	Required for diagnosis by GOLD criteria	Usually present
Post-BD FEV_1 ≥80% predicted	Compatible with diagnosis (good asthma control or interval between symptoms)	Compatible with GOLD classification of mild airflow limitation if post-BD FEV /FVC <0.7	Compatible with diagnosis of mild ACO
Post-BD FEV_1 <80% predicted	Compatible with diagnosis. Risk factor for asthma exacerbations	An indicator of severity of airflow limitation and risk of future events (e.g. mortality and COPD exacerbations)	An indicator of severity of airflow limitation and risk of future events (e.g. mortality and exacerbations)
Post-BD increase in FEV_1 ≥12% and 200 ml from baseline (reversible airflow limitation).	Usual at some time in course of asthma, but may not be present when well-controlled or on controllers	Common and more likely when FEV_1 is low	Common and more likely when FEV_1 is low
Post-BD increase in FEV_1 >12% and 400ml from baseline (marked reversibility)	High probability of asthma	Unusual in COPD. Consider ACO	Compatible with diagnosis of ACO

ACO - asthma-COPD overlap; BD - bronchodilator; FEV_1 - forced expiratory volume in 1 second; FVC - forced vital capacity; GOLD - Global Initiative for Obstructive Lung Disease.

GASTROENTEROLOGY



CHRONIC GASTRITIS

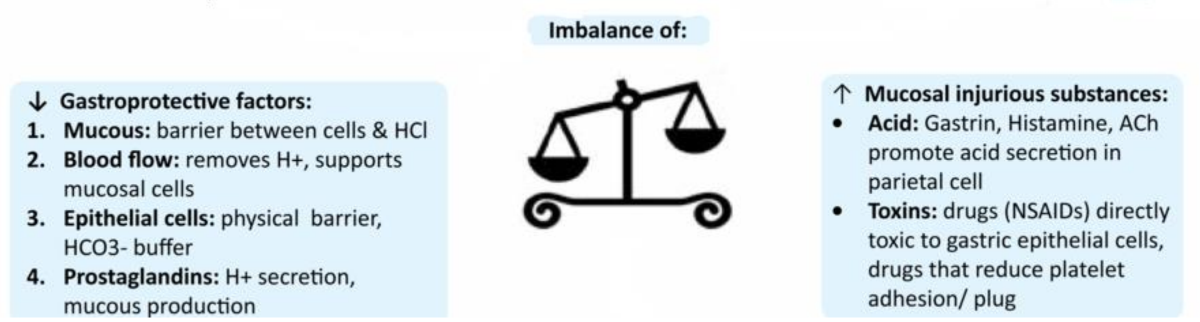


Fig. 41. Agressive and protective factors in pathogenesis of chronic gastritis and ulcer disease.
HCl - hydrochloric acid
NSAIDs - Nonsteroidal anti-inflammatory drugs

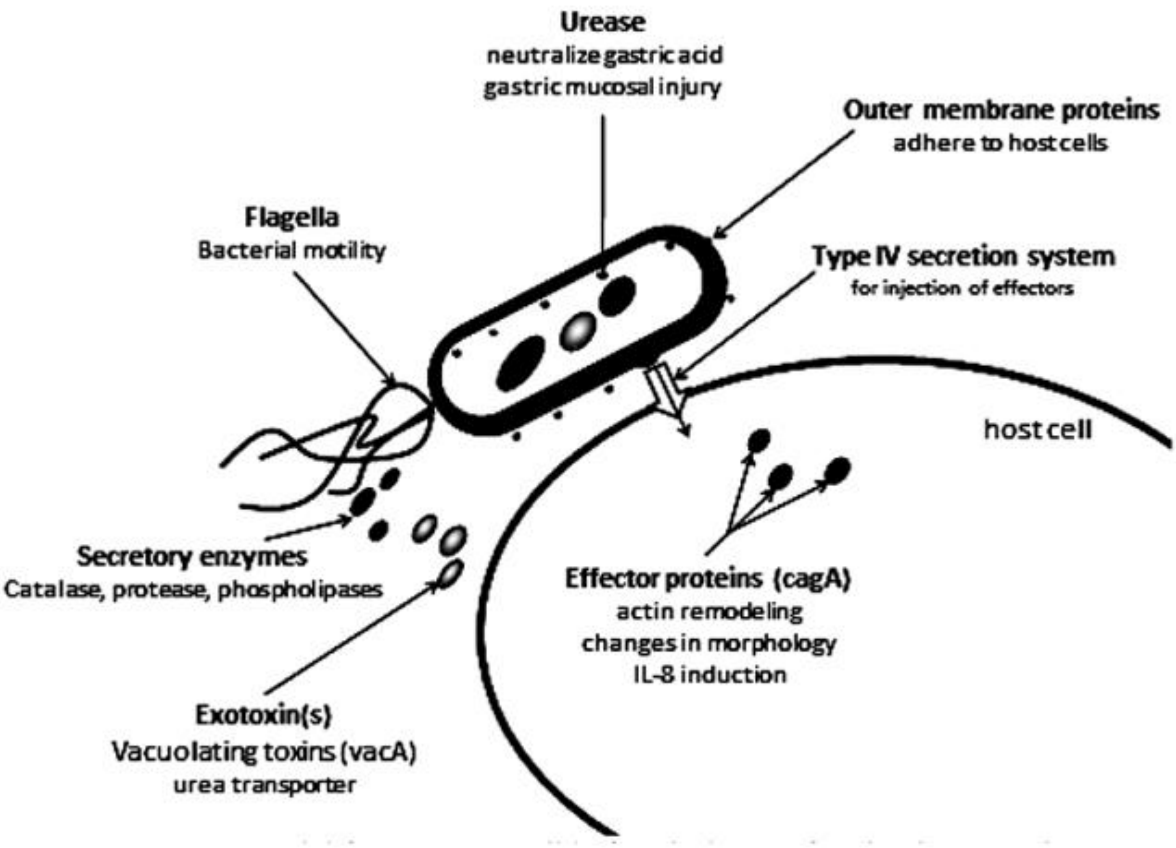


Fig. 42. Bacterial factors responsible for virulence of Helicobacter pylori

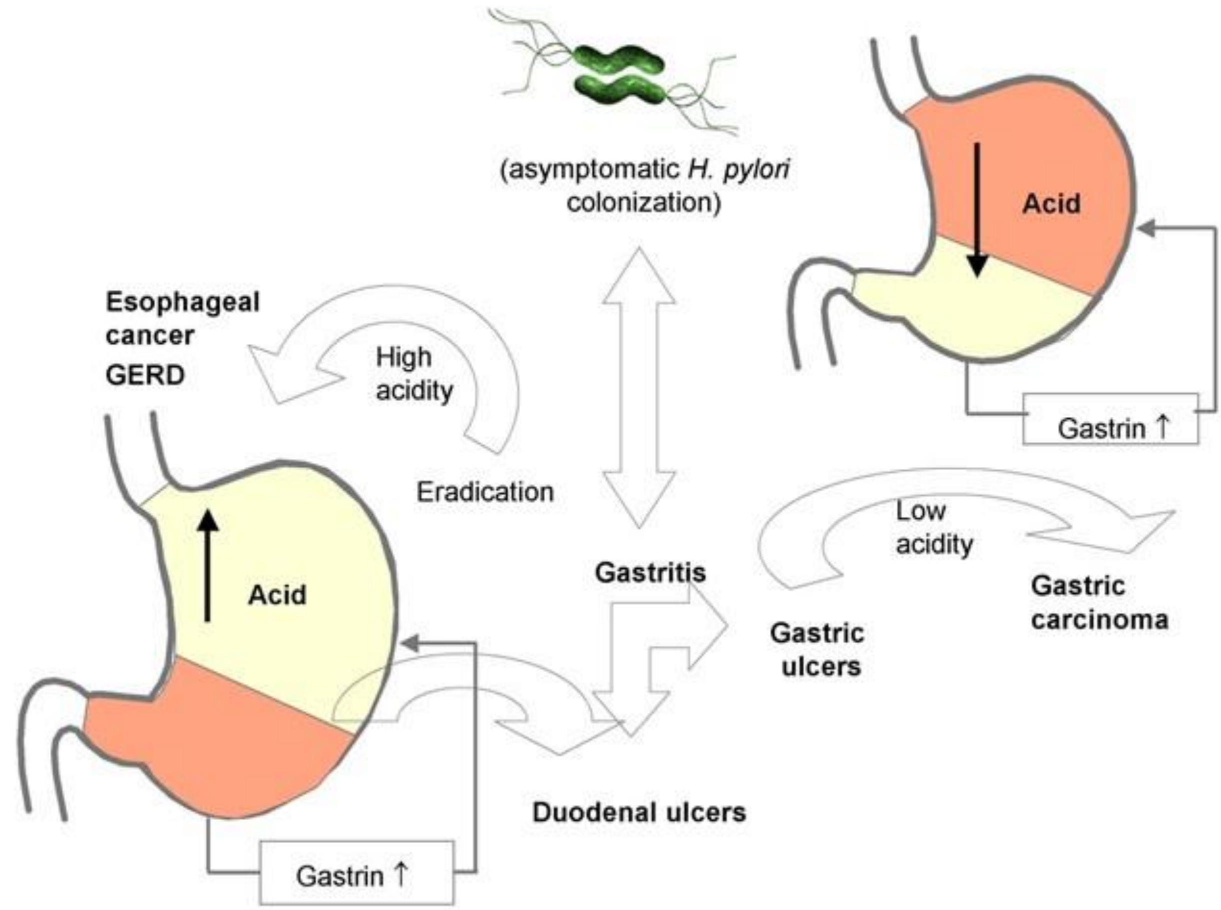


Fig. 43. H.Pylori disease profile

Fig. 44. ^{13}C-Urea Breath Test Kit

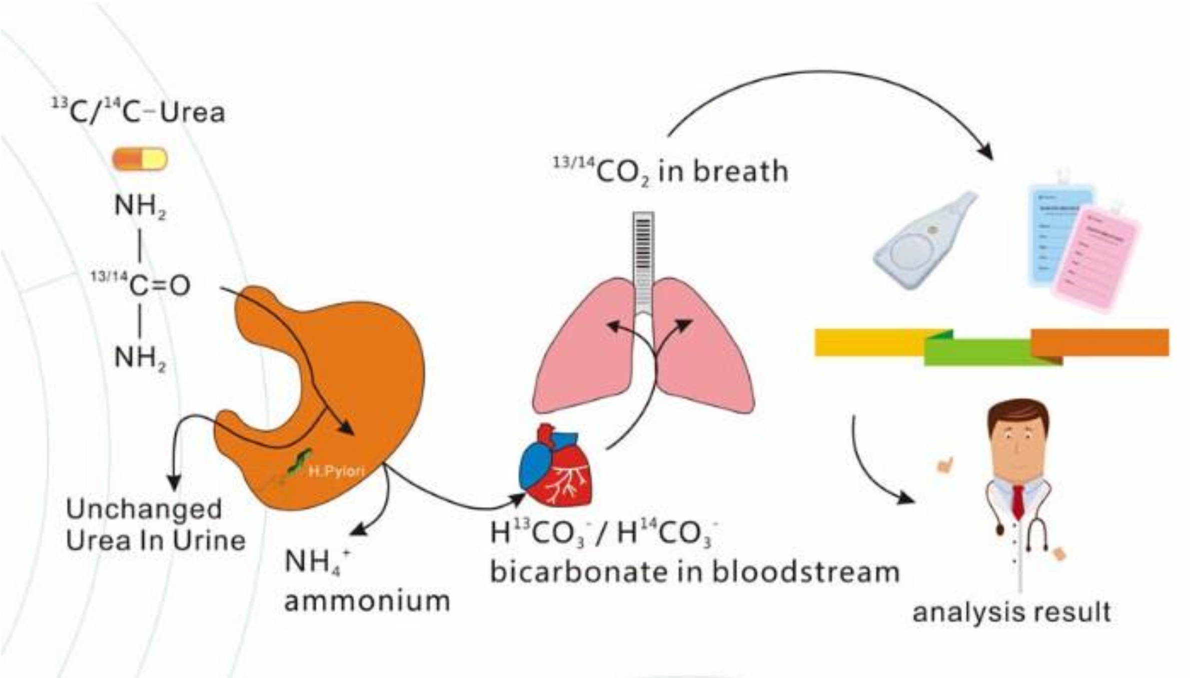


Fig. 45. Principle of $^{13}C/^{14}C$-Urea Breath Test Kit

GASTRIC AND DUODENAL ULCER

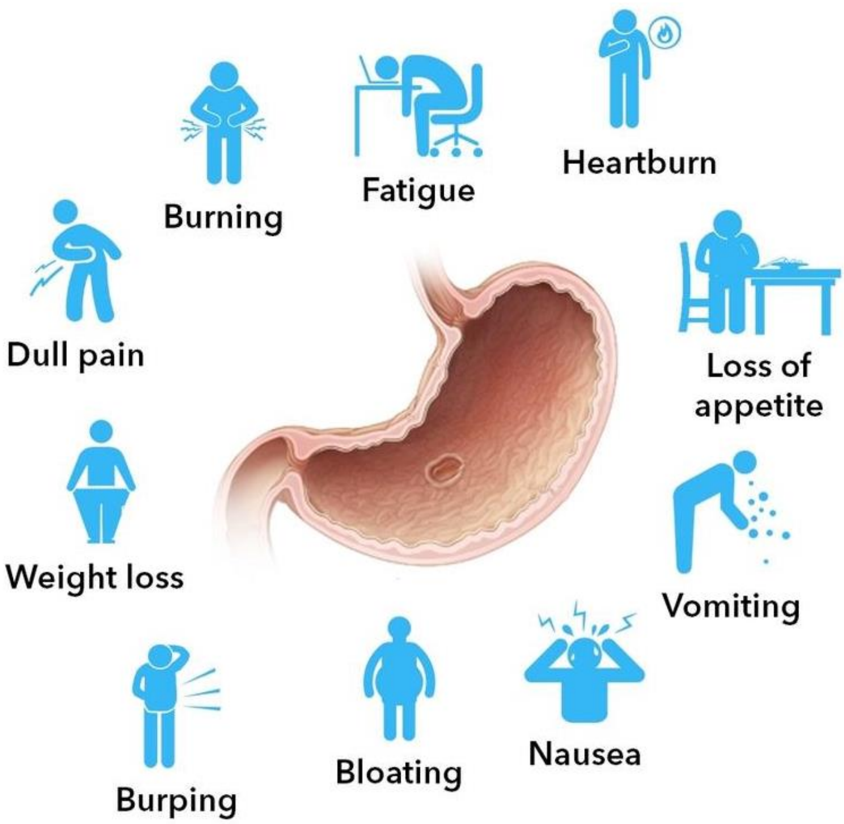


Fig. 46. Stomach ulcer symptoms

Table 35.

Differential diagnostics in ulcer disease

Duodenal ulcer	Gastric ulcer
Pain relieved by meal	Pain increased by meal
Occurs 2-3 hrs after meal	Occurs 30 min to 1hr after meal
Most common type	Not as common
Dark, tarry stools (Melena) occur	Vomiting occurs

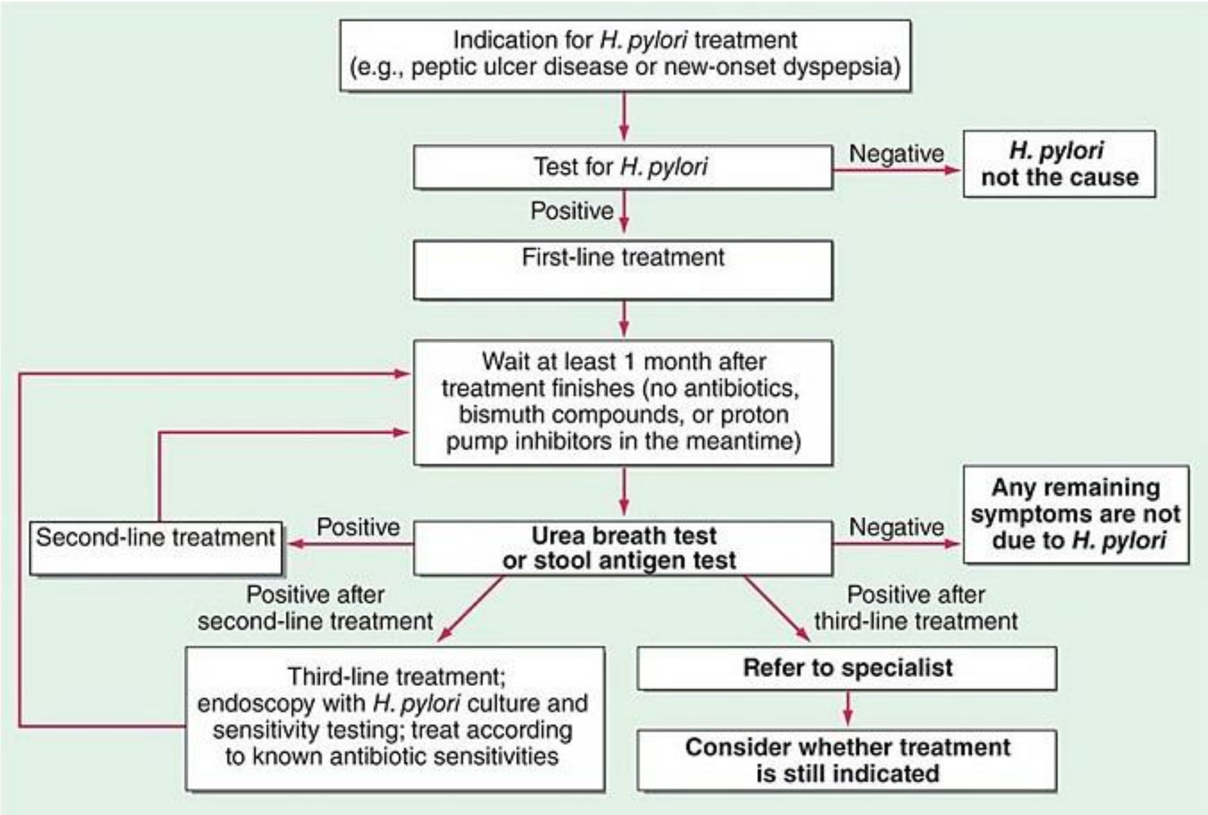


Fig. 47. Indication for H.pylori treatment

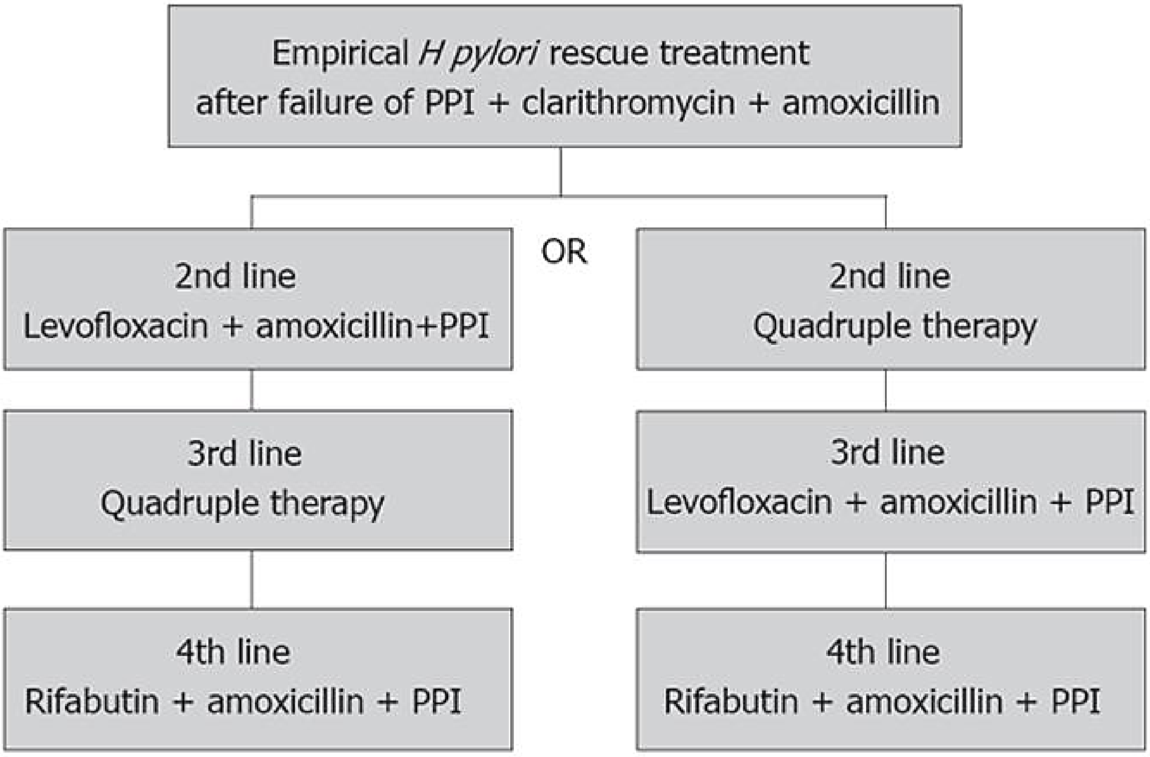


Fig. 48. Algorithm of eradication therapy

CHRONIC CHOLECYSTITIS

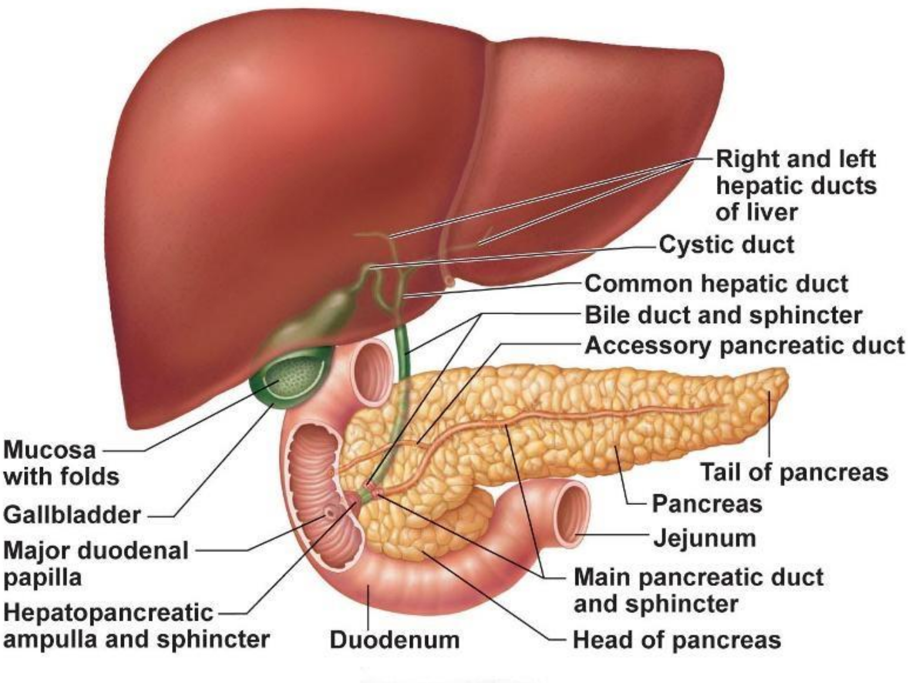


Fig. 49. Anatomy of hepatobiliary system

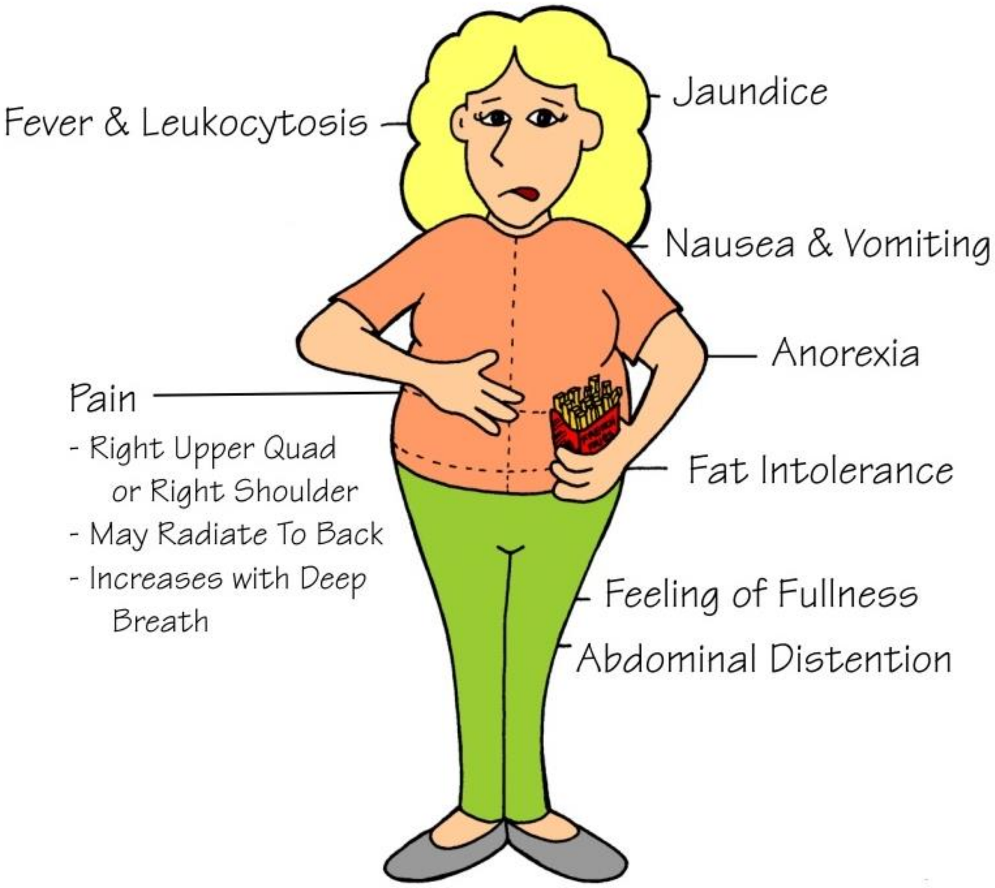


Fig.50. Clinical symptoms of chronic cholesystitis

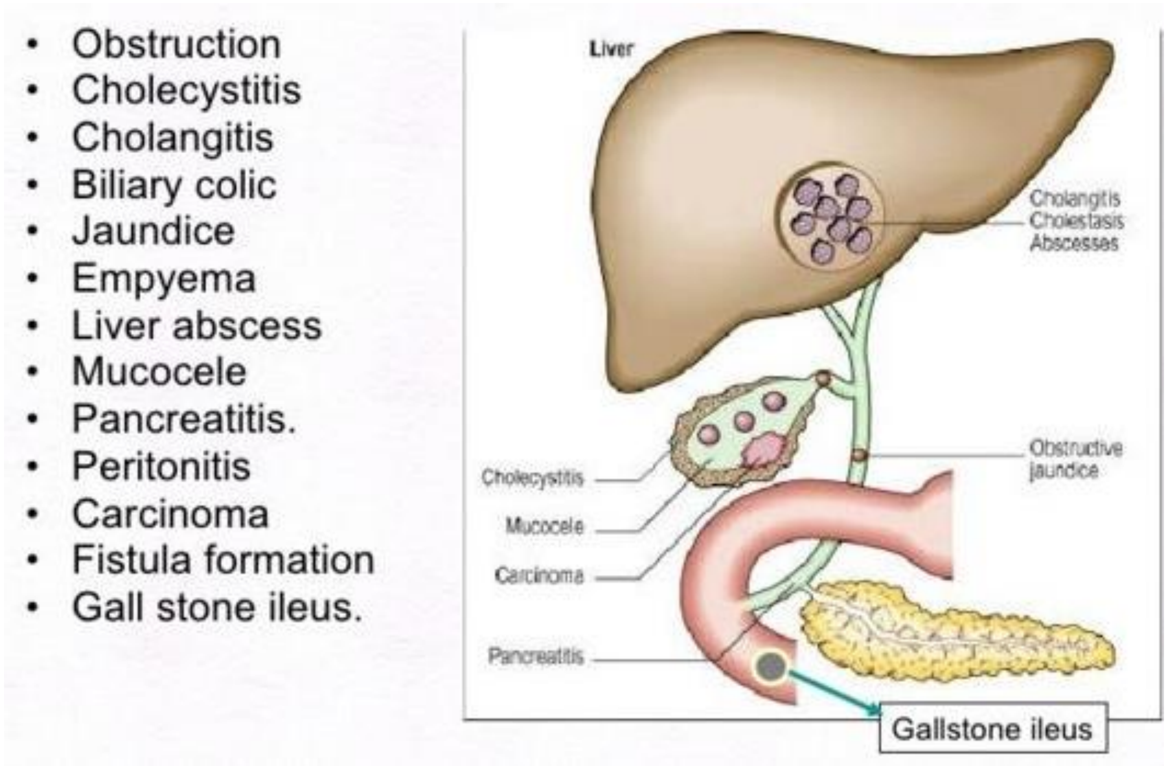


Fig. 51. Complications of cholecystitis

CHOLELITHIASIS

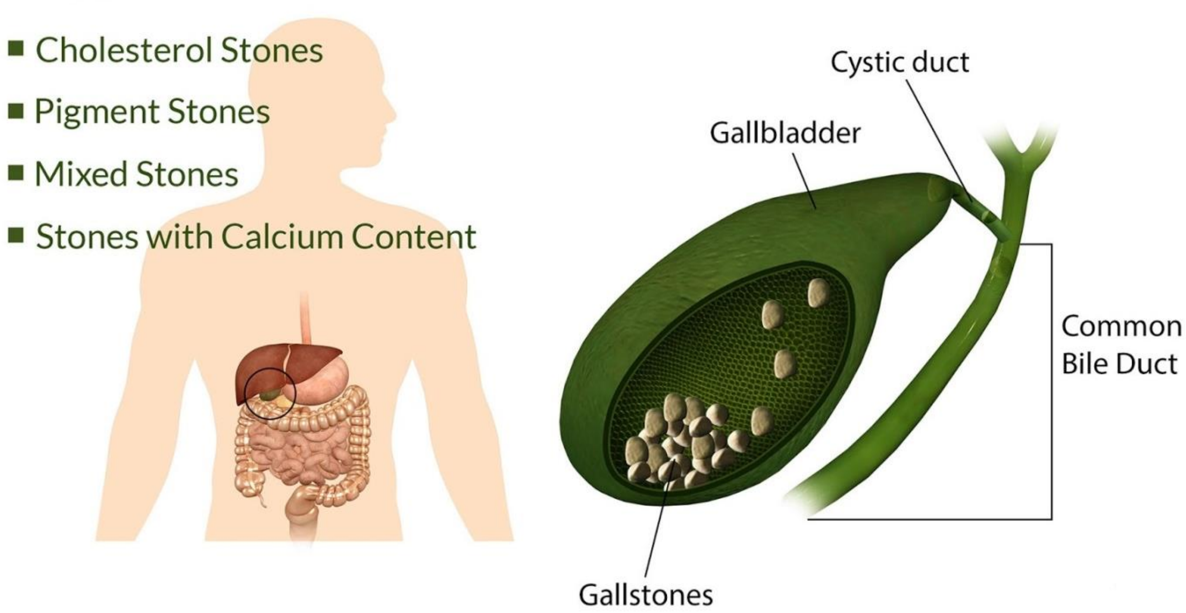


Fig. 52. Gall bladder stones, classification

CHRONIC PANCREATITIS

- Alcohol abuse
- Gallstones
- Tropical pancreatitis
- Toxic or metabolic pancreatitis
- Traumatic pancreatitis (e.g., caused by endoscopic retrograde cholangiopancreatography or surgery)
- Congenital pancreaticobiliary malformations (pancreas divisum, pancreaticobiliary maljunction, choledochocele, annular pancreas)
- Autoimmune pancreatitis
- Hereditary pancreatitis, such as cationic trypsinogen (serine protease 1 or PRSS1), serine protease inhibitor Kazal type 1 (SPINK1) and cystic fibrosis transmembrane conductance regulator
- Vascular or ischemic pancreatitis
- Post-irradiation pancreatitis

Fig. 53. Etiology of chronic pancreatitis

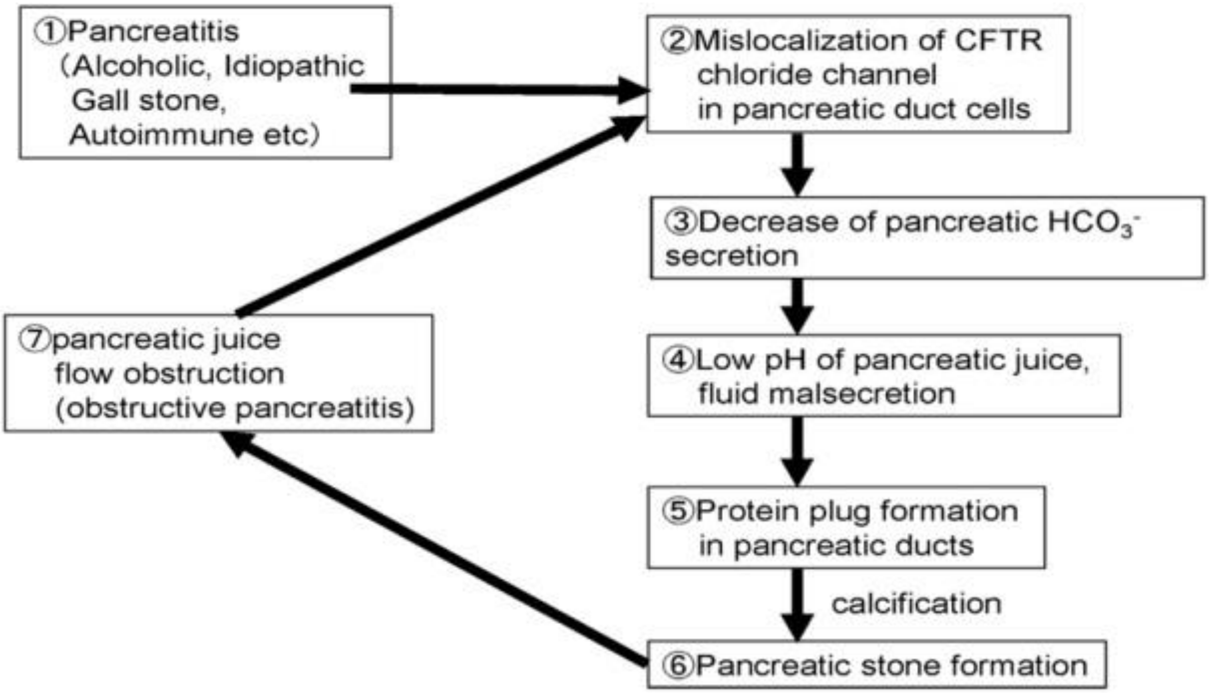


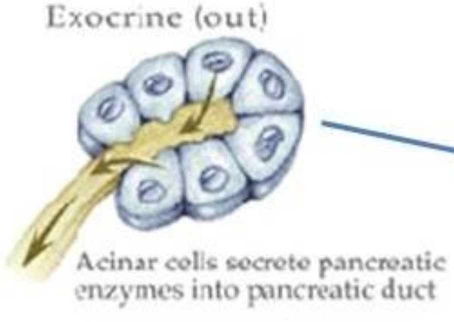
Fig. 54. Pathogenesis of chronic pancreatitis

Causes of **Pancreatitis**: (**BAD HITS**)

- **B**iliary
- **A**lcohol
- **D**rugs (Corticosteroids, HIV drugs, Diuretics, Valproic acid…)
- **H**ypertriglyceridemia/**H**ypercalcemia
- **I**diopathic
- **T**rauma
- **S**corpion sting

Fig. 55. Causes of pancreatitis (mnemonic)

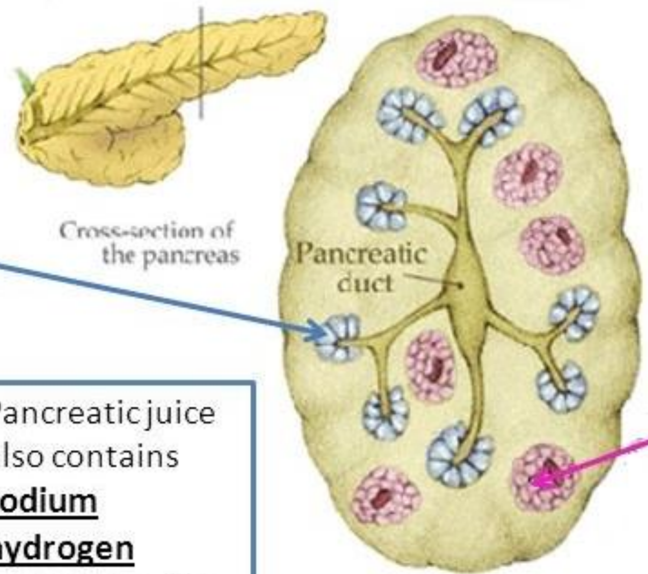
The <u>exocrine</u> function is the <u>release of enzymes.</u>



<u>Acinar cells</u> secrete **<u>pancreatic juice</u>** into the ***duodenum of the small intestine***. The pancreatic juice contains 3 types of enzymes:

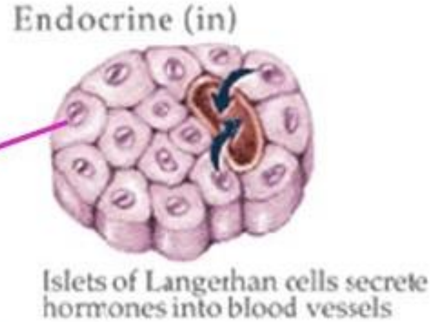
1. **<u>Amylose</u>** – breaks down starch into maltose
2. **<u>Trypsinogen</u>** – breaks down proteins into shorter polypeptides
3. **<u>Lipase</u>** – breaks down lipids onto glycerol and fatty acids.

Pancreatic juice also contains **<u>sodium hydrogen carbonate</u>**. This ***neutralises*** the acidic chyme entering the duodenum from the stomach, because enzymes there have an ***optimal pH*** of neutral or just above.



The **<u>α cells</u>** and **<u>β cells</u>** are specialised to manufacture and secrete hormones. They have **<u>lots of rough endoplasmic reticulum</u>** for protein synthesis; **<u>lots of golgi,</u>** for the modification and packaging of insulin and glucagon; **<u>lots of vesicles</u>** for secretion of hormones by exocytosis.

The <u>endocrine</u> function is the <u>secretion of hormones – glucagon and insulin.</u>



The **<u>islets of Langerhans</u>** are small patches of tissue in the pancreas **<u>containing α cells that secrete glucagon</u>** and **<u>β cells that secrete insulin.</u>**

Fig. 56. Endocrine and exocrine functions of pancreas

- Steatorrhea and azotorrhea (protein maldigestion) do not usually occur until pancreatic enzyme secretion is reduced to less than 10% of the maximum output

- Advanced chronic pancreatitis, maldigestion of fat, protein, and carbohydrates occur - present with diarrhea and weight loss

- median time to development of exocrine insufficiency was 13.1 years in patients with alcoholic chronic pancreatitis

- Deficiencies of fat-soluble vitamins
- Significant vitamin D deficiency and osteopenia or even osteoporosis occur
- Bleeding manifestations

Fig. 57. Symptoms of exocrine insufficiency

Endocrine insufficiency :

- Chronic pancreatitis also affects islet cell populations - 40% to 80% of patients will have clinical manifestations of *diabetes mellitus*
- Islet cells appear to be relatively resistant to destruction in chronic pancreatitis - Diabetes mellitus typically manifests late

Fig. 58. Symptoms of endocrine insufficiency

Extrapancreatic complications

- *Jaundice* may be seen in the presence of coexistent alcoholic liver disease or bile duct compression within the head of the pancreas. & duodenal obstruction
- A *palpable spleen* may also rarely be found in patients with thrombosis of the splenic vein as a consequence of chronic pancreatitis or in patients with portal hypertension due to coexistent chronic liver disease.

Fig. 59. Symptoms of extrapancreatic presentations

	NORMAL PANCREAS	EARLY CHRONIC PANCREATITIS	LATE CHRONIC PANCREATITIS
Clinical features		• Acute relapsing pancreatitis • Chronic or recurrent pain	• Chronic or recurrent pain • Pancreatic exocrine insufficency (PEI) • Pancreatogenic diabetes (DM) • Secondary pancreatic cancer
Diagnostic testing		• US and CT usually non-diagnostic • Endoscopic ultrasound • Secretin - MRCP • Pancreatic function tests	• CT usually diagnostic • MRI/MRCP usually diagnositic • Fecal elastase for PEI • HbA1c for DM
Therapy		• Medical therapy for pain • Avoid toxins (ETOH and tobacco) • Analgesics: – non narcotic – low potency narcotics (tramadol) • Gabapentoids, SSRIs, or TCAs	• Continued medical therapy for pain • Endoscopic or surgical therapy for pain – if pancreatic duct is dilated • Pancreatic enzyme replacement therapy – Add vitamin D and calcium – Nutritional assessment and monitoring • Therapy for DM
TIME		3-5 YEARS	5-10 YEARS AND BEYOND

Fig. 60. Diagnostics and treatment of chronic pancreatitis

Table 36.

Consequences of exocrine pancreatic insufficiency

Deficiency	Outcome
Vitamin A	Night blindness
Vitamin D	Osteomalacia, hypocalcemia
Vitamin E	Neuropathy, hemolytic anemia
Vitamin K	Coagulopathy
Fat malabsorbtion	Weight loss
Hypoproteinemia	Edema
Carbonhydrate malabsorbtion	Bloating, diarrhea

CHRONIC HEPATITIS

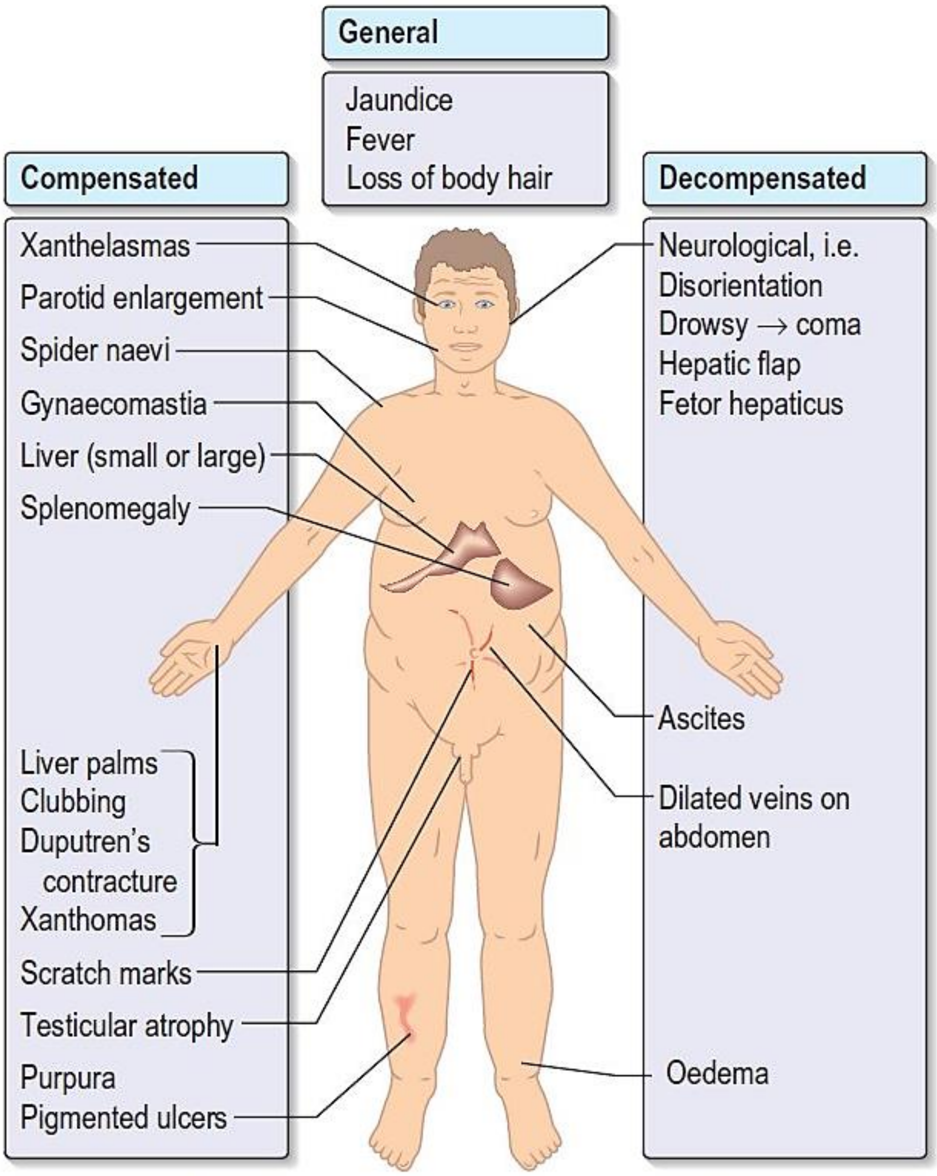


Fig. 61. Physical signs in chronic liver disease

LIVER CIRRHOSIS

Table 37.

Etiology of liver cirrhosis

Cause	Testing
Alcoholic liver disease	History, AST / ALT > 2
Chronic hepatitis C	Hep C Ab, Viral load
Primary biliary cirrhosis	Antimitochondrial antibodies
Primary sclerosing cholangitis	Contrast cholangiography, ANA, Anti smooth muscle Ab, ANCA
Autoimmune hepatitis	Type 1: ANA, ANCA antismooth muscle Ab Type 2: anti-LKM-1
Chronic hepatitis B	Hepatitis B serologies
Hemochromatosis	Ferritin, genetic testing
Wilson's disease	Ceruloplasmin
Alpha-1-antitrypsin deficiency	Serum AAT
Nonalcoholic fatty liver disease	Hx of DM or metabolic syndrome

Table 38.

Comparative characteristics of hepatitis and cirrhosis

Characteristic	Hepatitis	Cirrhosis
Cellular Behavior	Hepatocytes are infected with viral hepatitis. Innate immune response occurs; inflammatory cytokines result; some cytolytic T-cell response	Oxidative damage to hepatocytes; increase in stellate cell activation; deposition of collagen and other connective fibers
Clinical Manifestations	Acute and chronic; fever like conditions, jaundice, cirrhosis	Chronic by nature; palmar erythema, liver enlargement and nodular liver tissue
Cause of Infection/ Disease	Viral, autoimmune	Viral (caused by chronic hepatitis), behavioral (alcohol consumption), genetic
Mode of Diagnosis	Serologic patterns of antibody presence and RNA load, liver biopsy	Liver biopsy; moderate elevation of liver enzymes; fibrosis detection
Major Complications of Disease	Relapsing hepatitis, ALT and other liver enzyme elevation, jaundice of eyes and skin	Portal hypertension; hemorrhaging; reversible scar tissue formation

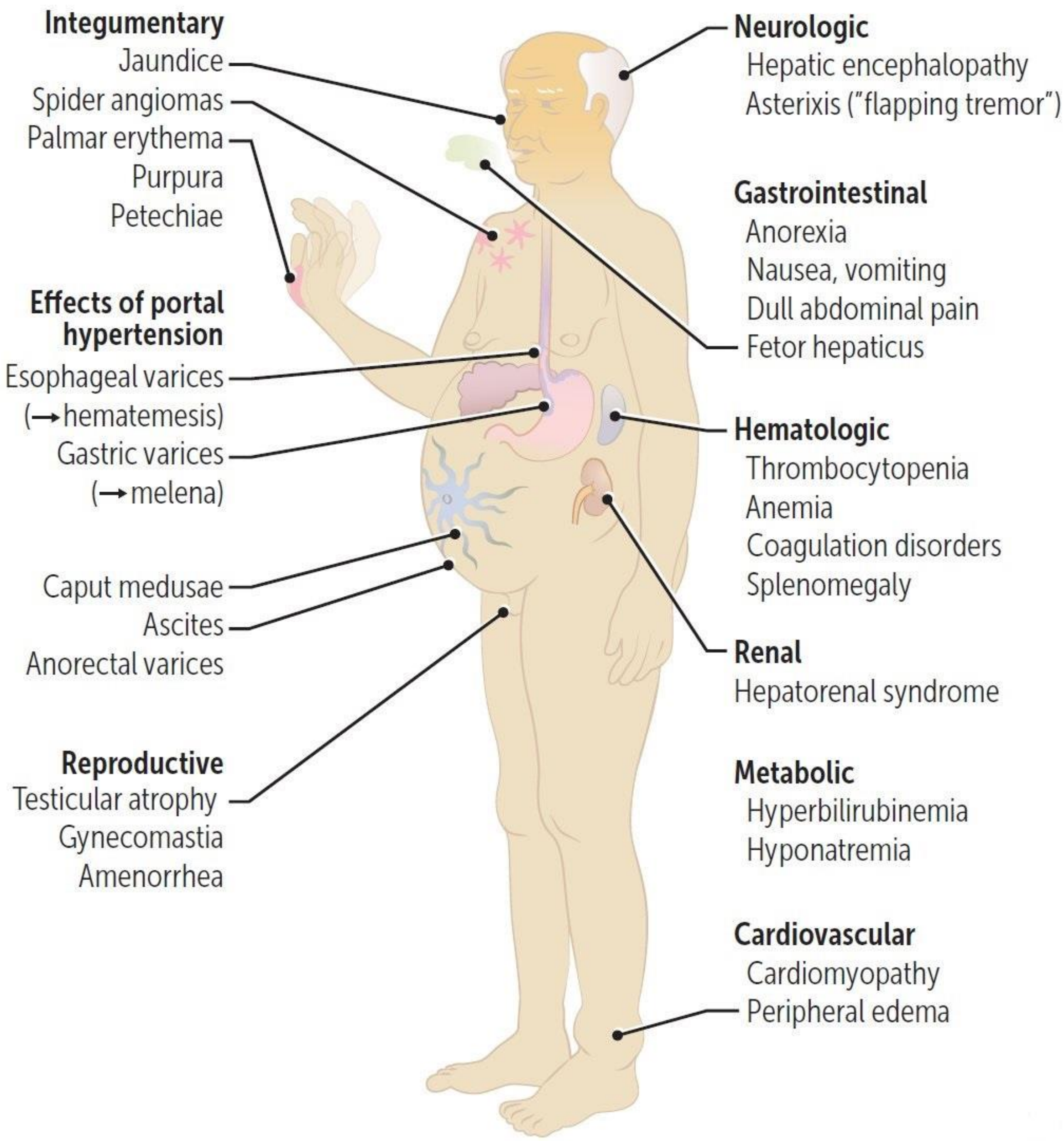


Fig. 62. Sighs of liver cirrhosis

Table 39.

Child-Pugh Score and interpretation

Clinical and laboratory criteria	Points		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	<2	2-3	>3
Albumin (g/dL)	>3,5	2,8-3,5	<2,8
Prothrombin time Seconds prolonged International normalized ratio	 <4 <1,7	 4-6 1,7-2,3	 >6 >2,3
Class A – 5 to 6 points (least severe liver disease) Class B – 7 to 9 points (moderately severe liver disease) Class c – 10 to 15 points (most severe liver disease)			

Table 40.

Syndromes in the clinical presentation of chronic liver disease

- Cytolytic syndrome
- Cholestatic syndrome
- Hepatocellular insufficiency
- Mesenchymal inflammatory syndrome
- Hypersplenism
- Pathological Regeneration and Tumor Growth Syndrome
- Portal hypertension
- Hemorrhagic syndrome
- Asthenoneurotic syndrome

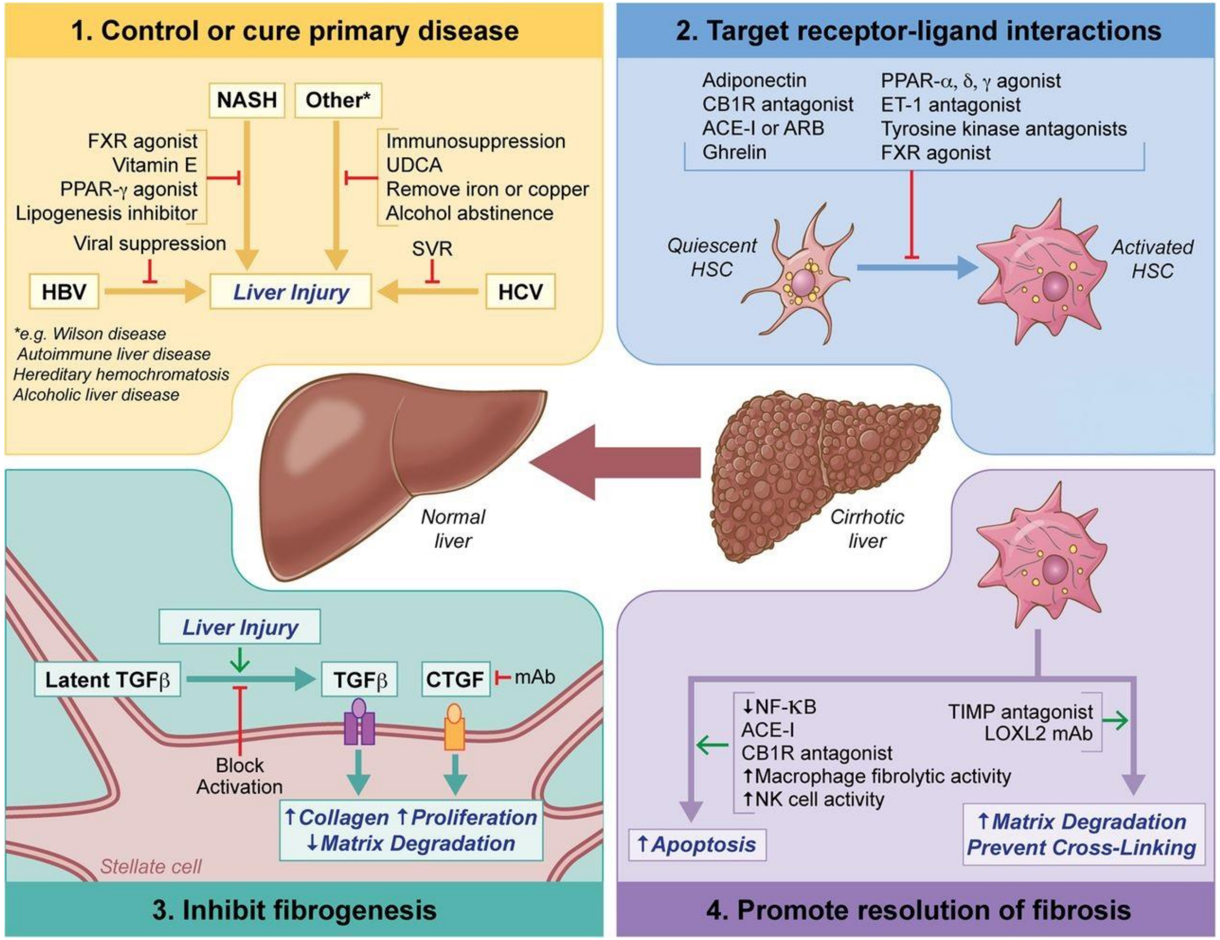


Fig.63. Possibilities of pathogenetic therapy of chronic hepatitis and cirrhosis.

FXR agonist - Farnesoid X receptor agonist, PPAR-γ agonist - Peroxisome proliferator-activated receptor- γ agonists, HBV – hepatitis B virus, HCV – hepatitis C virus, UDCA – Ursodeoxycholic acid , ET-1 antagonist – Endothelin-1 antagonist, CB1R antagonist – Cannabinoid 1 receptor antagonist , HSC – Hematopoietic stem cell, TGF – Transforming growth factor , CTGF – Connective tissue growth factor, TIMP antagonist – antagonist of tissue inhibitor of metalloproteinase , LOXL2 mAb – Lysyl Oxidase Homolog 2 Antibody, NF-KB – Nuclear factor kappa-light-chain-enhancer of activated B cells, SVR – Successful viral eradication, NK cell – Natural killer cell

COMPLICATIONS OF LIVER CIRRHOSIS

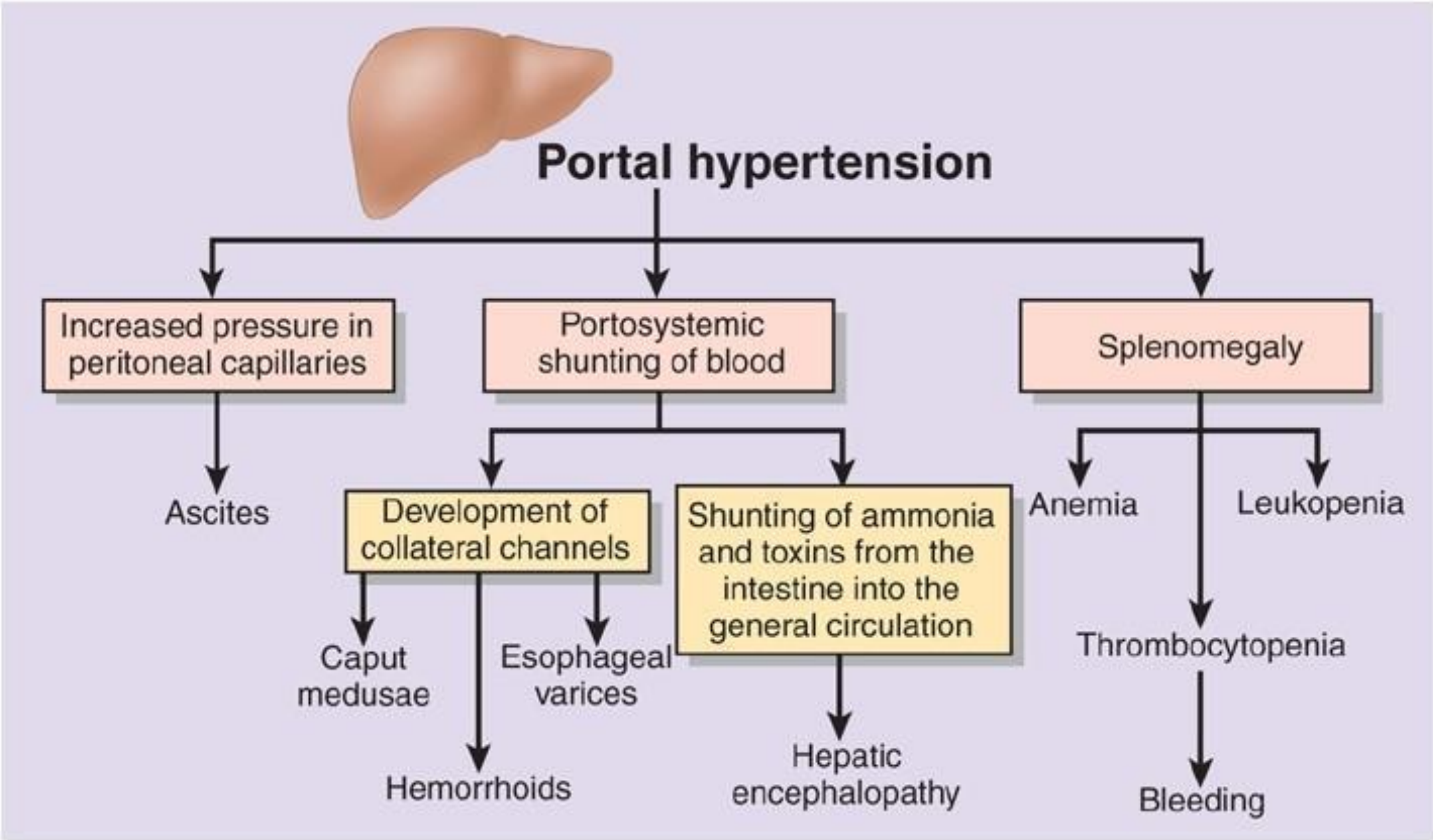


Fig. 64. Portal hypertention: pathophysiology

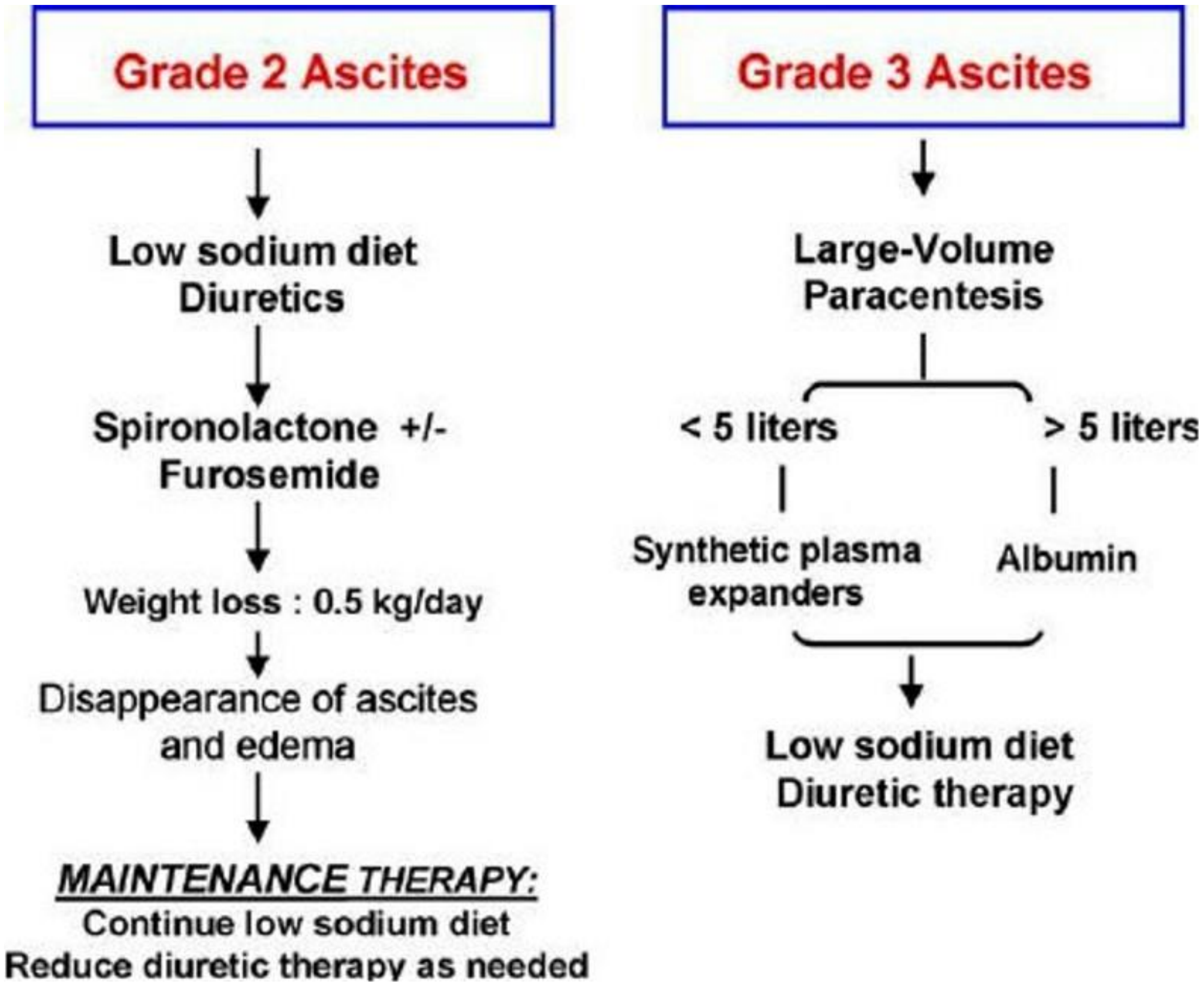


Fig. 65. Algorithm of ascites treatment

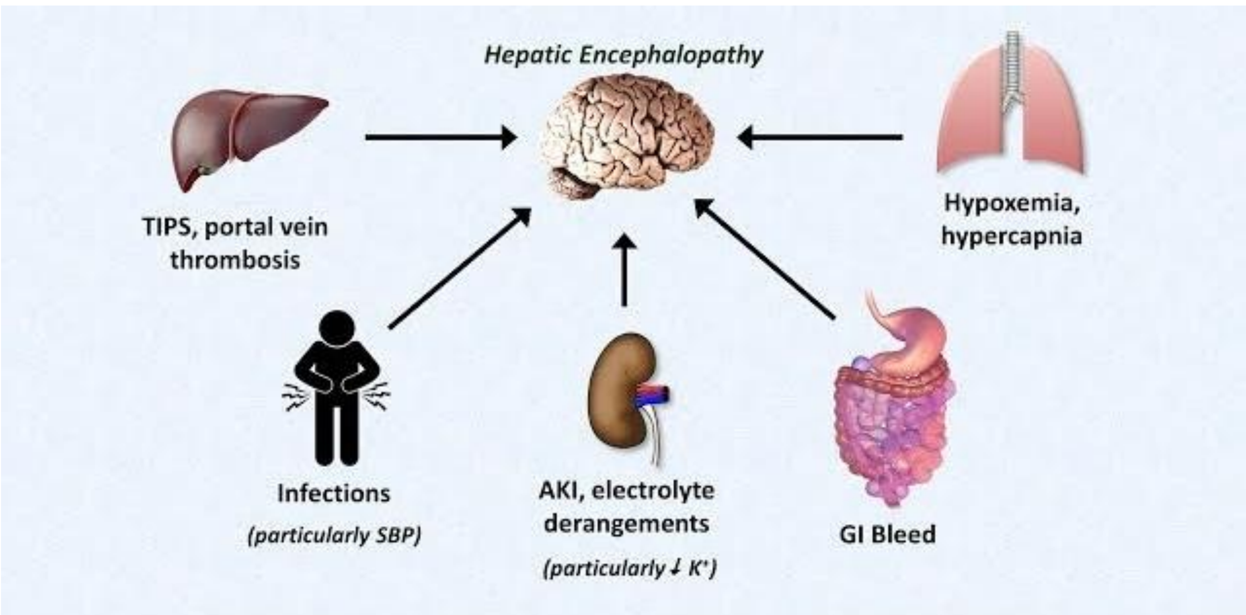


Fig. 66. Major risk factors of hepatic encephalopathy

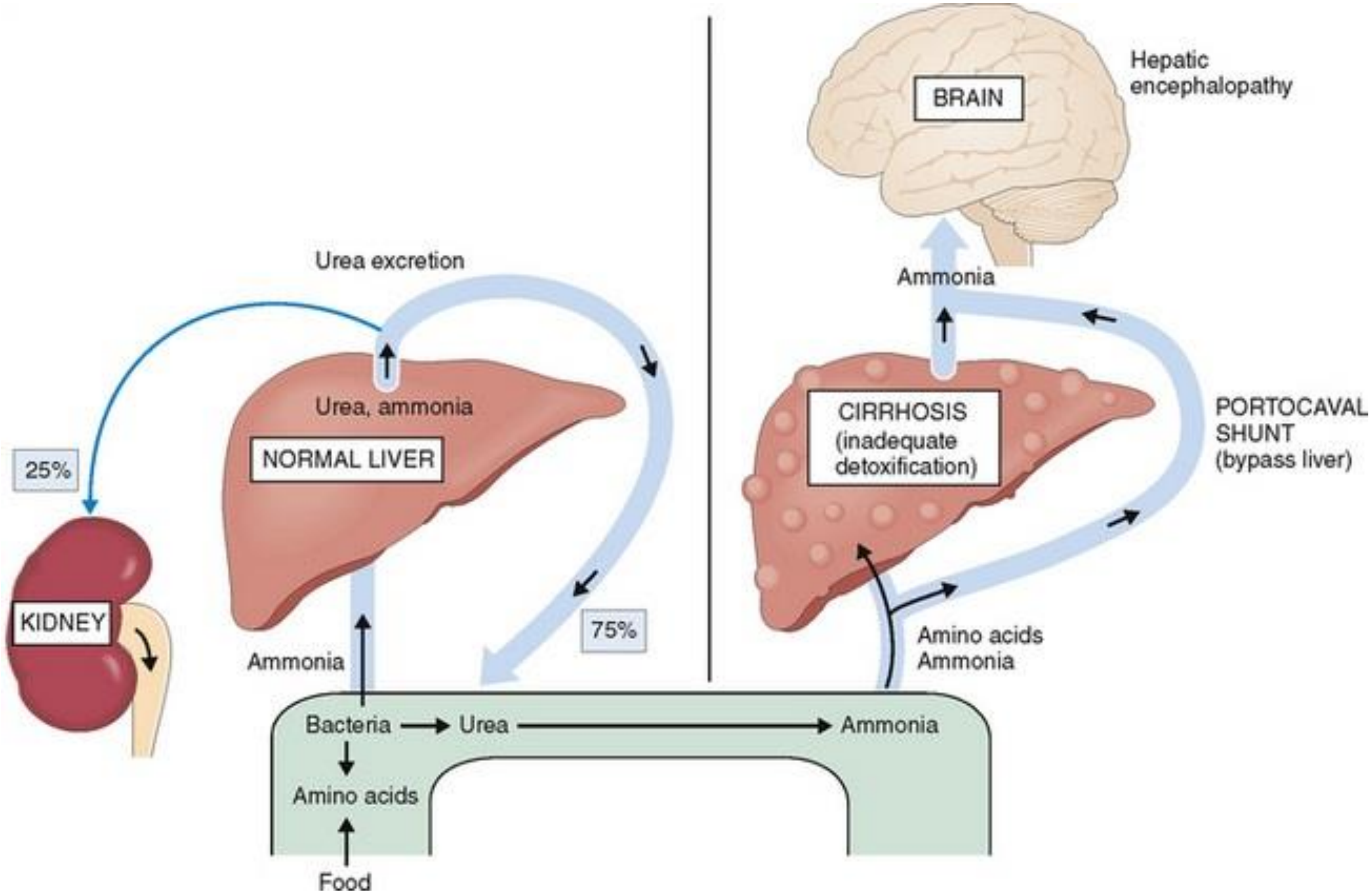


Fig. 67. Pathogenesis of hepatic encephalopathy

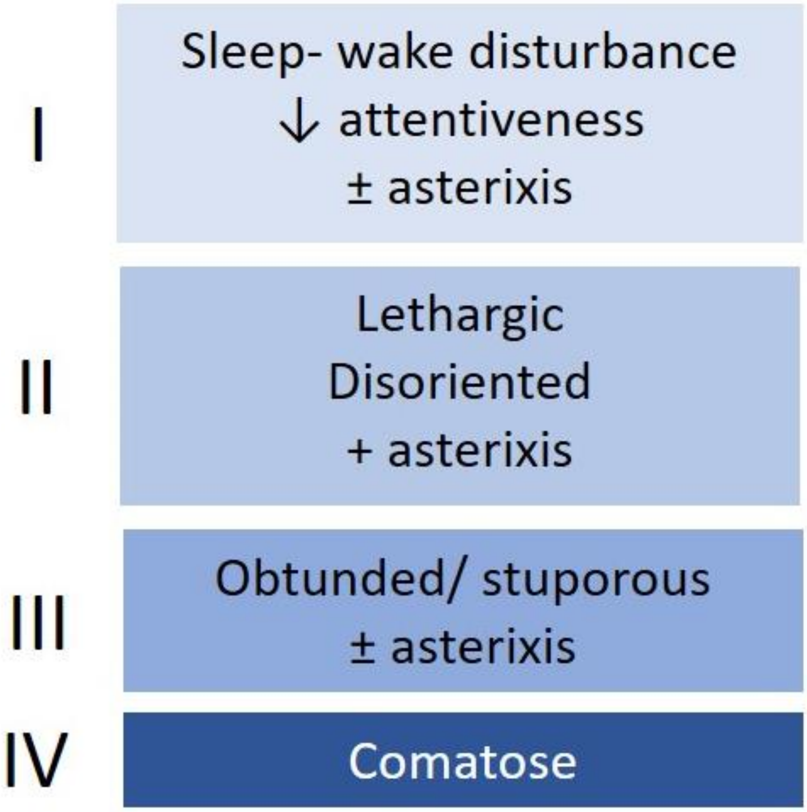


Fig. 68. Stages of hepatic encephalopathy

Table 41.

Current hepatic encephalopathy treatments

Drug	vs. lactulose	vs. placebo	Side effects
Lactulose		+	Excessive sweet taste, GI upset, hypernatremia
Lactitol	=	NE	Fewer than lactulose
Neomycin	=	=	Nephrotoxicity and ototoxicity
Metronidazole	NE	NE	GI upset, neurotoxicity
Rifaximin	NE	NE	Minimal

+: superior to control; =: equal to lactulose or placebo; NE: not evaluated;

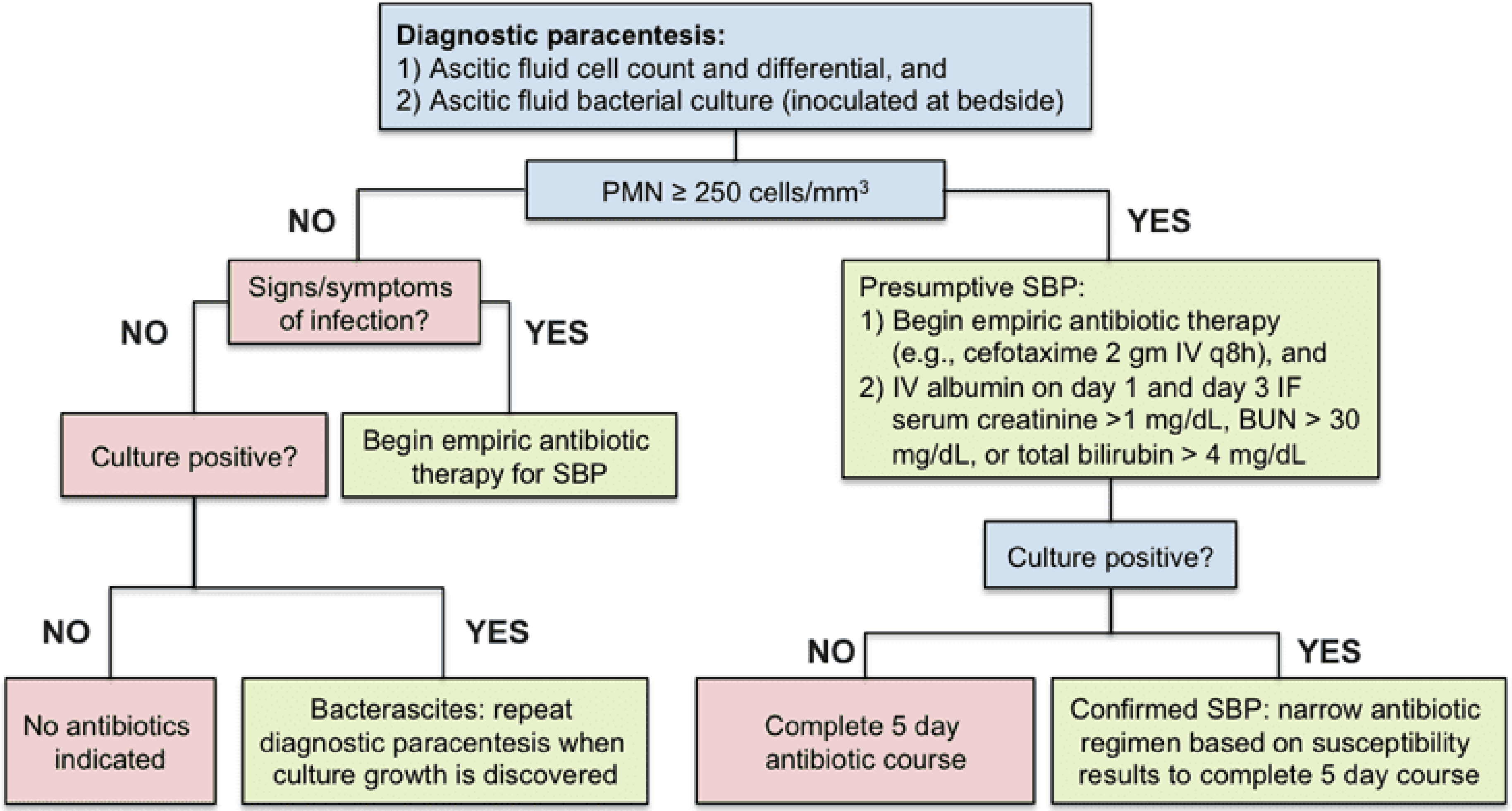
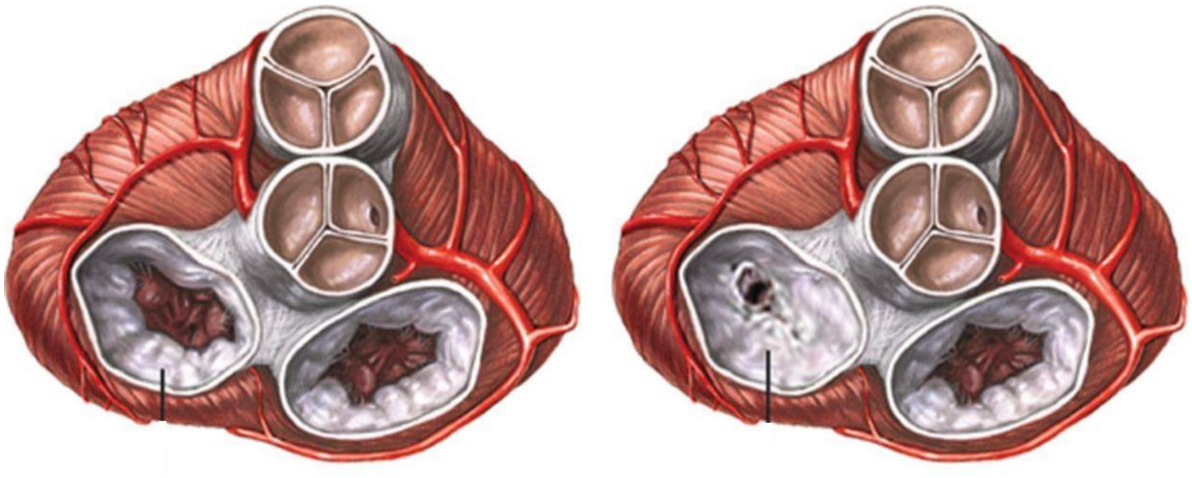


Fig. 69. Approach to the diagnosis and treatment of spontaneous bacterial peritonitis

RHEUMATOLOGY



ACUTE RHEUMATIC FEVER

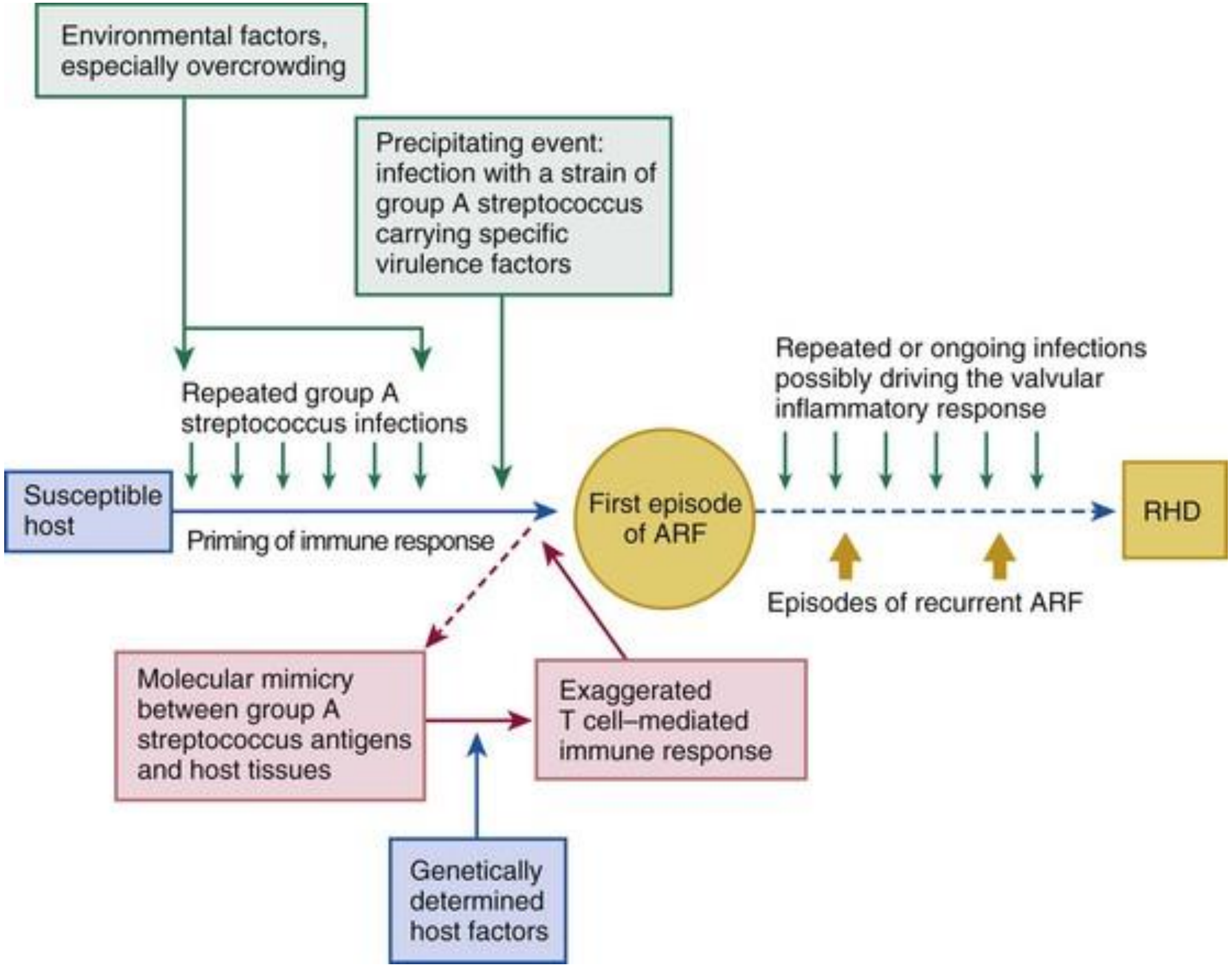


Fig. 70. Etiopathogenesis of Acute rheumatic fever (ARF). (RHD – rheumatic heart disease)

Major Manifestations
Carditis
Polyarthritis
Chorea
Erythema marginatum
Subcutaneous nodules

Minor Manifestations
Arthralgias
Fever
Increased erythrocyte sedimentation rate or C-reactive protein
Prolonged PR interval

Evidence of Preceding Streptococcal Infection
Positive throat culture for group A beta-hemolytic streptococci or positive rapid streptococcal antigen test
Elevated or rising streptococcal antibody titer, most often antistreptolysin O

Fig. 71. Revised Jones Criteria for the diagnosis of ARF,2015 (AHA)

The presence of 2 major criteria, or 1 major and 2 minor criteria, indicates a high probability of ARF, if supported by evidence of Group A streptococcal infection.

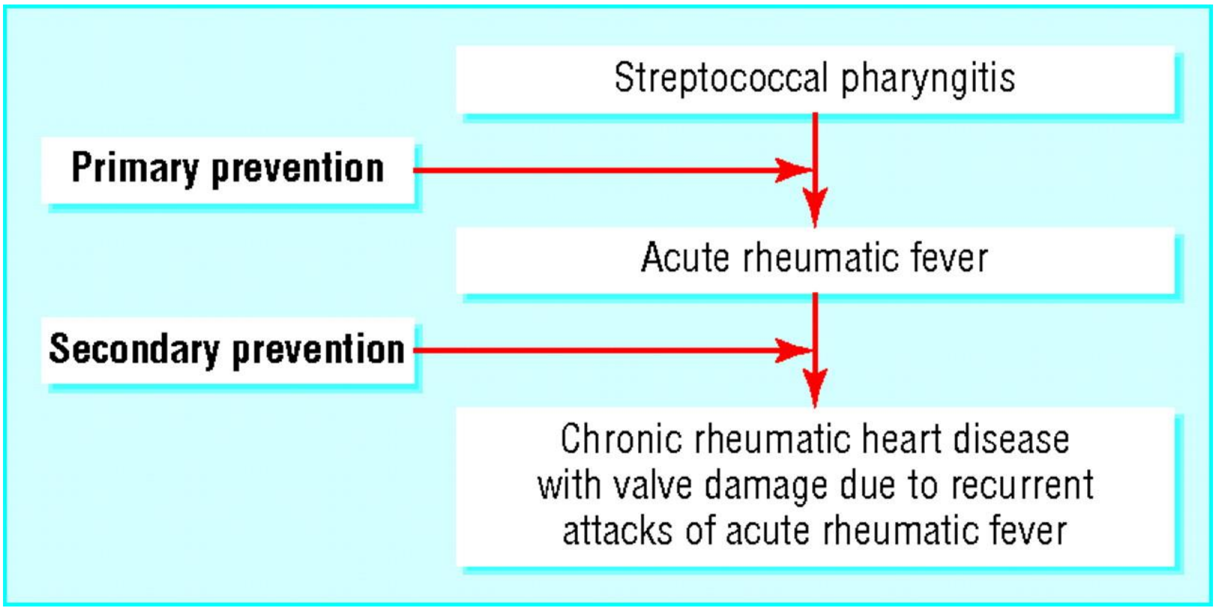


Fig. 72. Types of profilaxis of ARF

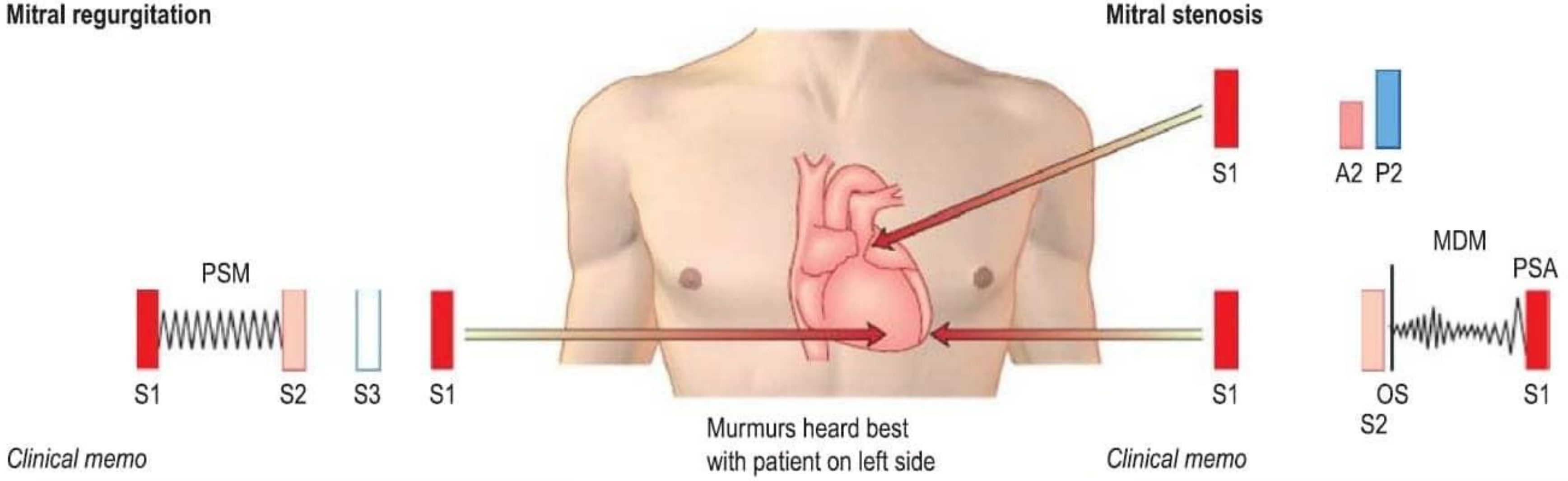
Table 42.

Secondary profilaxis of ARF (Adapted from AHA Scientific Statement: Prevention of Rheumatic Fever and Diagnosis and Treatment of Acute Streptococcal Pharyngitis, 2009)

CATEGORY OF PATIENT	DURATION OF PROPHYLAXIS
Rheumatic fever without carditis	For 5 years after the last attack or 21 years of age (whichever is longer)
Rheumatic fever with carditis but no residual valvular disease	For 10 years after the last attack, or 21 years of age (whichever is longer)
Rheumatic fever with persistent valvular disease, evident clinically or on echocardiography	For 10 years after the last attack, or 40 years of age (whichever is longer). Sometimes lifelong prophylaxis.

CHRONIC RHEUMATIC HEART DISEASE

MITRAL VALVE DISEASE



Clinical memo

Mitral regurgitation	
Pulse:	Sinus rhythm or atrial fibrillation
Apex:	Forceful, displaced, systolic thrill
Sounds:	Soft S1, S3
Murmurs:	Pansystolic

Clinical memo

Mitral stenosis	
Face:	Mitral facies
Pulse:	Atrial fibrillation
RV:	Heaving, sustained
Apex:	Localized, tapping
Sounds:	Loud S1, loud P2 (if pulmonary hypertension), opening snap
Murmurs:	Mid-diastolic rumble at apex

Fig. 73. Clinical picture of Mitral valve diseases

AORTIC VALVE DISEASE

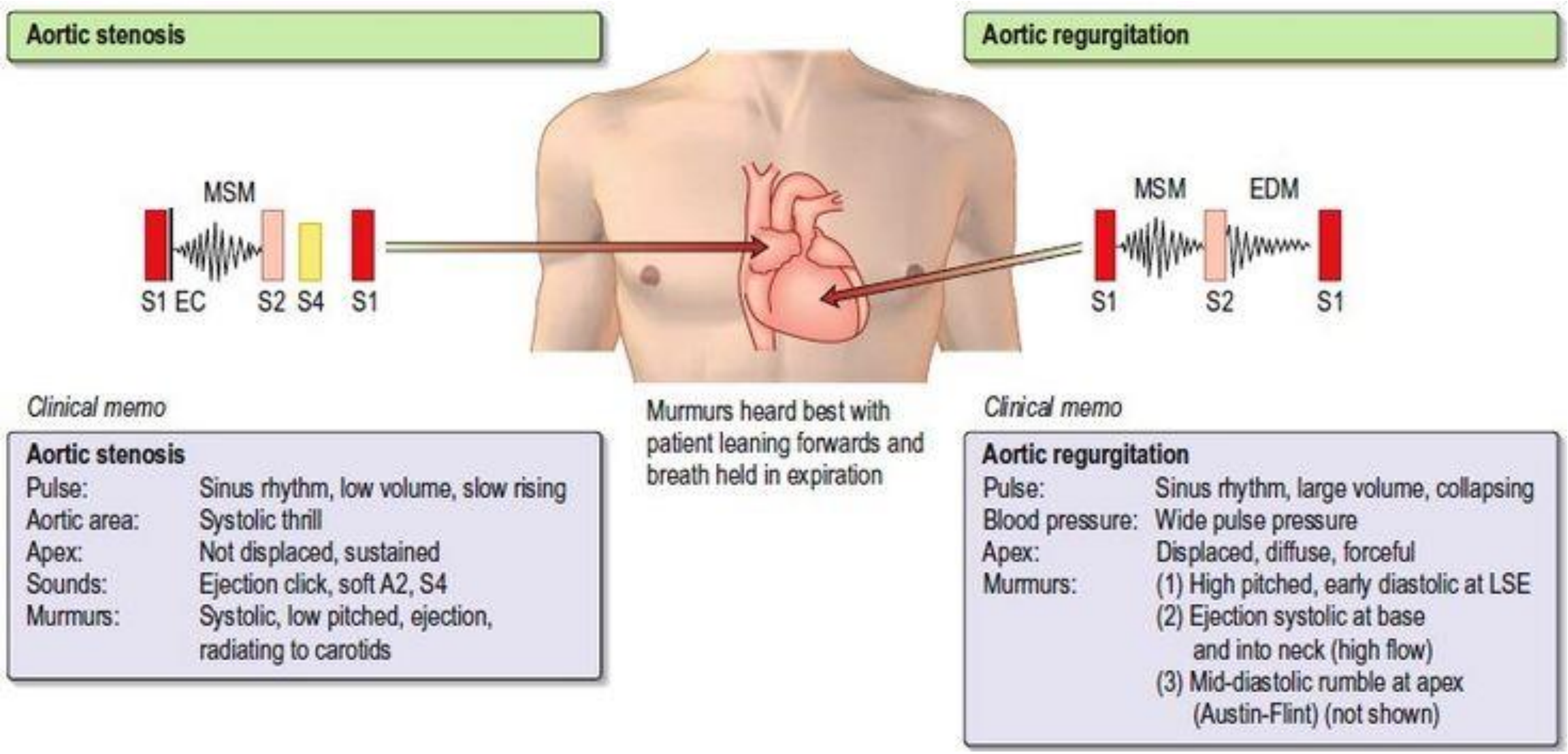


Fig. 74. Clinical picture of Aortic valve diseases

CASE 1

A 59-year-old man was hospitalized in a cardiology department from 01.09.2019 to 14.09.2019. for anterior Q+ myocardial infarction from 01.09.2019, He complained of intensive pressing chest pain with irradiation to the left shoulder, shortness of breath after a stressful situation.

From the history it is known that in the last 2 years periodically increased BP to a maximum of 160/90 mm Hg. He did not receive drug therapy regulary, occasionally he took enalapril 5 mg. During exercise, periodically there was discomfort in the heart, shortness of breath. He did not visit a doctor. He smokes more than 30 years on ½ pack of cigarettes a day. Family history: father died of myocardial infarction at the age of 54. He works as a machinist tower crane.

Upon admission, coronary angiography was performed, occlusion of the anterior interventricular artery was detected, and PCI was performed.

In the analysis: total cholesterol - 6.36 mmol/l, LDL - 3.69 mmol/l, HDL - 1.25 mmol/l, TG - 2.26 mmol/l, fasting glucose – 4.5 mmol/l.

EchoCG: left ventricular hypertrophy, enlargement of the left atrial cavity. Local hypokinesia of the lateral wall of the left ventricle. Mitral regurgitation 2 degree, tricuspid regurgitation 1 degree. Violation of diastolic function of the left ventricle (VE/VA <1,0). EF-48%.

His complaint on 30.09.2019. was shortness of breath on rapid walking. Heart pain does not bother. Constantly he takes aspirin 100 mg / day, clopidogrel 75 mg/day, atorvastatin 40 mg/day, bisoprolol 2.5 mg / day, lisinopril 5 mg 2 times a day.

On examination: satisfactory condition. BMI-37 kg / m^2. The skin is clean, normal color. In lungs - vesicular breathing, no rales. Breathing rate is 16 per minute. Heart tones are weakened, the rhythm is correct. Heart rate-70 BPM, BP-150/100 mmHg. Abdomen is soft, with palpation painless in all parts. Liver and spleen are not enlarged. There was no peripheral edema. There are no dysuric disorders.

1. Formulate and justify a preliminar diagnosis.
2. Comment on the results of the physical examination and additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 2

Patient 48 years woke up at night from pain in the epigastric region, accompanied by weakness, sweating, nausea. Previously, the pain did not bother, he considered himself healthy. An attempt to stop the pain with a solution of soda did not bring relief. After sublingual Nitroglycerin pain decreased, but not completely gone. He still had nausea, weakness, sweating. In the morning the ambulance was called.

ECG revealed a deep q wave in III and aVF leads; ST segment in the same leads is elevated, goes into the negative T wave; ST segment in leads I, aVL and V1 to V4 is depressed.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 3

The patient is 22 years old. Rhinorrhea, sore throat, and low-grade fever appeared after overcooling. After 3 days, general condition of the patient was improved, but there were attacks of dry cough, especially at night. Difficulty in breathing, profuse rhinorrhea, repeated sneezing, itching of the eyelids began to appear periodically. She noted that cough stops when she goes out. In childhood, urticaria, itching of the eyelids, and lacrimation occured eating strawberries and citrus fruits. These products have not been consumed for a long time.

Objectively: satisfactory condition, temperature is 36.7^{o}C. There is vesicular breathing, no wheezing in lungs. Chest X-ray is normal. Pathology was not detected in other organs and systems.

CBC: er.- $4.2*10^{12}$/l, Hb 132 g/l, color index 0.9; L-$5,3*10^{9}$/l, eos. - 12%, stab.- 2%, segm.- 60%, lymph. -21%; mon. -5%, ESR - 12 mm/h. Spirometry: VC = 92%, FEV_1 = 87%.

1. Primary diagnosis?
2. What medications should be prescribed:
   A) for cough and respiratory discomfort?
   B) to prevent the progression of the disease?

CASE 4

A 62-year-old patient complains of coughing, secretion of mucopurulent sputum with an admixture of blood, shortness of breath, repeated chills, general weakness. 5 days ago, the temperature increased up to 39°C. A dry cough, hoarseness appeared. Flu was diagnosed. On the 3rd day of the disease, the temperature decreased to subfebrile values, but after a day it increased again to 39.8°C. Chills appeared, the cough intensified, initially dry, then muco- purulent sputum with an admixture of blood occured, shortness of breath appeared, severe weakness.

Objectively: the condition is severe, skin is pale. Percussion of the lungs: dullness of sound in the lower parts of the right lung. In this zone, weakened vesicular breathing, wet rales. 28 breaths per min. Heart rhythm is regular, the tones are muffled. Heart rate is 110 beats per minute, BP 100/60 mm Hg. CBC: er.-$3,8*10^{12}$/l; Hb 118 g/l; color index 0.9; L-$18*10^{9}$/l; segm.-62%; lymph.-16%; mono-2%; ESR 50 mm/h.

Chest X-ray: there are multiple focal shadows, sometimes draining in the region of the lower lobe on the right lung with ring-shaped thin-walled cavities without liquid content.

1. Primary diagnosis and probable causative agent of the disease?
2. First-line drugs?

CASE 5

A patient 57 years old, complains of shortness of breath with a small load, the feeling of whistles and wheezes in the chest when breathing, coughing, with the release of mucous viscous sputum.

He smokes from the age of 20 for about 30 years. A cough has been disturbing, which the patient always associates with smoking. Respiratory discomfort and shortness of breath appeared 5-6 years ago. Then coughing attacks, wheezing began to appear. The severity of these symptoms varied depending on the weather. I felt better in the warm, dry season. The build up of shortness of breath and coughing has been especially noted over the past three years. There are 3-4 exacerbations of the disease in the year. There are 4 points on an mMRC scale.

Objectively: the chest is barrel-shaped, kyphosis of the thoracic spine. Percussion sound is a box sound. Auscultation: exhalation is elongated, expiratory wheezing are present. The determination of relative cardiac dullness is difficult, the epigastric pulsation of the right ventricle is revealed. Heart sounds are muffled, the rhythm is correct, the pulse is 92 per min, BP 160/80 mm Hg, SpO_2 88%.

Spirometry: VC = 71%; FVC = 56%; FEV_1 = 43%;

$FEF_{25\%}$ = 27%, $FEF_{50\%}$ = 23%; $FEF_{75\%}$ = 23%.

1. The diagnosis?
2. Type of ventilation disorder and severity?
3. First-line treatment?

CASE 6

Patient S., 24 years old, was admitted to the clinic with complaints of aching pain in the epigastric region, occurring 1.5-2 hours after eating, nausea and vomiting of gastric contents, occurring at the height of pain and bringing relief, loss of appetite, and constipation.

For the first time such complaints arose a year ago, but the pains were quickly relieved by taking Almagel within 2 weeks. From the anamnesis it is known that previously for many years it was observed in the clinic with a diagnosis of chronic gastritis.

Previous diseases: rubella, scarlet fever. Professional history: during the last year he has been working as an economist at the enterprise. At work often travels on business trips, irregular meals. Bad habits: often at banquets consume alcohol in large quantities. Family history: father and uncle have a duodenal ulcer, mother is healthy. Allergic history is not burdened.

On examination: general condition is of moderate severity. Body temperature 36.6 °C. Patient with reduced nutrition, asthenic constitution. The skin and visible mucous membranes are pale, clean, dry. On lungs auscultation there is vesicular breathing, no wheezing. BR - 17 per minute. Heart sounds are clear, the rhythm is correct, the heart rate is 70 beats / min. BP 120/80 mm Hg. The tongue is coated. The abdomen during palpation is soft, sensitive in the area of Shauffard. The size of the liver Kurlov: 9x8x7 cm. The spleen is not enlarged. The defecation is regular, feaces is of normal color.

Complete blood count: hemoglobin - 130 g / l, leukocytes - 6.4 $x10^9$ / l, stab. - 1%, segment. - 60%, eosinophils - 1%, lymphocytes - 30%, monocytes - 8%. ESR - 10 mm / h.

1. Highlight clinical syndromes in this patient.
2. Formulate a preliminary diagnosis.
3. What additional laboratory and instrumental studies are needed to clarify the diagnosis?
4. Prescribe treatment.

CASE 7

The patient, 38 years old, a loader, abuses alcohol, at the time of the examination complains of aching pain in the right hypochondrium, unformed stool, poor sleep. Considers himself ill for three days, after the next alcoholic excesses.

Objectively: a patient with reduced nutrition, an increase in the parotid salivary glands. On the skin of the shoulder girdle telangiectasia. Gynecomastia. The sclera are injected, subicteric. Breathing rate is 18 per minute. Percussion - sound pulmonary. Breathing is vesicular, no wheezing. The borders of the relative dullness of the heart are in the normal range. Heart sounds are clear, there is no murmurs. Heart rate is 92 per minute. The stomach is somewhat swollen. Palpation is painful in the right hypochondrium. Liver Kurlovs sizes are 18x16x15 cm, the edge of the liver is compacted, the surface is smooth. The spleen is not palpable. Blood bilirubin - 54 mmol / l. The activity of AST, ALT exceeds the norm by 4.5 times. Three times the urinary diastase activity.

1. Highlight clinical syndromes in this patient.
2. Formulate a preliminary diagnosis.
3. What additional laboratory and instrumental studies are needed to clarify the diagnosis?

CASE 8

Patient M., 20 years old, student, complains of weakness, an increase in body temperature to subfebrile values, a feeling of heaviness in the right hypochondrium, irregular menstruation, pain in small joints and their swelling. The patient considers herself to be sick for 2 months, when, on his return from Turkey (rested for 14 days), the indicated symptoms gradually began to increase. Alcohol is not consumed.

On examination: excess nutrition, moderate swelling of the joints of the hands, pink stria on the skin of the lower abdomen and buttocks, sporadic telangiectasias on the skin of the shoulder girdle, palneral erythema. The abdomen is soft on palpation, sensitive in the right hypochondrium. The size of the liver according to Kurlov: 15x12x10 cm. The lower edge of the liver is somewhat rounded, palpable 4 cm below the costal arch, smooth, soft, sensitive. The spleen is not palpable; percussive spleen sizes are 12x7 cm. Peripheral edema is not present.

In the clinical analysis of blood: ESR - 42 mm / hour, in the biochemical analysis of blood AST - 160 U / l, ALT - 240 U / l, total bilirubin - 34 mmol / l. Electrophoresis of proteins revealed severe hypergammaglobulinemia. Markers of viral hepatitis A, B, C were not detected in the blood.

Abdominal ultrasound: liver - 15x12x10 cm, diffuse heterogeneous structure, increased echogenicity. Gall bladder is of normal form. Stones are not revealed. Portal vein is 11 mm, splenic vein is 5 mm. Common bile duct is 6 mm. Spleen is 12.5x7 cm.

Pancreas is without pathology. The kidneys are usually located, without pathological changes.

1. Highlight the clinical syndromes of this patient.
2. Formulate a preliminary diagnosis.
3. What additional laboratory and instrumental studies are needed to clarify the diagnosis?
4. Prescribe treatment.

CASE 9

Patient K., 55 years old, went to the doctor with complaints of increased fatigue, weakness, abdominal discomfort, feeling of heaviness in the right hypochondrium, flatulence, paresthesias in the legs and feet.

He considers himself ill for several months, when after the next alcoholic exacerbation the indicated complaints appeared.

Over 20 years abuses alcohol. Works as a loader.

On examination: general condition is of moderate severity. The skin and visible mucous membranes are subicteric. The face is hyperemic. Parotid glands enlarged. Hyperemia of the hands in the area of the tenor and hypotenor. Moderate gynecomastia. Dupuytren's contracture is on the right. In the lungs - breathing is harsh, wheezing is not heard. The borders of the relative dullness of the heart are normal. Muffled heart sounds, no murmurs. Pulse is 88 beats / min. The rhythm of the heart is correct, blood pressure is 140/80 mm Hg.

Abdomen with palpation swollen, sensitive in the right hypochondrium. Dullness of percussion sound is revealed in flanks of abdomen. The size of the liver according to Kurlov: 14x12x10 cm.

Complete blood count: hemoglobin - 106 g / l, leukocytes - $4{,}6 \times 10^{9}$ / l, erythrocytes - $3{,}3 \times 10^{12}$ / l, platelets - $135{,}0 \times 10^{9}$/ l. ESR - 20 mm / h.

Biochemical analysis of blood: total protein - 7.8 g / l, albumin - 31 g /l, cholesterol - 3.2 mmol / l, urea - 4.1 mmol / l, creatinin - 88 mmol / l, glucose - 5.2 mmol / l, total bilirubin - 63 mmol / l, indirect bilirubin - 12 mmol /l, ALT - 74 U/l, AST - 258 U/l, Alkaline phosphotase - 403 U/l, y-glutamyl transpeptidase - 304.2 U/l, amylase - 255 U / l.

Serological tests for viral hepatitis markers are negative.

Ultrasound examination of abdominal organs: small ascites, hepatosplenomegaly. V. porta 1.2 cm. Diffuse changes of the pancreas. Endoscopy: varicose veins of the esophagus I-II degree. Hernia of the esophageal ostium of the diaphragm. Pronounced gastritis, duodenitis. ECG: sinus rhythm, heart rate - 88 per minute, normal position of axis.

1. Highlight the clinical and laboratory syndromes that are present in the patient.
2. Formulate a diagnosis.
3. What additional laboratory and instrumental methods of research must be performed to clarify the diagnosis?
4. Prescribe treatment.

CASE 10

Patient S. 45 years old turned to the therapist with complaints of dizziness, arrhithmia, flashing "flies" before his eyes. For 5 years he suffers from hypertension, occasionally with increasing BP he takes Captopril. Today after heavy physical activity there were similar complaints.

Objectively: the condition is satisfactory, the skin of the face and upper half of the trunk are hyperemic. Arrhythmic heart tones, heart rate-120 beats / min, frequent extrasystoles. BP – 180/100 mm Hg. During auscultation - vesicular breathing, no wheezing sounds, breathing rate – 18 per minute. There is no peripheral edema.

ECG: sinus rhythm interrupted by frequent atrial extrasystoles, signs of left ventricular hypertrophy, heart rate-120 beats / min.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 11

A 35-year-old man came to a general practitioner with complaints of a fever up to 37.6 ° C for five days, a cough with yellow sputum. He took antipyretic drugs without much effect. He did not take other medicines. Ten days before he was treated due to acute respiratory viral infection. Drug allergy is not noted. There are no concomitant diseases.

The patient is in satisfactory condition, respiratory rate - 19 per min. The skin is clean, normal color. During auscultation, a moderate amount of moist creaking rales is heard in the lower posterior parts of the chest on the right, in other parts of the lungs breathing isvesicular, without wheezing. Heart sounds are clear, the rhythm is correct, heart rate 82 beats per minute, BP - 120/70 mm Hg. The abdomen is soft, painless on palpation in all departments. The liver and spleen are not enlarged. There is no dysuria.

Chest X-ray on the direct and lateral projection: infiltration is determined on the right side in 9-10 segments of the lower lobe.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 12

Patient K. 48 years old went to the clinic with complaints of compressive pain behind the sternum and in the heart, radiating to the left shoulder, occurring when walking after 100 meters, sometimes at rest, relieved by taking 1-2 tablets of nitroglycerin after 2-3 minutes, shortness of breath, palpitations with little exercise. Heart pain first appeared about 5 years ago. He takes Nitroglycerin for pain relief, Cardiket 20 mg 2 times a day-for the prevention of pain in the heart, Aspirin 100 mg at night. He took statins for about two years, the last two years he does not take. Over the past six months, patient noticed the decreased exercise tolerance. The patient smokes for about 20 years, 1 pack per day. Heredity: the father died at the age of 62 years from myocardial infarction.

The general condition is satisfactory. There is normosthenic constitution.there is no peripheral edema. Breathing rate - 18 per minute, on auscultation - vesicular breathing, no rales. The borders of the heart on percussion: right- on the right border of the sternum IV intercostal space, upper-III intercostal space, left-1.0 cm inside the left midclavicular line in V intercostal space. The heart tones are muffled, the rhythm is correct, there is an accentuation of the II tone above the aorta. Heart rate-82 beats per min. BP-135/85 mm Hg. The Liver and spleen are not palpable.

Blood lipid spectrum: total cholesterol-6.8 mmol / l; triglycerides-1.7 mmol/ l; HDL cholesterol-0.9 mmol /l, LDL – 2.4 mmol / l.

ECG at rest: rhythm-sinus, heart rate-80 beats per minute. Normal position of electrical axis of heart. Single ventricular extrasystole.

Echo-CG: The thickness of the posterior wall of the left ventricle is 1.0 cm; the thickness of the interventricular septum is 1.0 cm. the chambers of the heart are not dilated. Left ventricular ejection fraction - 57%. Violations of local and global contractility of the left ventricle were not revealed.

Veloergometry test: during the first stage of the load, there was a compressive pain behind the sternum, accompanied by the appearance of ST-segment depression up to 3 mm in I, II, V2-V6, which disappeared in the recovery period.

Coronary angiography: stenosis of the upper third of the left coronary artery-80%, the middle third of the left coronary artery - 80%.

1. Formulate and justify a preliminary diagnosis.
2. List the known risk factors for CVD modifiable and non-modifiable.
3. Prescribe treatment.

CASE 13

A 25-year-old man complains of fever up to 40°C, accompanied by chills; inspiratory shortness of breath with little exercise; pain in the heart, not associated with physical activity, moderate intensity, prolonged.

From the anamnesis it is known that he uses heroin within 4 years (injections in ulnar veins). 2 weeks before hospitalization, he noted an increase in temperature to 40°C. The patient took nonsteroidal anti-inflammatory drugs as antipyretics. After 3 days, the temperature dropped to 37.2-37.4°C, his condition improved somewhat. However, after 10 days, the fever resumed, and the patient was hospitalized.

On examination: pale skin, clean. Peripheral lymph nodes are not enlarged. BMI-18 kg / m^2.Body temperature-38.9°C. In the lung vesicular breathing is carried out in all parts. BR-18 per minute. Heart tones are clear, on the basis of the xiphoid process – systolic murmur, increasing at inhalation with breath retention. The accentuation of 2^{nd} tone on a. pulmonalis is head. BP-110/60 mm Hg., Heart rate-100 beats per minute. The abdomen is soft, painless on palpation. The liver protrudes 2 cm from the edge of the rib arch, the edge of the liver is smooth. Swelling of feet is present. The Pasternatskiy symptom is negative on both sides. Urination is not impaired.

In analyses: erythrocytes-$3,3\times10^{12}$/l, hemoglobin-126 g/ l, leukocytes-$15,8\times10^{9}$/l, rod neutrophils-15%, ESR-42 mm / h, serum albumin-29 g/l, creatinine - 66 mmol/l, GFR – 92 ml/min/1,73m2, CRP – 120 mg/l (normally – up to 5 mg / l). In the general analysis of urine: specific gravity-1016, erythrocytes-0-1 in the field of view.
Inoculation of blood for sterility twice isolated S. aureus, sensitive to Oxacillin, Ceftriaxone.

Echocardiography data: the heart chambers are not enlarged. Mitral valve: the valves are sealed, The nature of the movement of the valves is multidirectional. Tricuspid valve: casps are thickened, structures are visualized on the middle and front casps of 1.86 and 1.11×0.89 cm in diameter, tricuspid regurgitation of III-IV degree.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 14

A patient 60 years old, complains of shortness of breath expiratory character at the slightest load (scale mMRC - 4 points), it coughs with viscous yellowish sputum, swelling of feet.

The cough is for more than 20 years, shortness of breath has arisen and began to progress over the past two years. Swelling of the legs and feet appeared a few days ago. Over the past year, 3 exacerbations of the disease.

Smokes for more than 40 years at 1 pack of cigarettes per day.

Objective status: a state of moderate severity. The skin is pale, dry. Swelling of the legs and feet. Above the lungs, breathing is weakened, the exhalation is lengthened, there is a lot of dry wheezing and buzzing rales during exalation. Breath rate - 20 in 1 min. Heart rate - 90 per min, BP-140/80 mm Hg. Accent ofII tone over the pulmonary artery. The abdomen is soft, painless. SpO_2-85%.

Chest X-ray : diffuse pneumosclerosis. Emphysema.

CBC: erytrocytes-$7.2*10^{12}$/l, Hb-178 g/l, L-$6.0*10^{9}$/l, eos-4%, lymph.-30%, mon.-5%, ESR -1 mm/h.

Spirography: VC=68%, FEV_1=28%.

1. Formulate and justify a preliminary diagnosis.
2. What type of spirometry abnormalities was detected during the study?
3. Prescribe treatment.

CASE 15

Patient R. 59 years old, driver. The night before, he was walking home from the parking lot when he noticed the appearance of severe pain behind the sternum with irradiation in the lower jaw and the left upper limb. At home, on the advice of his wife, he tried to stop the pain syndrome with Nitroglycerin without significant effect. The total duration of the pain syndrome is more than 20 minutes, the patient called an ambulance.

It is known from the medical history that during last 10 years the patient's blood pressure rises to a maximum of 170/90 mm Hg. He has been smoking 20 cigarettes a day for the past 20 years. During the month for the first time noted the appearance of chest pain after intense exercise and passing at rest. He was not examined and received no treatment. Heredity: mother-76 years old, suffers from hypertension, suffered a myocardial infarction, father-died at 55 years of myocardial infarction.

On examination: condition of moderate severity. The skin is pale. Height-168 cm, weight-90 kg, BMI-32 kg / m^2.. The heart tones are muffled, the accent of the second tone on the aorta is heard, the rhythm is correct. BP-160 and 90 mm Hg. Heart rate-92 beats per minute. Breathing is vesicular, there are no rales. Breathing rate-22 per minute. The abdomen is soft, painless. The size of hepatic dullness according to Kurlov is 11×9×8 cm. There are no peripheral edema.

In the analyses: total cholesterol – 6.7 mmol/l, TG – 2.8 mmol/l, HC-HDL – 0.62 mmol/l; fasting glucose – 5.2 mmol/l; creatinine – 124 mmol/l, GFR (formula CKD-EPI) = 54.5 ml/min/1.73 m2 (according to the outpatient card reduction of GFR to 55 ml/min/1.73 m2 was also recorded 4 months ago), albuminuria – 40 mg/day.

The ECG recorded sinus rhythm with heart rate-92 per minute, ST segment elevation up to 4 mm in I, AVL, V1-5, ST segment depression up to 2 mm in II, III, AVF.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 16

A 48-year-old patient complains of shortness of breath on small physical exertion, dry cough, fever up to 38.8°C, chilling. Three days ago after hypothermia, body temperature increased, a slight cough appeared, then pain in the right half of the chest with deep breathing and coughing. Subsequently, the temperature increased to febrile values, chest pain decreased, then disappeared, but shortness of breath appeared and began to increase.

Objectively: the condition is satisfactory. Over the lungs during percussion, dull percussion sound to the right side below the level of the 3rd rib is determined. In this zone, voice trembling is not carried out, breathing is sharply weaked, in the lower parts of the right side is not determined. Heart: apex of the heart is in the 5th intercostal space, shifted to the left of the mid-clavicular line, the rhythm is regular, the tones are cleared. Pulse 92/min, BP - 120/70 mmHg. Liver and spleen are not enlarged.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 17

A patient 57 years old, complains of shortness of breath with a small load, the feeling of whistles and wheezes in the chest when breathing, coughing, with the release of mucous viscous sputum.

He smokes from the age of 20 for about 30 years. A cough has been disturbing, which the patient always associates with smoking. Respiratory discomfort and shortness of breath appeared 5-6 years ago. Then coughing attacks, wheezing began to appear. The severity of these symptoms varied depending on the weather. I felt better in the warm, dry season. The build up of shortness of breath and coughing has been especially noted over the past three years. There are 3-4 exacerbations of the disease in the year. There are 4 points on an mMRC scale.

Objectively: the chest is barrel-shaped, kyphosis of the thoracic spine. Percussion sound is a box sound. Auscultation: exhalation is elongated, expiratory wheezing are present. The determination of relative cardiac dullness is difficult, the epigastric pulsation of the right ventricle is revealed. Heart sounds are muffled, the rhythm is correct, the pulse is 92 per min, BP 160/80 mm Hg, SpO_2 88%.

Spirometry: VC = 71%; FVC = 56%; FEV_1 = 43%;
$FEF_{25\%}$ = 27%, $FEF_{50\%}$ = 23%; $FEF_{75\%}$ = 23%.

1. Formulate and justify a preliminary diagnosis.
2. Type of ventilation disorder and severity?
3. Prescribe treatment.

CASE 18

The patient is 19 years old, delivered by the ambulance. The patient for the first time in his life had an asthma attack. According to the patient, until now she considered herself a healthy person. Due to the fact that in childhood, the patient had allergic reactions in the form of a skin rash after contact with animals - a cat, dog, horse and a reaction in the form of vasomotor rhinitis to dust, the patient was observed by an allergologist. 3 months ago, the patient got a new job as an administrator in a beauty salon. A month later, the patient became suffer from dry cough, which became constant and gradually intensified. Day before an attack of suffocation, the patient was at a party and had a contact with a dog. In the evening after coming back, the cough intensified, shortness of breath, shortness of breath appeared. During the night, these symptoms were intensified, there was a feeling of lack of air, a feeling of incomplete exhalation due to suffocation. The patient called an ambulance and was hospitalized.

Family history: the father of the patient has bronchial asthma, the mother is healthy.

On examination, the state of moderate severity. Body temperature 36.7 degrees. The skin is clear, moist, slight acrocyanosis. 26 breaths per min. Comparative percussion – band box sound. The mobility of the lower pulmonary margin along the mid axillary line is ± 2.5 cm, along the midclavicular and scapular lines ± 2 cm. Auscultation of the lungs: exhalation is almost 2 times longer than the inspiration. Breathing is carried out in all parts, a large number of dry wheezing is heard. Heart rate - 90 beats/min, BP - 120/80 mm Hg. The abdomen is soft, painless.

CBC: Hb - 130 g/l, L-7,8*10^9/l, eos.-10%, segm-65%, lymph.-20%, mon.-2%, ESR - 5 mm/h.

Chest X-ray: pulmonary fields are transparent, high airiness. There are no focal and infiltrative shadows in lungs. Sinuses are free.

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

CASE 19

Patient D., 67 years old, complains of constant pain and heaviness in the epigastric region, bad taste in the mouth, loss of appetite, belching with air, nausea, unstable defecation (up to three times a day), abdominal bloating, malaise, weakness, weight loss of 3 kg over the past two months.

He is sick for 10 years, when for the first time there was a feeling of heaviness in the upper half of the abdomen, belching with air, alternation of constipation and diarrhea. Discomfort in the upper half of the abdomen, an unstable stool occurred periodically, were more often associated with a violation of the diet (fatty, fried food, alcohol) and passed after some time independently. He didn't apply to doctors, he wasn't treated. In the past five years, dyspeptic disorders began to bother more often. In the clinic at the place of residence in the study of gastric contents, according to the patient, there were signs of a decrease in the secretory function of the stomach. Fibrogastroscopy was not performed. He was treated with no-spa, enzyme preparations (pancreatin), with a short-term positive effect. The real deterioration of well-being notes in the last two months, when they began to increase dispeptic phenomena, lost 3 kg.

His condition is satisfactory. The skin and visible mucous membranes are pale, atrophy and smoothness of the papillae of the tongue, imprints of the teeth on the lateral surface of the tongue, angular stomatitis. In the lungs vesicular breathing, no wheezing. Heart sounds are clear, rhythmic. BP - 130/80 mm Hg. Pulse -68 per minute, rhythmic. The abdomen is moderately swollen, with palpation diffuse pain in the epigastric region, the liver and spleen are not enlarged.

In general the blood test: red blood cells - $5.3x10^{12}/l$, hemoglobin -110 g / l, leukocytes - $6.2\ x10^{9}/l$, color index - 1.2, ESR - 12 mm / h. Gastroduodenoscopy: the esophagus is not changed, there is a small amount of fluid in the stomach, the mucous folds are smoothed, the gastric mucosa is pale, atrophic. A mucous membrane biopsy was taken from the stomach body (atrophy of the glandular epithelium, signs of intestinal metaplasia, minimal signs of inflammation, Helicobacter pylori and antibodies to Helicobacter pylori were not found).

1. Formulate and justify a preliminary diagnosis.
2. Make and justify a plan for additional laboratory and instrumental studies.
3. Prescribe treatment.

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