

SIGNS AND SYMPTOMS OF CARDIOVASCULAR SYSTEM DISEASES (syndrome of heart failure)

LECTURE IN INTERNAL MEDICINE PROPAEDEUTICS

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Plan of the lecture



Heart failure

- Definition
- Pathophysiology
- Systolic dysfunction
- Diastolic dysfunction
- Left ventricle failure
- Right ventricle failure
- Cardiovascular response
- Causes
- Risk factors
- Classification
- Clinical picture
- Diagnosis
- Complications

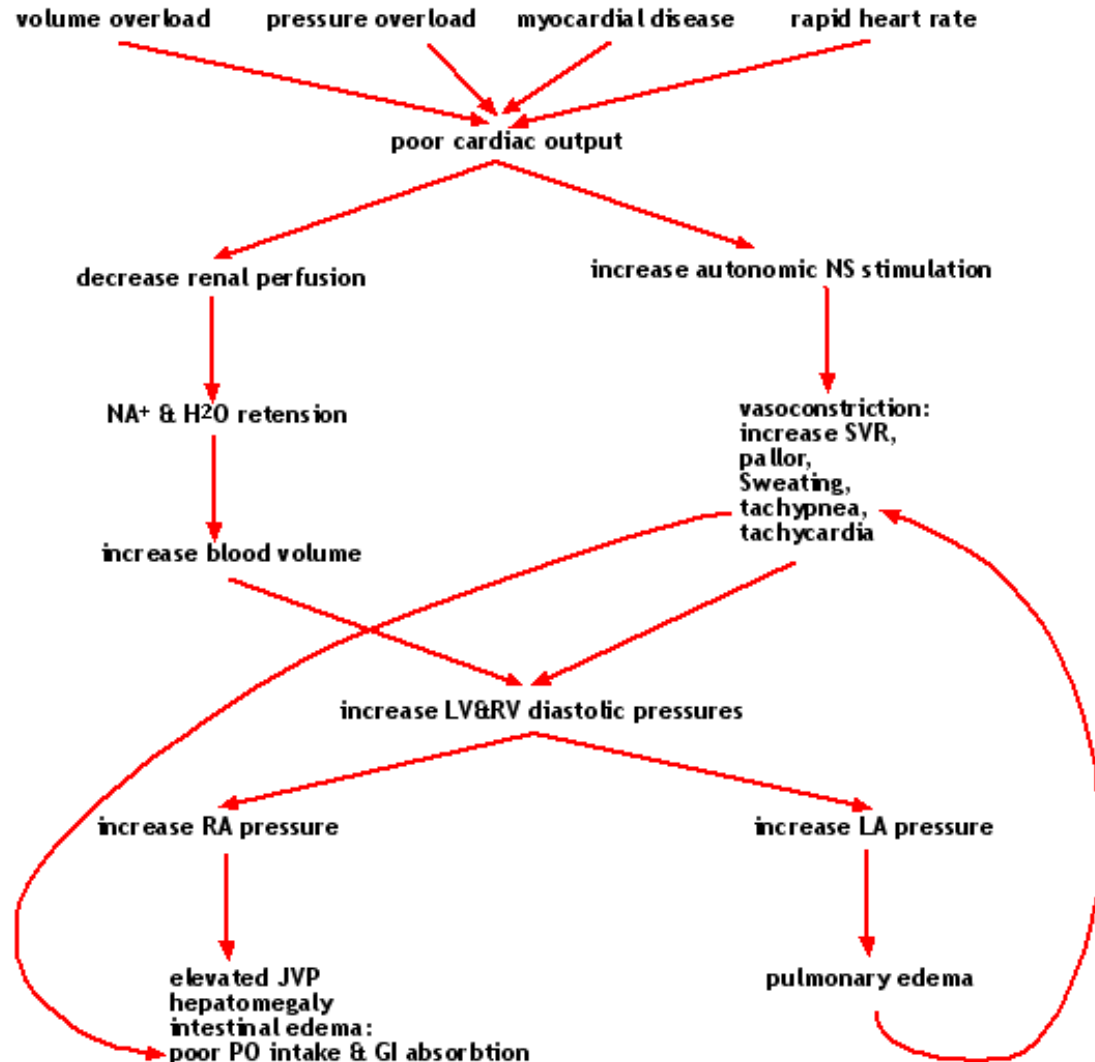
Definition

- Heart failure (HF) can be defined as an abnormality of cardiac structure or function leading to failure of the heart to deliver oxygen at a rate commensurate with the requirements of the metabolizing tissues, despite normal filling pressures (or only at the expense of increased filling pressures)
- HF is defined, clinically, as a syndrome in which patients have typical symptoms (e.g. breathlessness, ankle swelling, and fatigue) and signs (e.g. elevated jugular venous pressure, pulmonary crackles, and displaced apex beat) resulting from an abnormality of cardiac structure or function

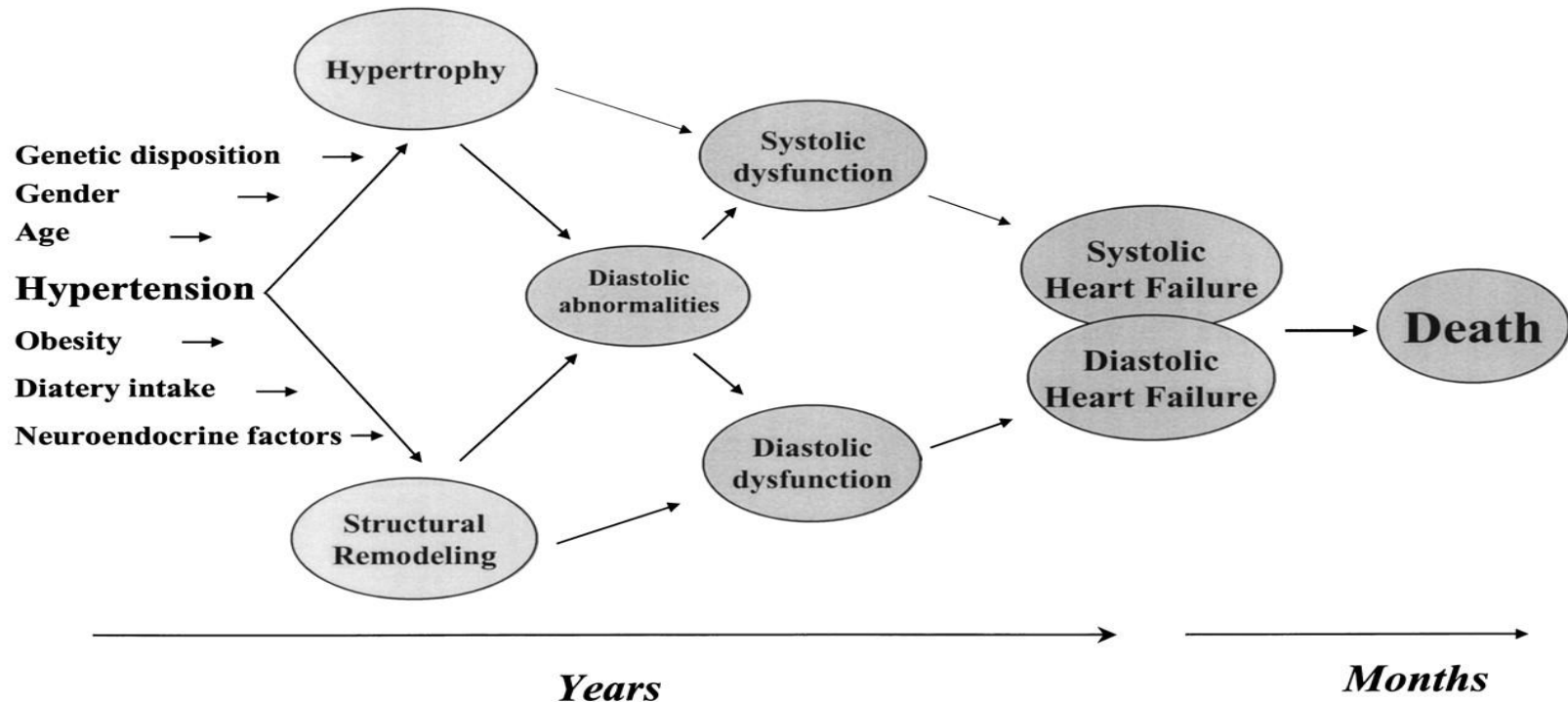
Pathophysiology 1

- In HF, the heart may not provide tissues with adequate blood for metabolic needs, and cardiac-related elevation of pulmonary or systemic venous pressures may result in organ congestion
- This condition can result from abnormalities of systolic or diastolic function or, commonly, both
- Although a primary abnormality can be a change in myocyte function, there are also changes in collagen turnover of the extracellular matrix

Pathophysiology 2



Pathophysiology 3



Progression from hypertrophy to diastolic HF. In the presence of congestive symptoms the time course of HF may become progressive and may end with sudden cardiac death or intractable end-stage failure.

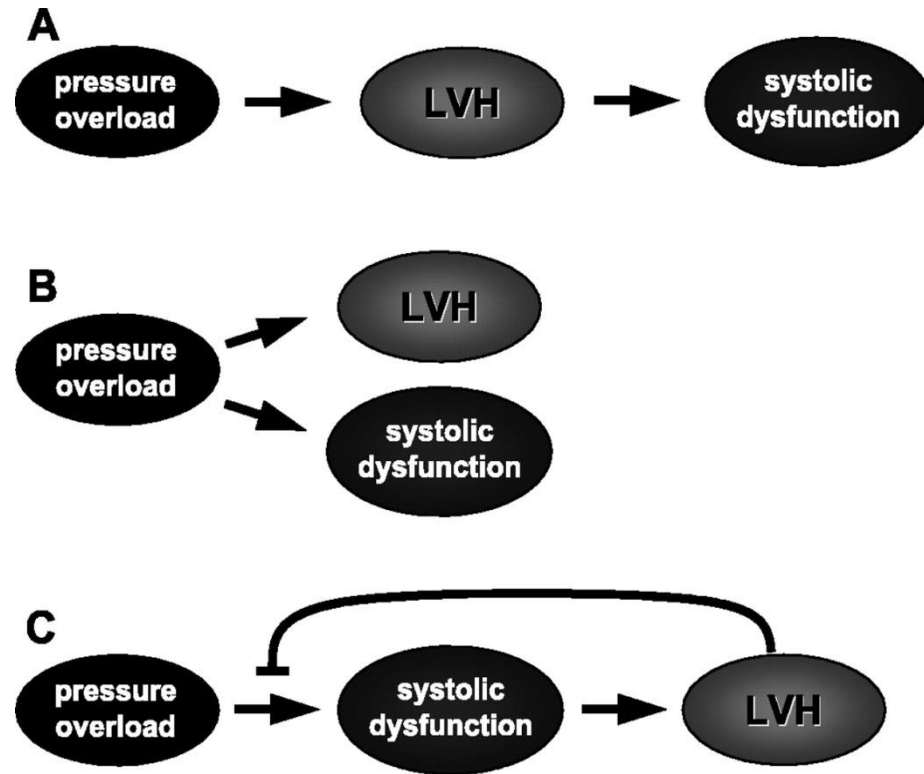
Systolic dysfunction 1

- In systolic dysfunction with reduced ejection fraction (EF)), the ventricle contracts poorly and empties inadequately, leading initially to increased diastolic volume and pressure and decreased EF
- Many defects in energy utilization, energy supply, electrophysiologic functions, and contractile element interaction occur, with abnormalities in intracellular Ca modulation and cAMP production

Systolic dysfunction 2

- Predominant systolic dysfunction is common in HF due to MI, myocarditis, and dilated cardiomyopathy
- Systolic dysfunction may affect primarily the left ventricle (LV) or the right ventricle (RV)
- LV failure often leads to RV failure

Systolic dysfunction 3



A, B: Left ventricular hypertrophy (LVH) and systolic dysfunction
C: LVH is a compensatory response to stress-mediated systolic dysfunction.

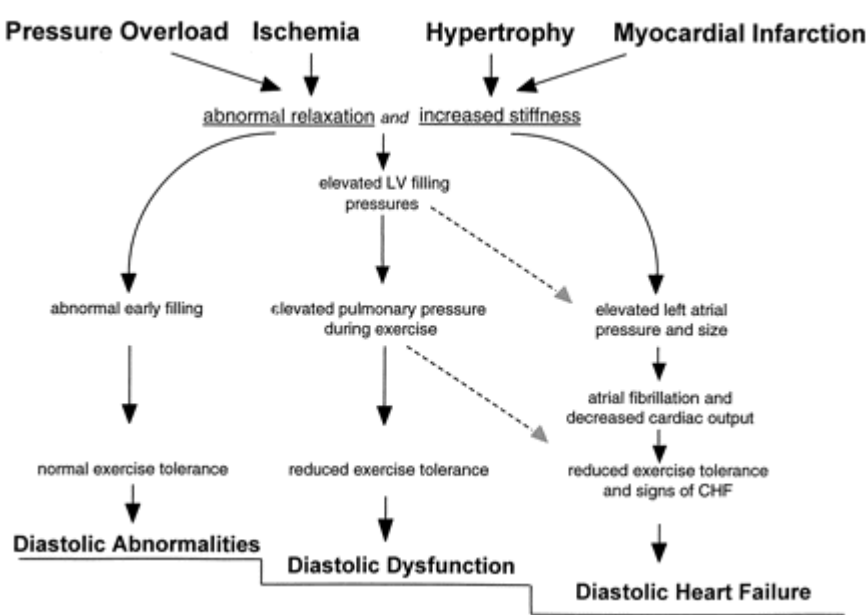
Diastolic dysfunction 1

- In diastolic dysfunction (heart failure (HF) with preserved ejection fraction (EF), ventricular filling is impaired, resulting in reduced ventricular end-diastolic volume, increased end-diastolic pressure, or both
- Contractility and hence EF remain normal
- EF may even increase as the poorly filled LV empties more completely to maintain cardiac output (CO)

Diastolic dysfunction 2

- Markedly reduced LV filling can cause low CO and systemic symptoms Elevated left atrial pressures can cause pulmonary hypertension and pulmonary congestion
- Diastolic dysfunction usually results from impaired ventricular relaxation, increased ventricular stiffness due to valvular disease, constrictive pericarditis, acute myocardial ischemia, hypertrophic cardiomyopathy, disorders with ventricular hypertrophy, and amyloid infiltration of the myocardium

Diastolic dysfunction 3



Abnormal relaxation and increased stiffness are associated with diastolic filling abnormalities and normal exercise tolerance in the early phase of diastolic dysfunction

Left ventricle failure 1

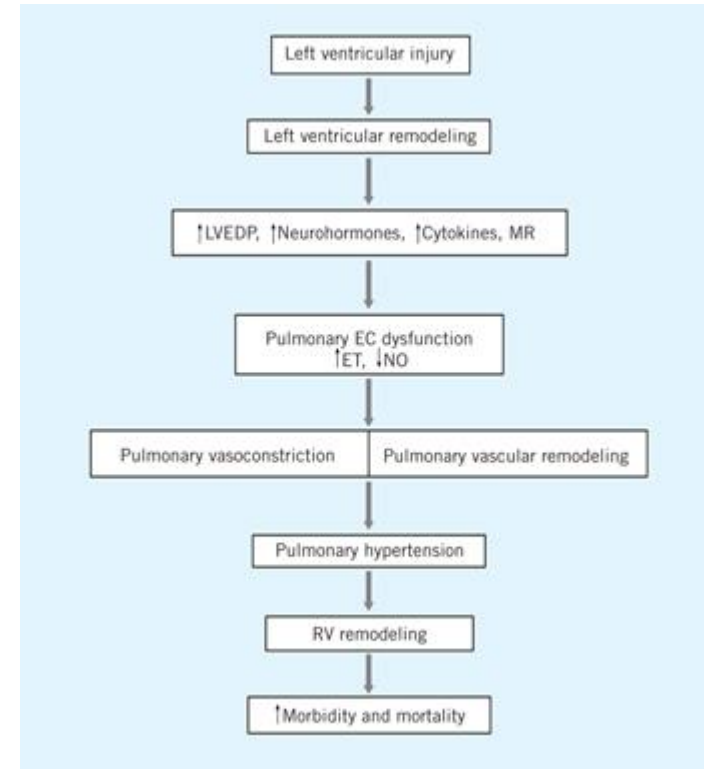
- Cardiac output (CO) decreases and pulmonary venous pressure increases
- When pulmonary capillary pressure exceeds the oncotic pressure of plasma proteins (about 24 mm Hg), fluid extravasates from the capillaries into the interstitial space and alveoli, reducing pulmonary compliance and increasing the work of breathing
- Lymphatic drainage increases but cannot compensate for the increase in pulmonary fluid

Left ventricle failure 2

- Marked fluid accumulation in alveoli (pulmonary edema) significantly alters ventilation/perfusion relationships
- In severe or chronic LV failure, pleural effusions characteristically develop in the right hemithorax and later bilaterally, further aggravating dyspnea

Left ventricle failure 3

Left ventricular injury leading to structural remodeling and dysfunction is the seminal event in the progression of heart failure



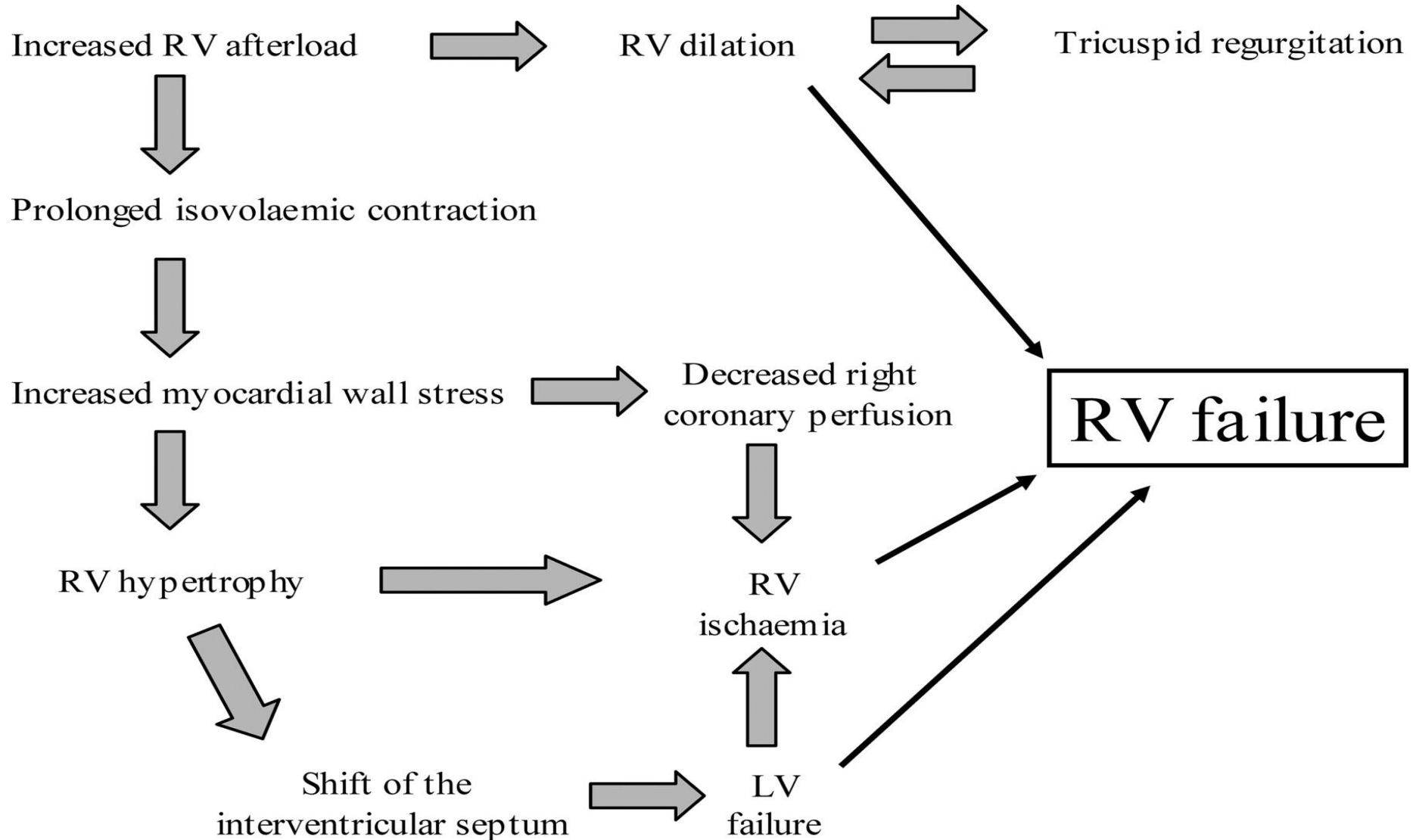
Right ventricle failure 1

- Systemic venous pressure increases, causing fluid extravasation and consequent edema, primarily in dependent tissues (feet and ankles of ambulatory patients) and abdominal viscera
- The liver is most severely affected, but the stomach and intestine also become congested
- Fluid accumulation in the peritoneal cavity (ascites) can occur

Right ventricle failure 2

- Right ventricle failure causes moderate hepatic dysfunction, with modest increases in conjugated and unconjugated bilirubin, hepatic enzymes, etc.
- The impaired liver breaks down less aldosterone, further contributing to fluid accumulation
- Chronic venous congestion in the viscera can cause anorexia, malabsorption of nutrients and drugs, protein-losing enteropathy (characterized by diarrhea and marked hypoalbuminemia), chronic GI blood loss, and rarely ischemic bowel infarction

Right ventricle failure 3



Cardiovascular response 1

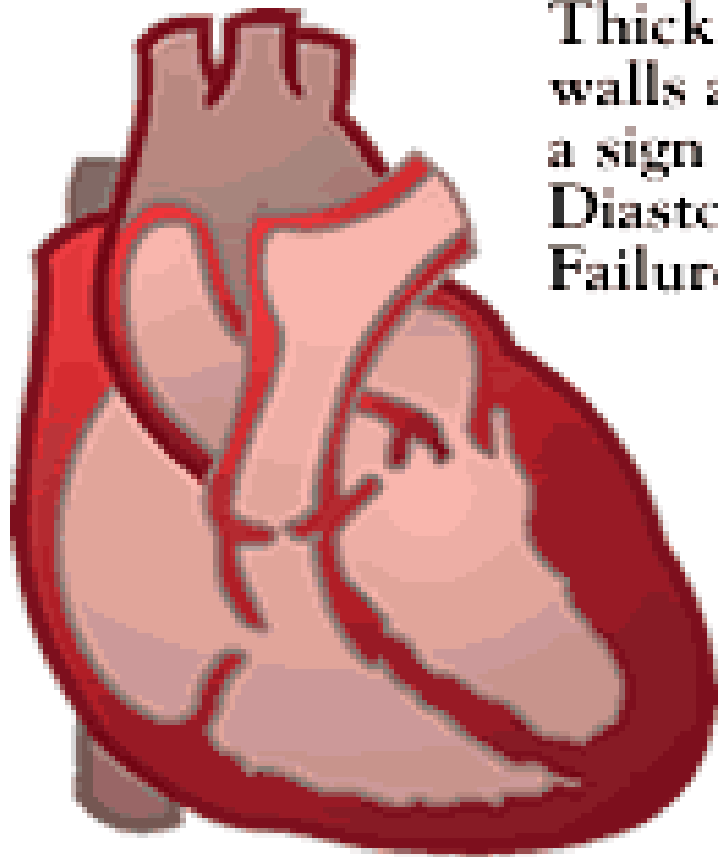
- If ventricular function is impaired, a higher preload is required to maintain cardiac output (CO)
- The ventricles are remodeled over time: the left ventricle (LV) becomes less ovoid and more spherical, dilates, and hypertrophies; the right ventricle (RV) dilates and may hypertrophy.
- Initially compensatory, these changes eventually increase diastolic stiffness and wall tension (i.e., diastolic dysfunction develops), compromising cardiac performance, especially during physical stress

Cardiovascular response 2

- Increased wall stress raises O_2 demand and accelerates apoptosis (programmed cell death) of myocardial cells
- Dilation of the ventricles can also cause mitral or tricuspid valve regurgitation with further increases in end-diastolic volumes

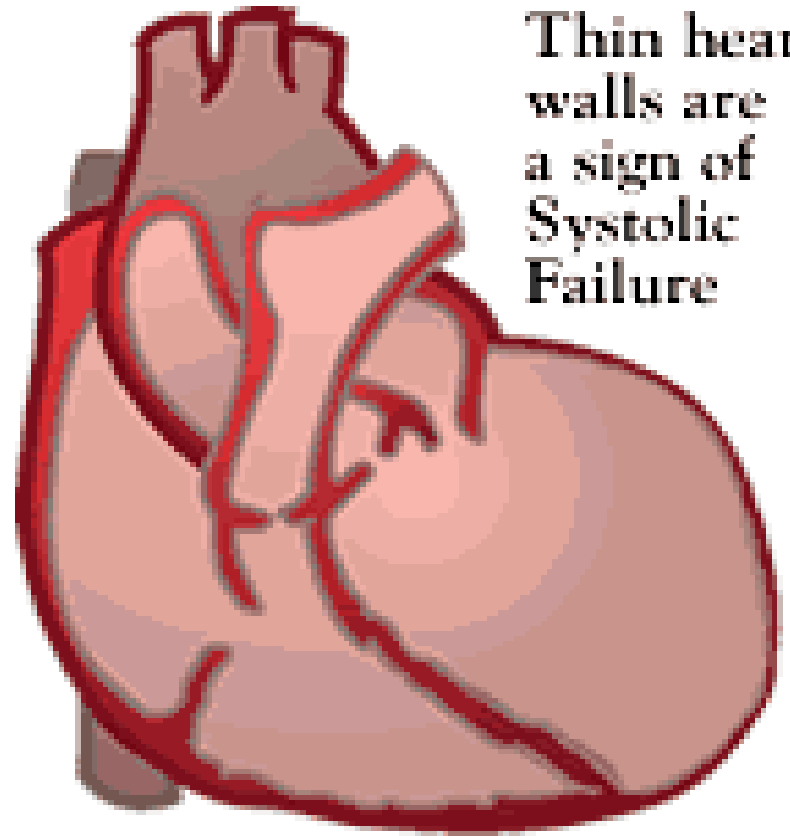
Cardiovascular response 3

Diastolic Failure



Thick heart
walls are
a sign of
Diastolic
Failure

Systolic Failure



Thin heart
walls are
a sign of
Systolic
Failure

Cardiovascular response 4

- With reduced cardiac output (CO), O₂ delivery to the tissues is maintained by increasing O₂ extraction and sometimes shifting the oxyhemoglobin dissociation curve to the right to favor O₂ release
- Reduced CO with lower systemic blood pressure (BP) activates arterial baroreflexes, increasing sympathetic tone and decreasing parasympathetic tone

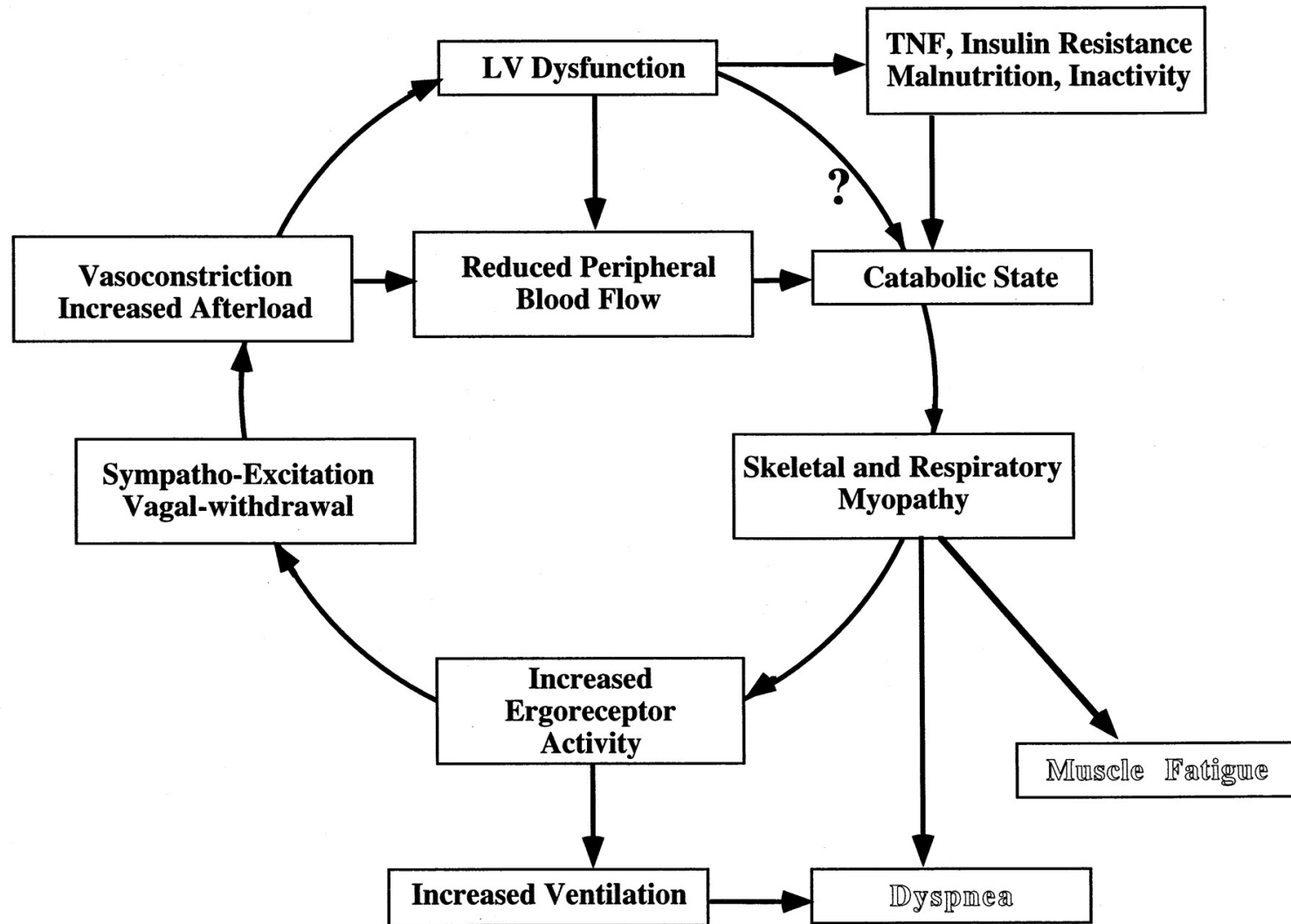
Cardiovascular response 5

- Heart rate and myocardial contractility increase, arterioles in selected vascular beds constrict, venoconstriction occurs, and Na and water are retained
- These changes compensate for reduced ventricular performance and help maintain hemodynamic homeostasis in the early stages of heart failure (HF)

Cardiovascular response 6

- These compensatory changes increase cardiac work, preload, and afterload; reduce coronary and renal perfusion; cause fluid accumulation resulting in congestion; increase K excretion; and may cause myocyte necrosis and arrhythmias

Cardiovascular response 7



Cardiovascular response 8

- As cardiac function deteriorates, renal blood flow and Glomerular Filtration Rate (GFR) decrease, and blood flow within the kidneys is redistributed
- The filtration fraction and filtered Na decrease, but tubular resorption increases, leading to Na and water retention
- Blood flow is further redistributed away from the kidneys during exercise, but renal blood flow improves during rest, possibly contributing to nocturia

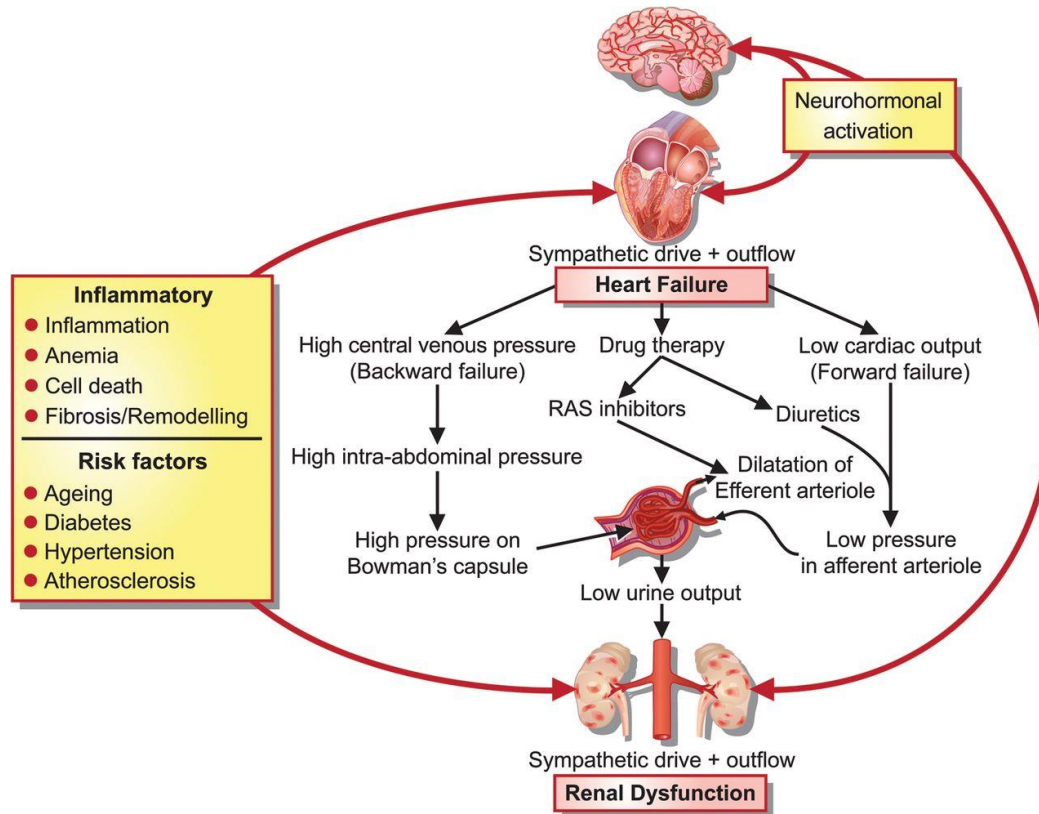
Cardiovascular response 9

- Decreased perfusion of the kidneys (and possibly decreased arterial systolic stretch secondary to declining ventricular function) activates the renin-angiotensin-aldosterone system, increasing Na and water retention and renal and peripheral vascular tone; these effects are amplified by the intense sympathetic activation accompanying heart failure (HF)

Cardiovascular response 10

- These processes causes a cascade of potentially deleterious long-term effects with myocardial and vascular collagen deposition and fibrosis, vascular and myocardial hypertrophy, thus contributing to the remodeling of the heart and peripheral vasculature, potentially worsening HF

Cardiovascular response 11



Potential pathogenetic pathways linking heart failure with renal dysfunction. RAS, renin–angiotensin system.

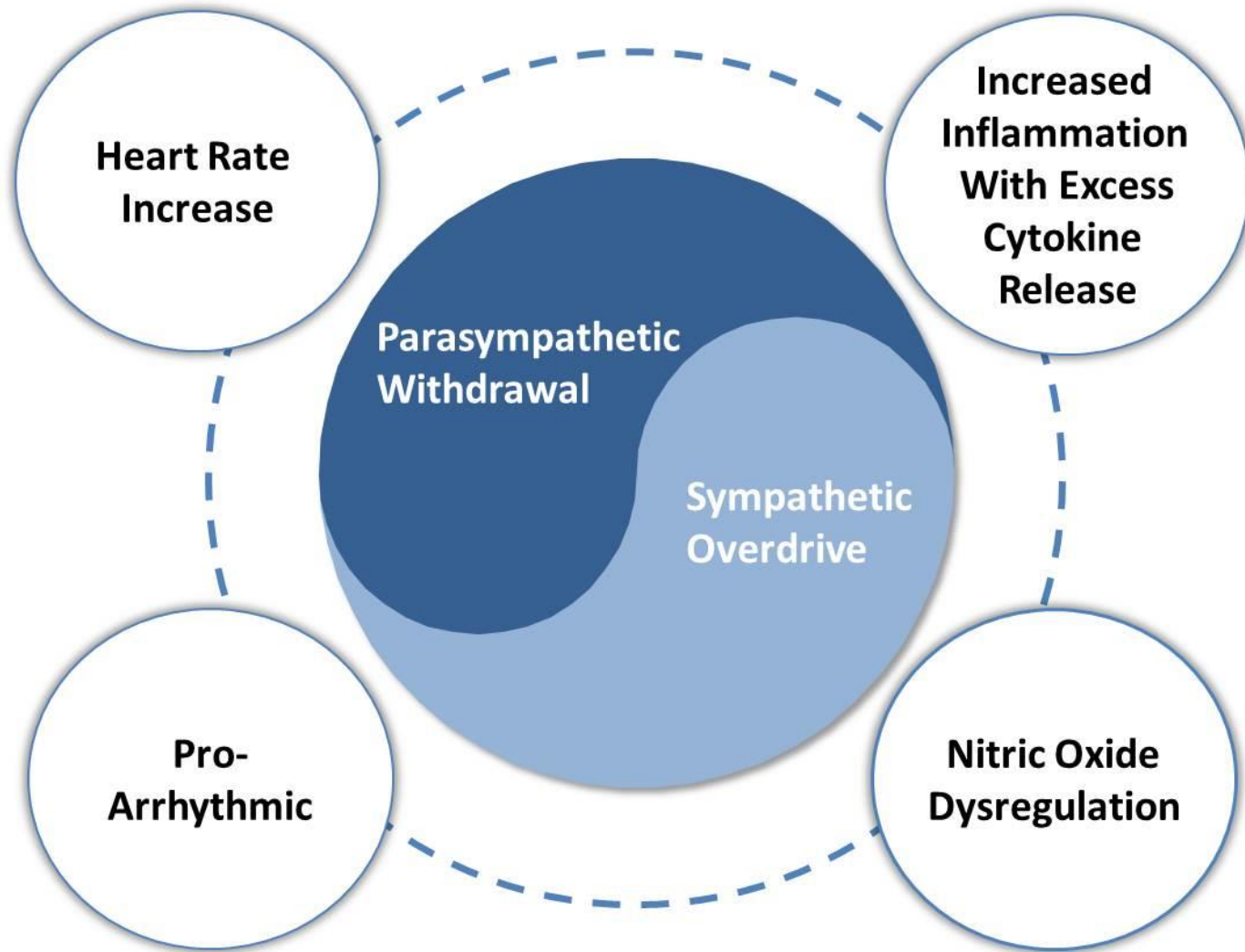
Cardiovascular response 12

- Chronic activation of neurohumoral responses is detrimental to the normal balance between myocardial-stimulating and vasoactive hormones:
 - β_1 receptors are downregulated, probably in response to intense sympathetic activation
 - Plasma norepinephrine levels are increased, largely reflecting sympathetic nerve stimulation as plasma epinephrine levels are not increased
 - Vasopressin is released in response to a fall in blood pressure (BP)

Cardiovascular response 13

- Atrial natriuretic peptide is released in response to increased atrial volume and pressure
- Brain (B-type) natriuretic peptide (BNP) is released from the ventricle in response to ventricular stretching
- Fewer endogenous vasodilators are produced, and more endogenous vasoconstrictors are produced, thus increasing afterload
- The failing heart and other organs produce tumor necrosis factor (TNF)- α

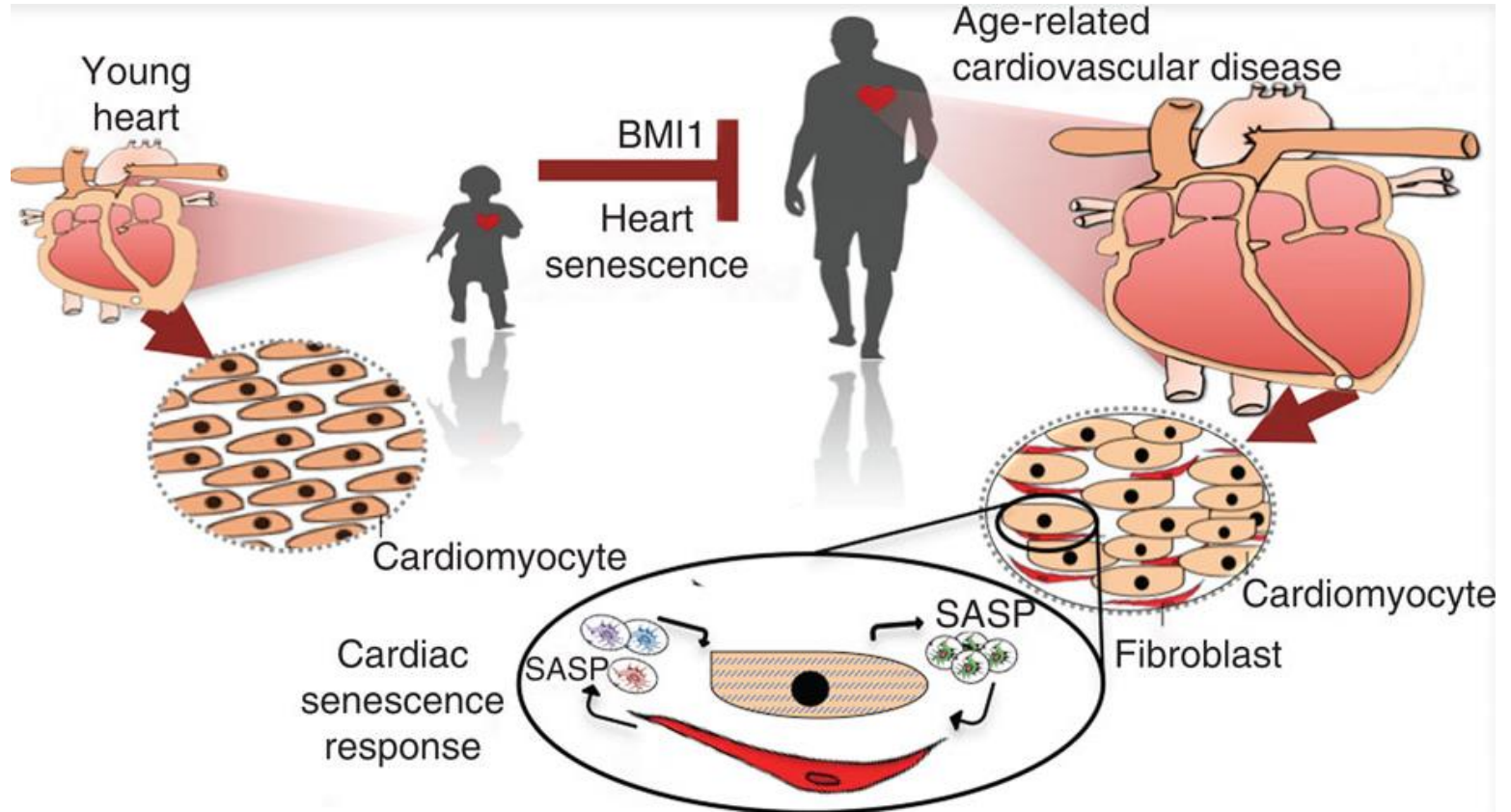
Cardiovascular response 14



Cardiovascular response 15

- Age-related changes in the heart and cardiovascular system lower the threshold for expression of HF
- Interstitial collagen within the myocardium increases, the myocardium stiffens, and myocardial relaxation is prolonged with a significant reduction in diastolic LV function, even in healthy people
- An age-related decrease in myocardial and vascular responsiveness to β -adrenergic stimulation further impairs the ability of the cardiovascular system to respond to increased work demands

Cardiovascular response 15

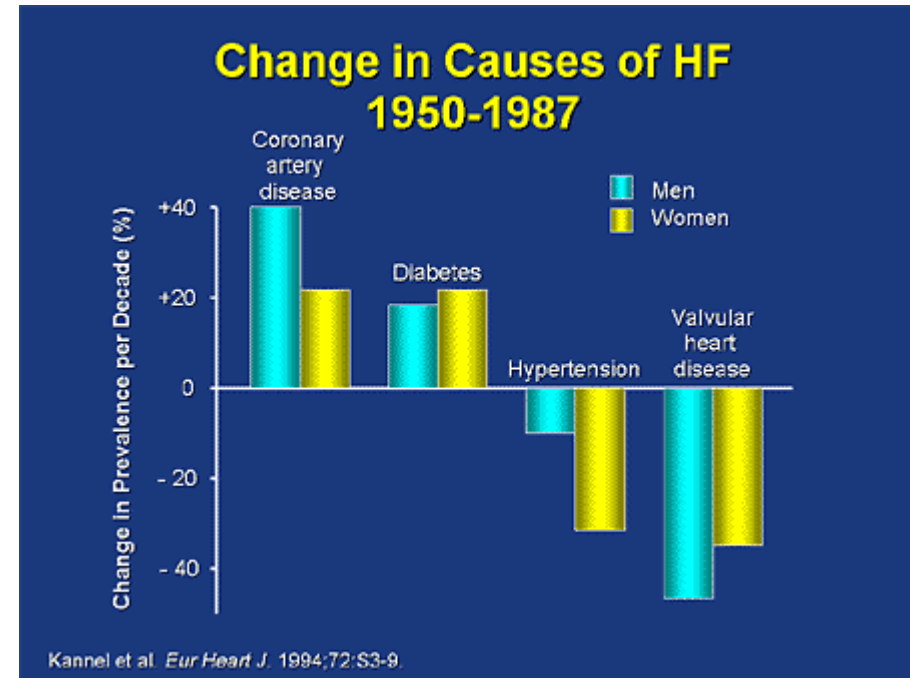


Changes with aging

Causes 1

Heart failure can complicate any cardiovascular disease sooner or later

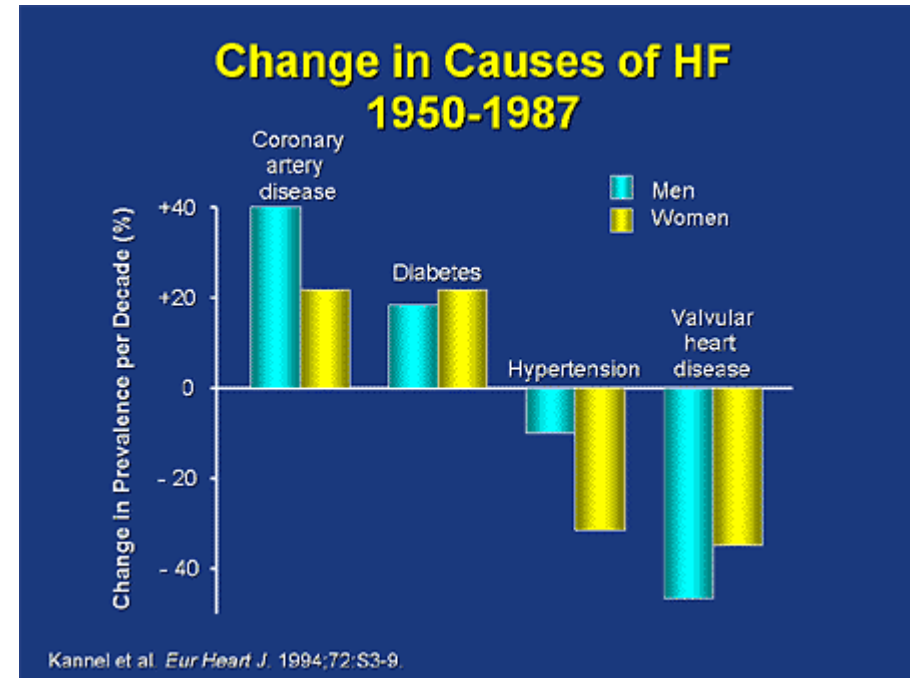
- Coronary artery disease
- High blood pressure
- Atrial fibrillation
- Valvular heart disease
- Excess alcohol use
- Infection
- Cardiomyopathy



Causes 2

Heart failure can complicate any cardiovascular disease sooner or later

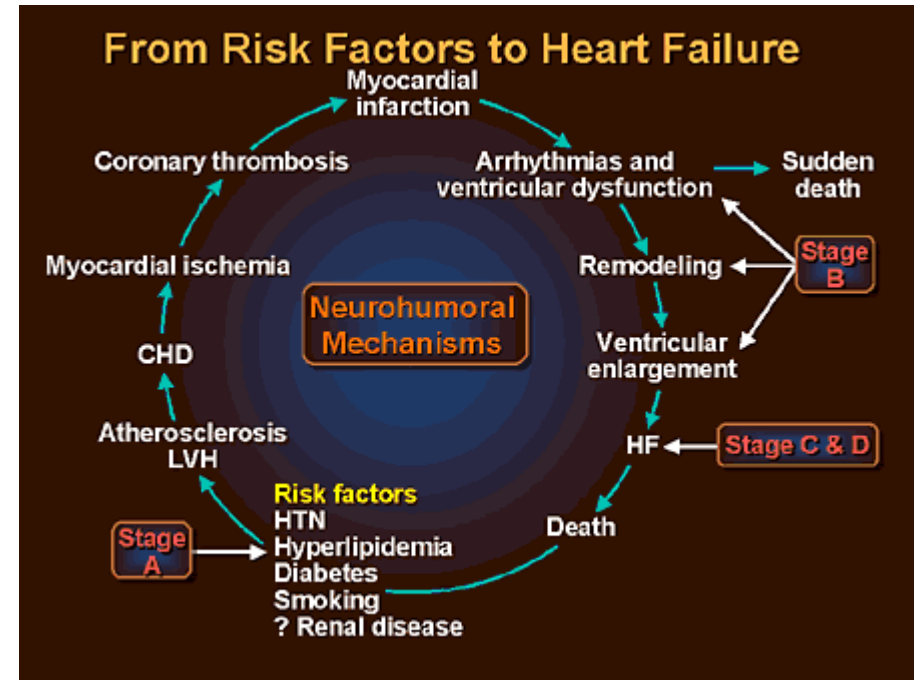
- Thyroid disease
- Kidney disease
- Diabetes
- Heart defects present at birth



Risk factors 1

Risk factors for cardiovascular diseases = risk factors for heart failure

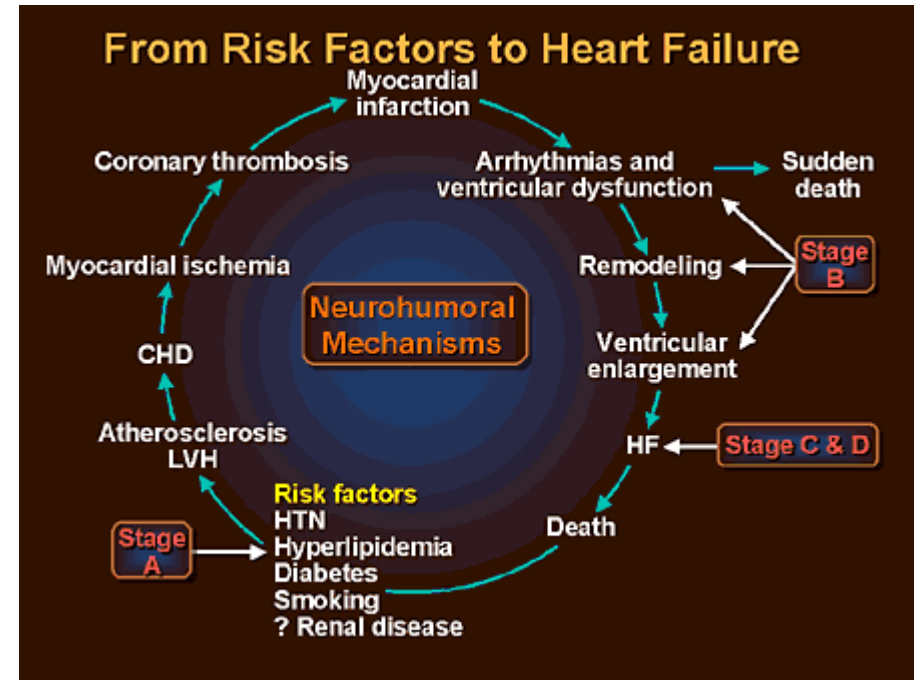
- High blood pressure
- Coronary artery disease
- Heart attack
- Diabetes
- Some diabetes medications
- Sleep apnea
- Congenital heart defects



Risk factors 2

Risk factors for cardiovascular diseases
= risk factors for heart failure

- Valvular heart disease
- Viruses
- Alcohol use
- Tobacco use
- Obesity
- Irregular heartbeats



Classification 1

- Acute or chronic (congestive)
- High output or low output
- Systolic or diastolic
- Left heart or right heart or biventricular
- Dilated or nondilated
- Cause: ischemic, hypertensive, idiopathic dilated cardiomyopathy, etc.



Classification 2

heart failure stage (the American College of Cardiology classification)

Stage	Definition of Stage
A	High risk of heart failure (HF) but no structural heart disease or symptoms
B	Structural heart disease but no symptoms of HF
C	Structural heart disease with symptoms of HF
D	Refractory HF requiring specialized interventions

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Stages of heart failure (CHF)

A – Asymptomatic high risk patients

B – Myocardial damage without symptoms

C – LV systolic dysfunction and symptomatic CHF

D – End-stage or refractory chronic heart failure

Source: Geriatrics Aging © 2008 1453987 Ontario, Ltd.

Classification 2

heart failure class (New York Heart Association (NYHA) Classification)

NYHA Class	Definition	Limitation
I	Ordinary physical activity does not cause undue fatigue, dyspnea, or palpitations.	None
II	Ordinary physical activity causes fatigue, dyspnea, palpitations, or angina.	Mild
III	Comfortable at rest; less than ordinary physical activity causes fatigue, dyspnea, palpitations, or angina.	Moderate
IV	Symptoms occur at rest; any physical activity increases discomfort.	Severe

Clinical picture 1

- Shortness of breath (dyspnea)
- Fatigue and weakness
- Swelling (edema) in legs, ankles and feet
- Rapid or irregular heartbeat
- Reduced ability to exercise
- Persistent cough or wheezing with white or pink blood-tinged phlegm
- Increased need to urinate at night

Clinical picture 2

- Swelling of abdomen (ascites)
- Sudden weight gain from fluid retention
- Lack of appetite and nausea
- Difficulty concentrating or decreased alertness
- Sudden, severe shortness of breath and coughing up pink, foamy mucus
- Chest pain if heart failure is caused by a heart attack

Clinical picture: left heart failure 1

- The most common symptoms are dyspnea, reflecting pulmonary congestion, and fatigue, reflecting low cardiac output (CO)
- Dyspnea occurs during exertion and is relieved by rest, and as heart failure (HF) worsens, it can occur during rest and at night, sometimes causing nocturnal cough

Clinical picture: left heart failure 2

- In paroxysmal nocturnal dyspnea (PND), dyspnea awakens patients several hours after they lie down and is relieved only after they sit up for 15 to 20 min
- In severe HF, periodic cycling of breathing (Cheyne-Stokes) can occur during the day or night
- Sleep-related breathing disorders, such as sleep apnea, are common in HF and may aggravate HF
- Severely reduced cerebral blood flow and hypoxemia can cause chronic irritability and impair mental performance

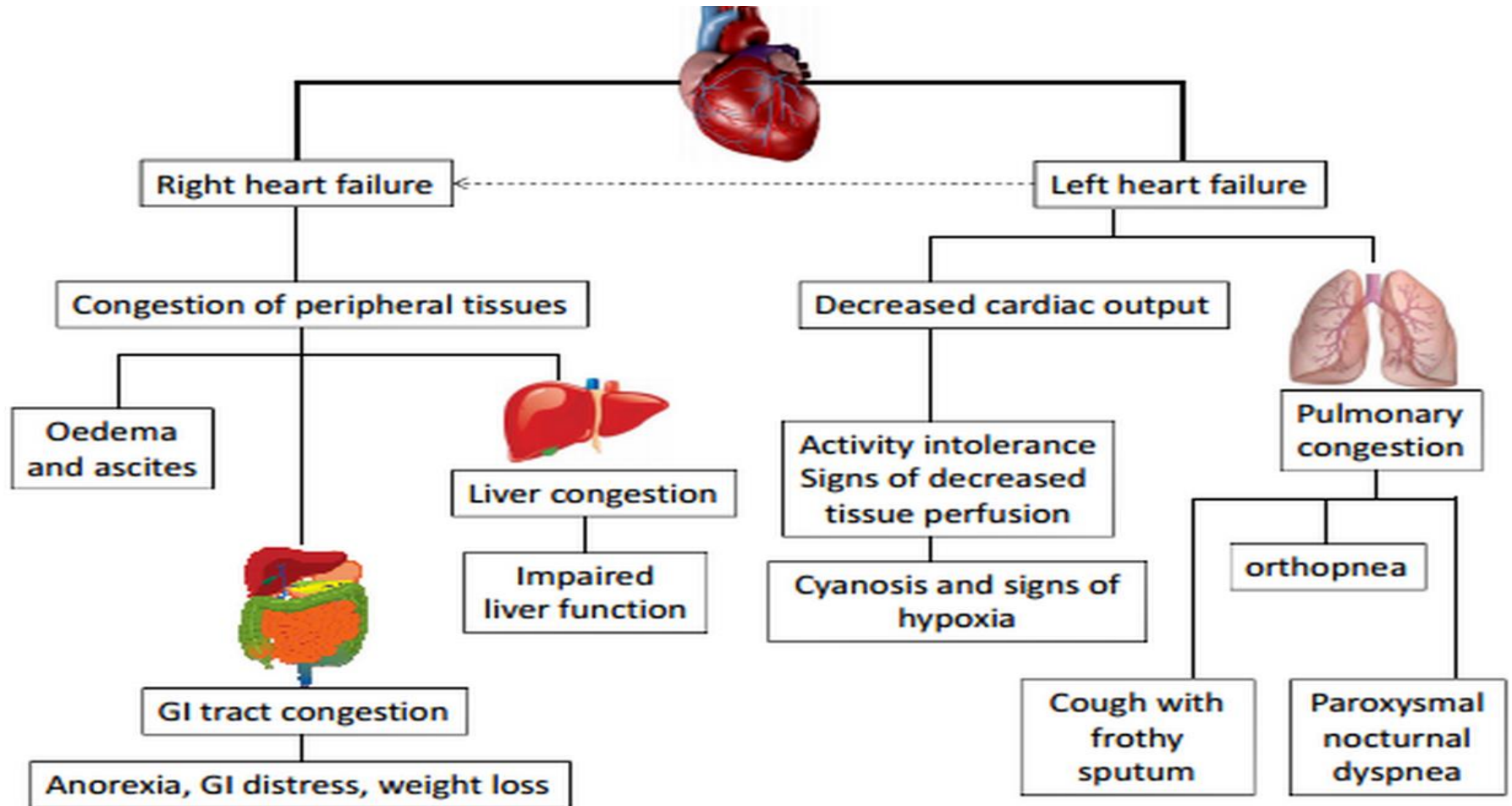
Clinical picture: right heart failure 1

- The most common symptoms are ankle swelling and fatigue
- Hepatic congestion can cause right upper quadrant abdominal discomfort, and stomach and intestinal congestion can cause anorexia and abdominal bloating
- Less specific symptoms include cool peripheries, postural light-headedness, nocturia, and decreased daytime micturition

Clinical picture: right heart failure 2

- Skeletal muscle wasting can occur in severe biventricular failure and may reflect increased catabolism with increased cytokine production
- Significant weight loss (cardiac cachexia) is an ominous sign associated with high mortality
- In the elderly, presenting complaints may be atypical, such as confusion, delirium, falls, sudden functional decline, nocturnal urinary incontinence, or sleep disturbance
- Coexisting cognitive impairment and depression

Clinical picture: left and right heart failure



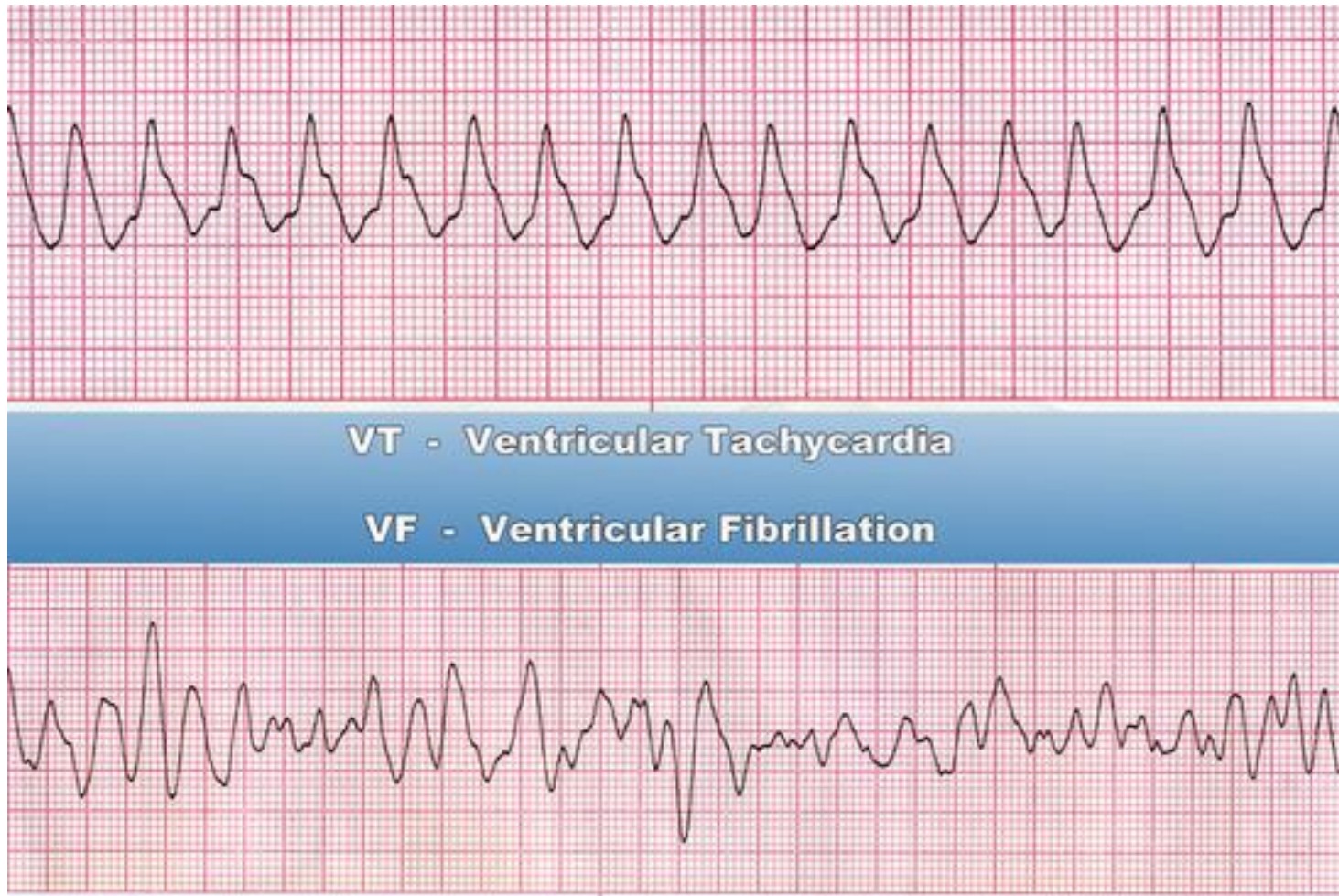
Diagnosis 1

- In left heart (left ventricle - LV) failure, tachycardia and tachypnea, hypotension, and confusion because of hypoxia and poor cerebral perfusion may occur
- Central cyanosis reflects severe hypoxemia; peripheral cyanosis of the lips, fingers, and toes reflects low blood flow with increased O₂ extraction

Diagnosis 2

- LV systolic dysfunction include a diffuse, sustained, and laterally displaced apical impulse; audible and occasionally palpable 3rd (S_3) and 4th (S_4) heart sounds, and an accentuated pulmonic component (P_2) of the 2nd heart sound (S_2); a pansystolic murmur of mitral regurgitation at the apex may occur
- Pulmonary findings include early inspiratory basilar crackles that do not clear with coughing and, if pleural effusion is present, dullness to percussion and diminished breath sounds at the lung base(s)

Diagnosis: tachycardia



Diagnosis: central cyanosis

Central cyanosis is caused by abnormal composition of hemoglobin such as sulphaemoglobinaemia and methaemoglobinaemia or decreased in the saturation of the oxygen because of cyanotic congenital heart disease, pulmonary embolism, pulmonary edema (required urgent treatment) and severe respiratory disease



Diagnosis: peripheral cyanosis

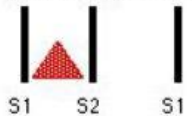


- Peripheral cyanosis can be a result of the causes of central cyanosis or can occur in isolation
- Common causes of peripheral cyanosis without central cyanosis are:
 - Peripheral vasoconstriction due to cold, Raynaud's phenomenon or beta blocker drugs
 - Reduced cardiac output due to cardiac failure or hypovolemia
 - Peripheral vascular disease
 - Venous obstruction (deep vein thrombosis, etc.)

Diagnosis: heart sounds

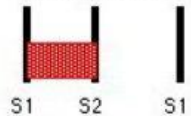
Murmurs and Extra Sounds

Systolic Ejection



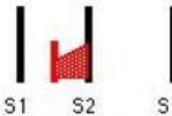
Innocent/Physiologic
Aortic/Pulmonic Stenosis

Pansystolic



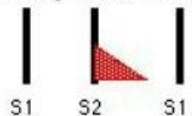
Mitral/Tricuspid Regurgitation

Systolic Click Late Systolic



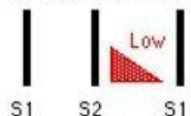
Mitral Valve Prolapse

Early Diastolic



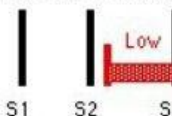
Aortic Regurgitation

Mid Diastolic



Mitral/Tricuspid Stenosis

Opening Snap Diastolic Rumble



Mitral Stenosis

Ejection Sound



Aortic Valve Disease

S3

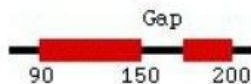


Normal in Children
Heart Failure

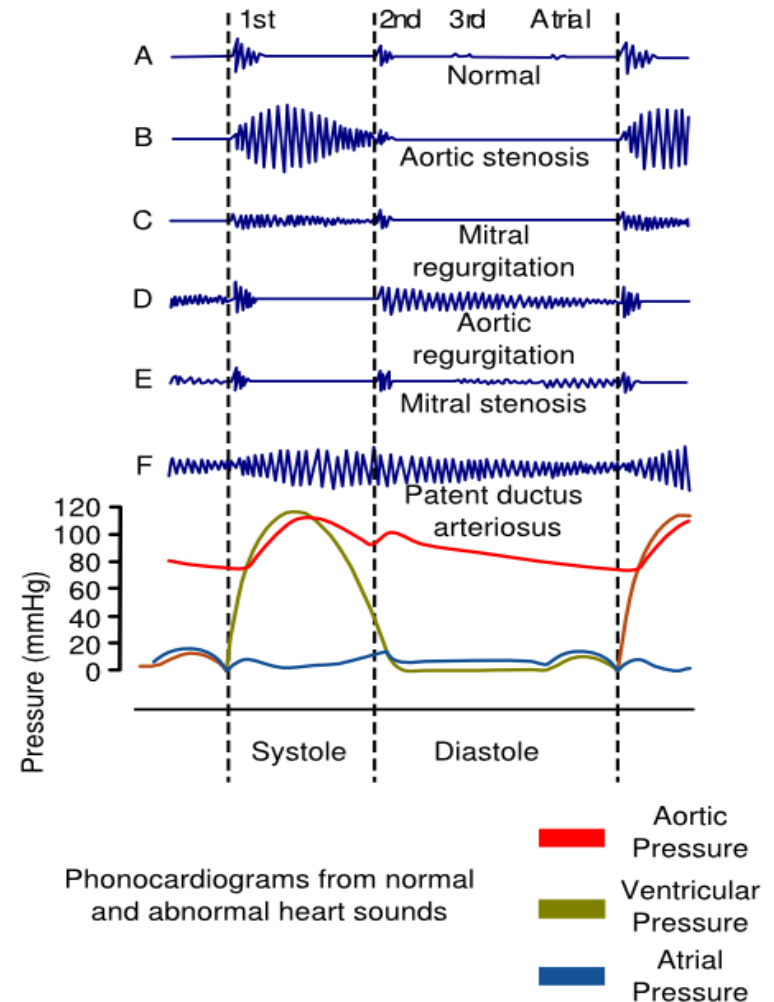
S4



Physiologic
Various Diseases



False BP 150/90
Actual BP 200/90



Diagnosis: right heart failure 1

- Nontender peripheral pitting edema (digital pressure leaves visible and palpable imprints, sometimes quite deep) in the feet and ankles; an enlarged and sometimes pulsatile liver palpable below the right costal margin; abdominal swelling and ascites; and visible elevation of the jugular venous pressure, sometimes with large *a* or *v* waves that are visible even when the patient is seated or standing

Diagnosis: right heart failure 2

- In severe cases, peripheral edema can extend to the thighs or even the sacrum, scrotum, lower abdominal wall, and occasionally even higher (anasarca); edema may be asymmetric if patients lie predominantly on one side
- With hepatic congestion, the liver may be palpably enlarged or tender, and hepato jugular or abdominal-jugular reflux may be detected

Diagnosis: right heart failure 3

- Precordial palpation may detect the left parasternal lift of RV enlargement, and auscultation may detect the murmur of tricuspid regurgitation or the RV S_3 along the left sternal border; both findings are augmented upon inspiration

Diagnosis: peripheral edema



Diagnosis: anasarca)



Anasarka is whole body edema

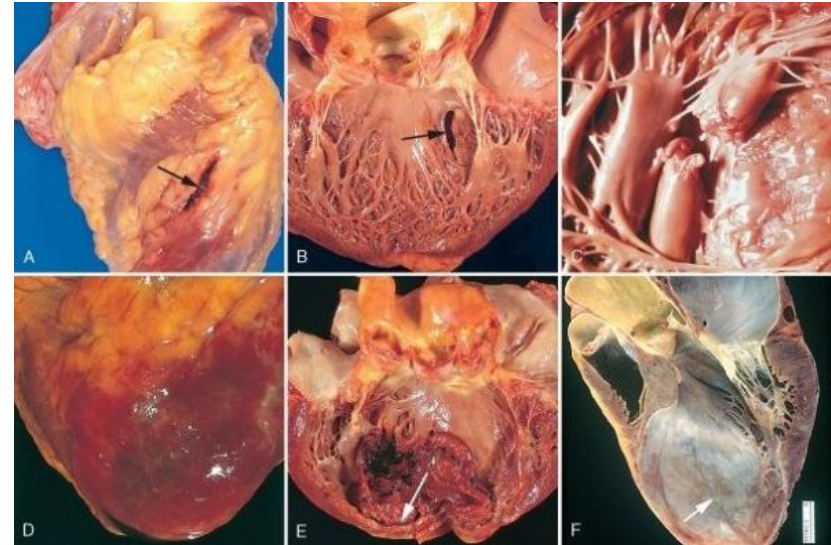
Diagnosis

Left HF	Right HF
Cardio, Fatigue dyspnea	Fatigue dyspnea Cardiomegaly
S3/S4 L	

- Sometimes only clinical evaluation
- Visualization (chest x-ray, electrocardiography, echocardiography, cardiac radionuclide scan, magnetic resonance imaging)
- Sometimes blood NP/BNP or N-terminal-pro-BNP (NT-pro-BNP) levels (other blood tests are not used for diagnosis but are useful for identifying cause of heart failure)
- Other tests for etiology as needed

Diagnosis: clinical evaluation

- Clinical findings suggest heart failure (HF) but are usually not apparent early
- Similar symptoms may result from chronic obstructive pulmonary disease (COPD) or recurrent pneumonia or may be erroneously attributed to obesity or old age
- Suspicion for HF should be high in patients with a history of myocardial infarction (MI), hypertension, or valvular disorders or murmurs and should be moderate in any patient who is elderly or has diabetes



Complications of acute myocardial infarction

- A. Anterior wall rupture**
- B. Interventricular septum rupture**
- C. Papillary muscle rupture**
- D. Fibrinous pericarditis**
- E. Mural thrombus**
- F. Ventricular aneurysm**

Diagnosis: chest x-ray



- Chest x-ray findings include an enlarged cardiac silhouette, pleural effusion, fluid in the major fissure, and horizontal lines in the periphery of lower posterior lung fields (Kerley B lines)
- The x-ray may also suggest alternative diagnoses (e.g., chronic obstructive pulmonary disease (COPD), pneumonia, interstitial pulmonary fibrosis, lung cancer)

Diagnosis: electrocardiography

- Electrocardiography (ECG) findings are not diagnostic, but an abnormal ECG, especially showing previous myocardial infarction (MI), left ventricle (LV) hypertrophy, left bundle branch block, or tachyarrhythmia (e.g., rapid atrial fibrillation), increases suspicion for heart failure (HF) and may help identify the cause
- An entirely normal ECG is uncommon in chronic HF

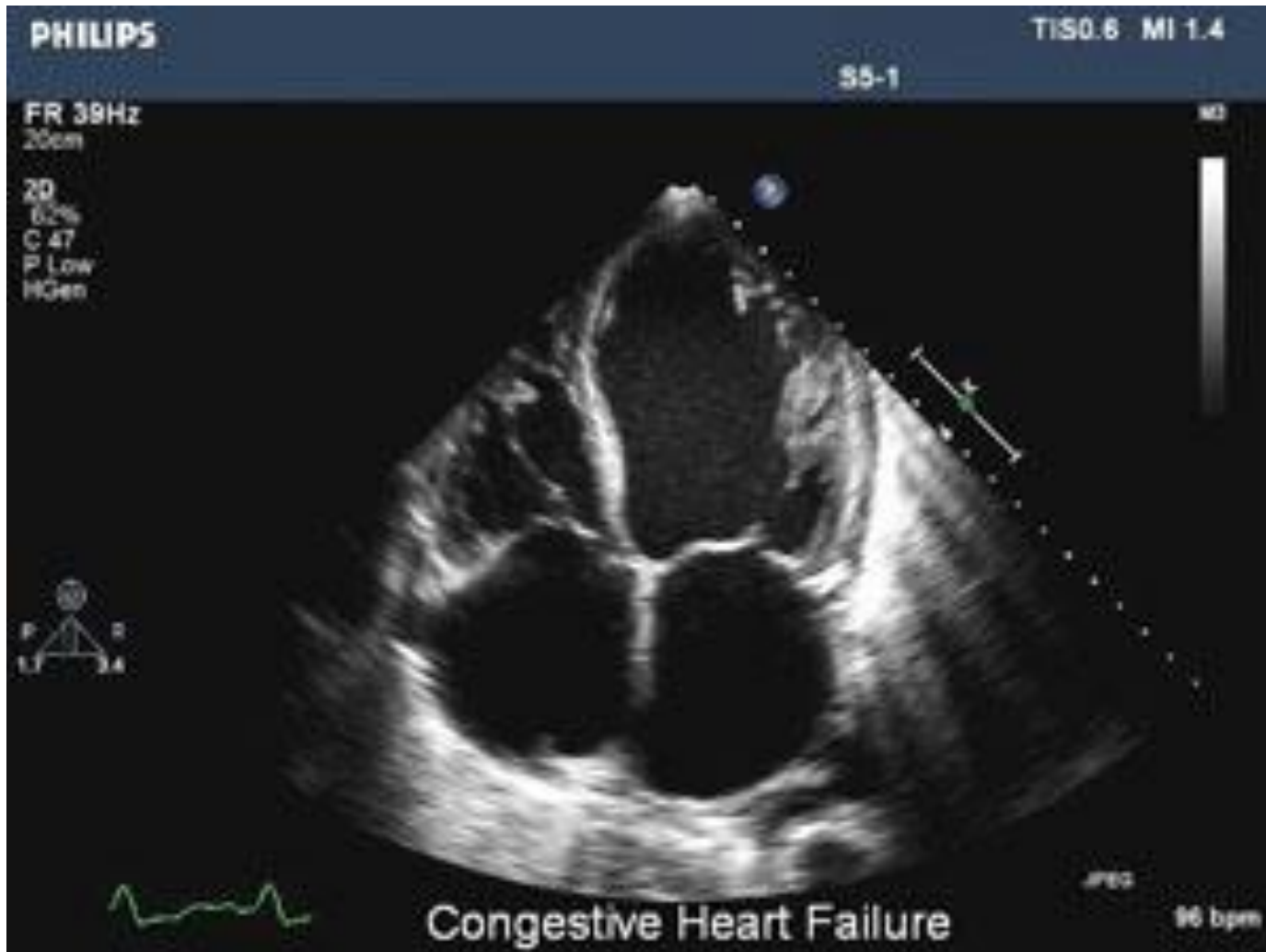
Diagnosis: echocardiography 1

- Echocardiography can help evaluate chamber dimensions, valve function, ejection fraction (EF), wall motion abnormalities, left ventricle (LV) hypertrophy, and pericardial effusion
- Measuring LV EF can distinguish between predominant diastolic dysfunction ($EF > 0.50$) and systolic dysfunction ($EF < 0.40$)
- Intracardiac thrombi, tumors, and calcifications within the heart valves, mitral annulus, and aortic wall abnormalities can be detected

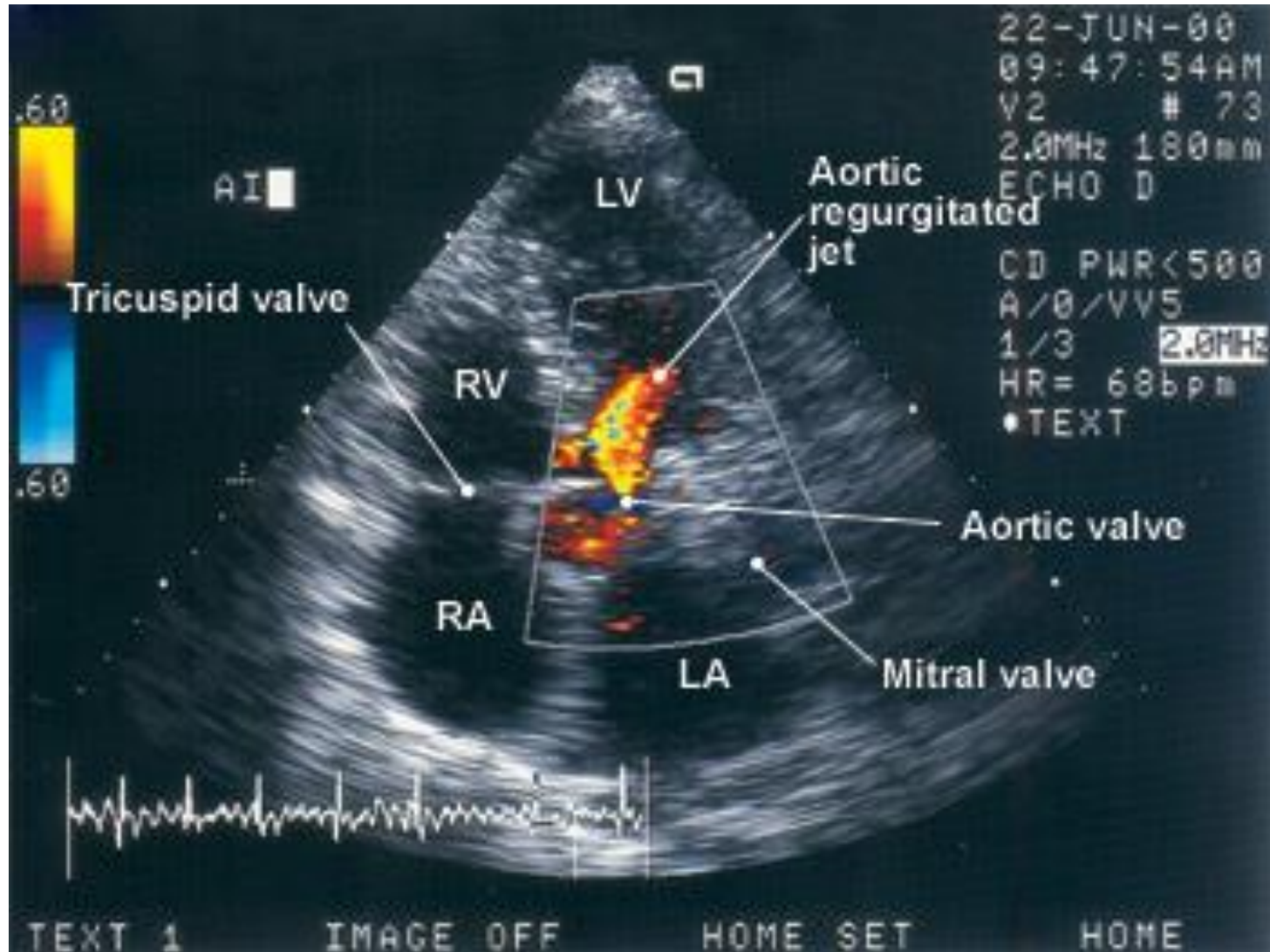
Diagnosis: echocardiography 2

- Localized or segmental wall motion abnormalities strongly suggest underlying coronary artery disease (CAD) but can also be present with patchy myocarditis
- Doppler echocardiography accurately detects valvular disorders and shunts, can help identify and quantify LV diastolic dysfunction

Diagnosis: echocardiography 3



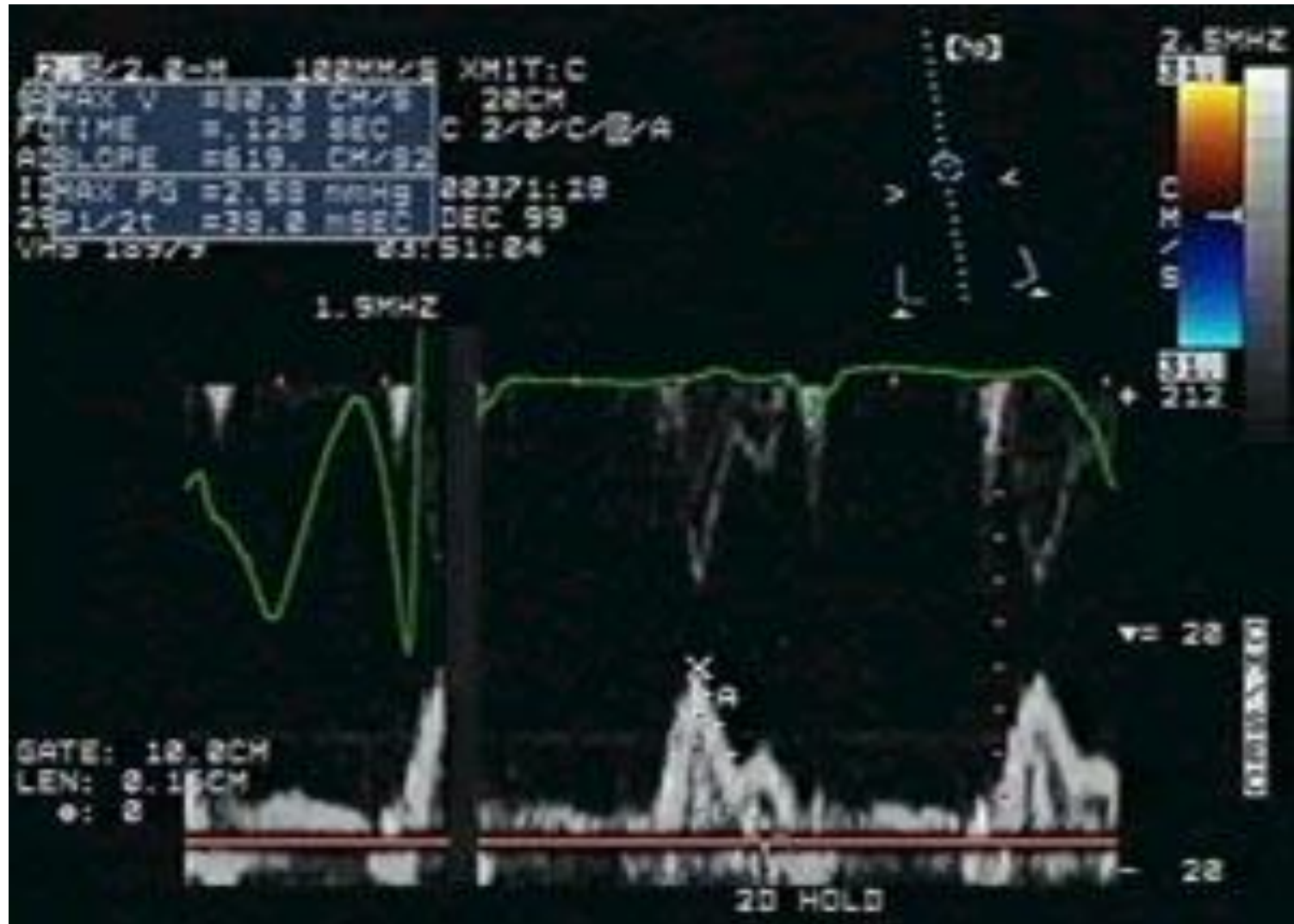
Diagnosis: echocardiography 4



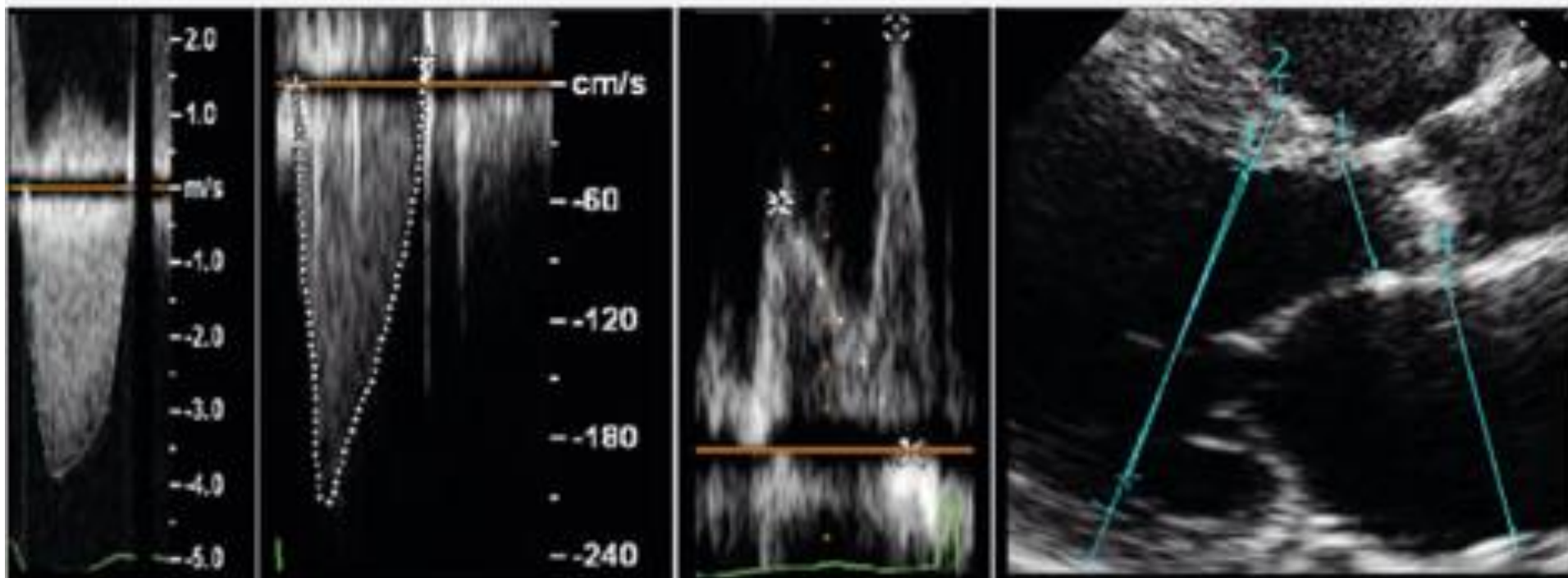
Diagnosis: echocardiography 5



Diagnosis: echocardiography 6

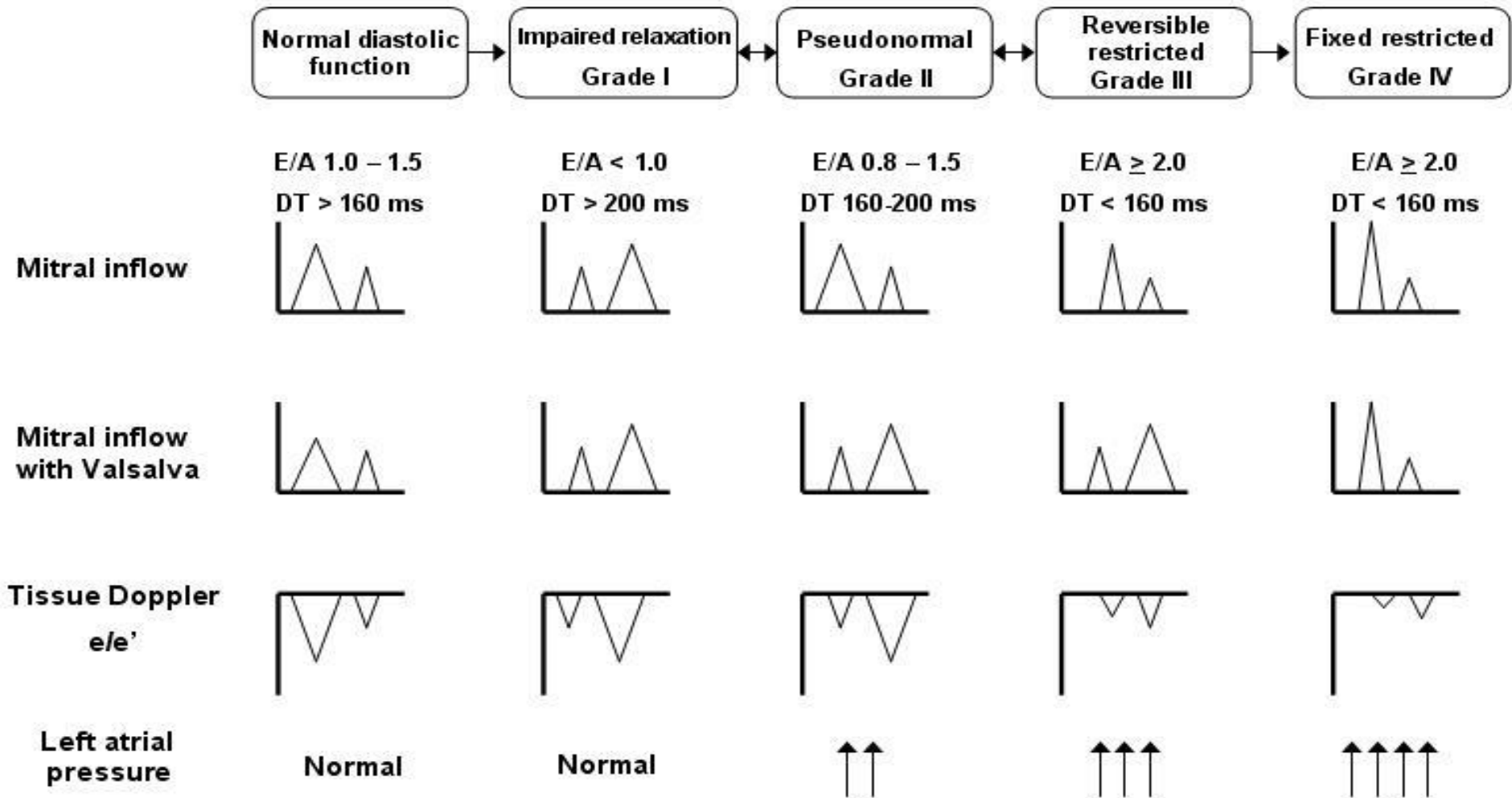


Diagnosis: echocardiography 7

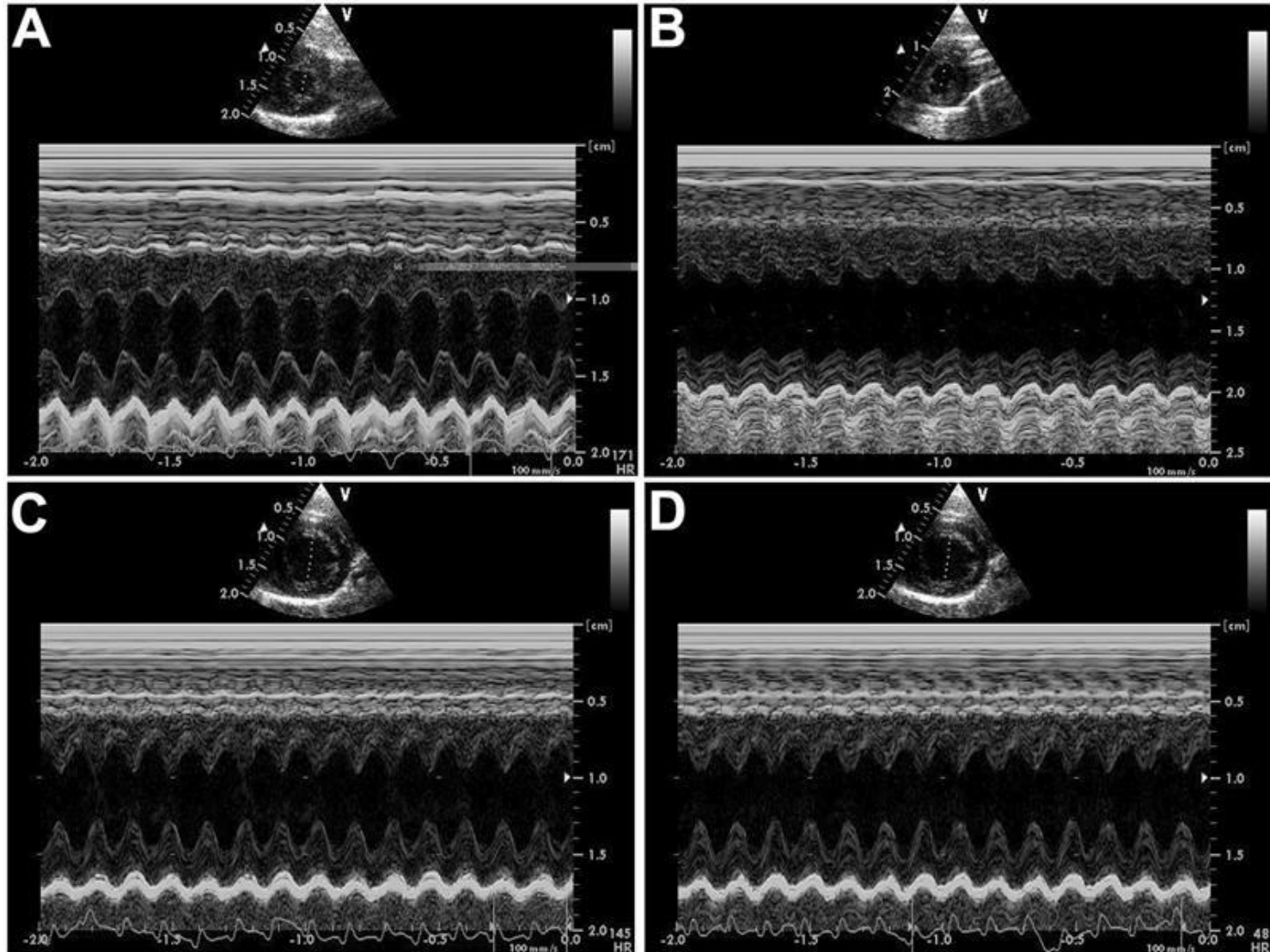


Diagnosis: echocardiography 8

Echocardiographic classification of diastolic dysfunction

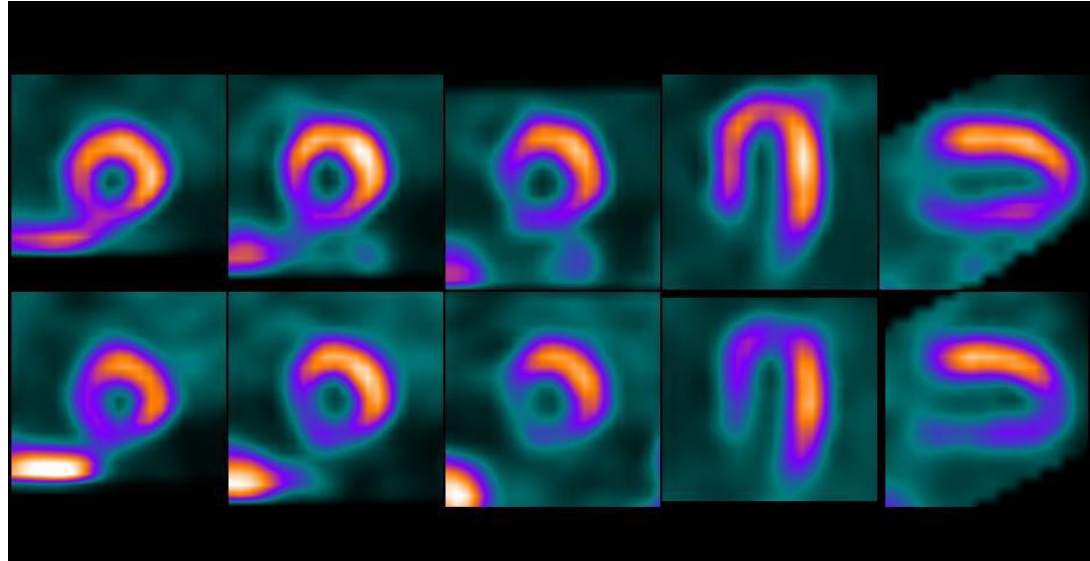


Diagnosis: echocardiography 9



Diagnosis: cardiac radionuclide scan

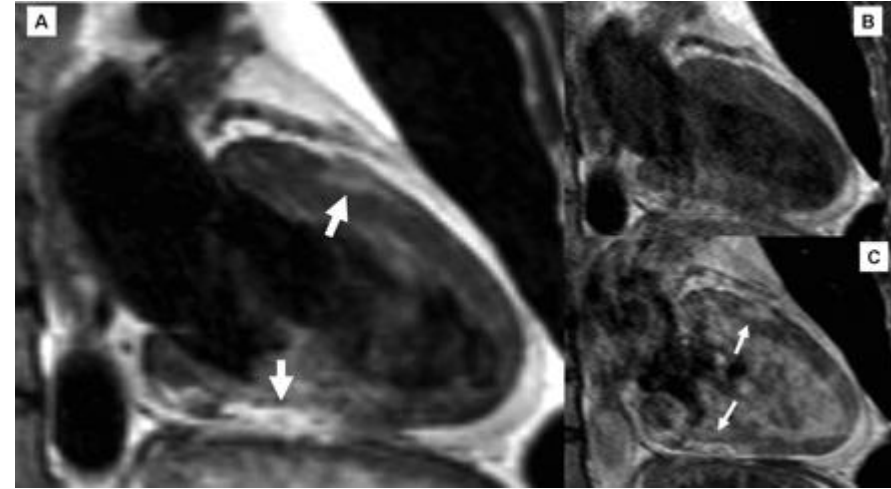
Radionuclide imaging can help assess systolic and diastolic function, previous myocardial infarction (MI), and inducible ischemia or myocardial hibernation



A myocardial perfusion SPECT (Single Photon Emission Computed Tomography) study, also called a cardiac stress-rest test, is used to evaluate the heart's blood supply. Two sets of images showing blood flow are obtained: the first following a period of rest, and the second following a period of stress, which involves exercise on a treadmill.

Diagnosis: magnetic resonance imaging

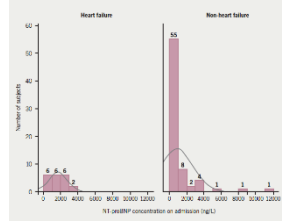
Cardiac magnetic resonance imaging (MRI) provides accurate images of cardiac structures and is becoming more widely available



T2-weighted dark-blood imaging of the left ventricular long axis showing high signal intensity in the left ventricular anterior and inferior wall, a sign of myocardial edema. A, T1-weighted dark-blood imaging (early enhancement) shows high signal intensity directly after gadolinium administration in the corresponding area of the anterior left ventricular wall. Arrows indicate myocardial edema. B, T1-weighted imaging before gadolinium administration. C, Arrows indicate myocardial early enhancement.

Diagnosis:

NP/BNP or N-terminal-pro-BNP levels



- Serum NP/BNP levels are high in heart failure (HF); this finding may help when clinical findings are unclear or other diagnoses (e.g., chronic obstructive pulmonary disease (COPD)) need to be excluded
- It may be particularly useful for patients with a history of both pulmonary and cardiac disorders
- NT-pro-BNP, an inactive moiety created when pro BNP is cleaved, can be used similarly to NP/BNP

Diagnosis: the Framingham criteria 1

- Diagnosis requires the simultaneous presence of at least 2 of the major criteria or 1 major criterion in conjunction with 2 of the minor criteria
- Major criteria include an enlarged heart, an S3 gallop, acute pulmonary edema, episodes of waking up from sleep gasping for air, crackles on lung auscultation, central venous pressure of more than 16 cm H₂O at the right atrium, jugular vein distension, positive abdomino jugular test, and weight loss of more than 4.5 kg in 5 days in response to treatment

Diagnosis: the Framingham criteria 2

- Minor criteria include an abnormally fast heart rate of more than 120 beats per minute, nocturnal cough, difficulty breathing with physical activity, pleural effusion, a decrease in the vital capacity by one third from maximum recorded, liver enlargement, and bilateral ankle swelling

Complications

- Kidney damage or failure
- Heart valve problems
- Heart rhythm problems
- Liver damage

