===Heart failure describes the clinical syndrome that develops when the heart cannot maintain an adequate cardiac output, or can do so only at the expense of an elevated filling In practice, heart failure may be diagnosed whenever a patient with significant .pressure. heart disease develops the signs or symptoms of a low cardiac output, pulmonary

congestion or systemic venous congestion.

زAlmost all forms of heart disease can lead eventualy to heart failure

===:Mechanisms of heart failure

=:Reduced ventricular contractility.

MI (segmental dysfunction): In coronary artery disease, 'akinetic' or 'dyskinetic' segments :contract poorly and may impede the function of normal segments -by distorting their contraction and relaxation patterns

Myocarditis/cardiomyopathy (global dysfunction) :Progressive ventricular dilatation

=Ventricular outflow.

(obstruction (pressure overload

Hypertension, aortic stenosis (left heart failure):Initially, concentric ventricular hypertrophy allows the ventricle to

maintain a normal output by generating a high systolic pressure. Later, secondary changes in the myocardium and increasing obstruction

.lead to failure and rapid clinical deterioration

(Pulmonary hypertension, pulmonary valve stenosis (right heart failure

=Ventricular inflow obstruction.

Mitral stenosis: Small vigorous ventricle, dilated hypertrophied atrium. Atrial fibrillation is common and often causes marked deterioration because ventricular filling depends .heavily on atrial contraction,esp. in elderlies

=Ventricular volume overload.

.Ventricular septal defect_PDA

(Right ventricular volume overload (e.g. atrial septal defect

Increased metabolic demand (high output .

Dilatation and hypertrophy allow the ventricle to generate a high stroke volume and help to .maintain a normal cardiac function ,on expense of gradually elevated LVEDP

,=Arrhythmia Atrial fibrillation: Tachycardia does not allow for adequate filling of the heart .

resulting in reduced cardiac output and back pressure

=.Tachycardia cardiomyopathy :Incessant tachycardia causes myocardial fatigue,esp. AT.

=Complete heart block :Bradycardia limits cardiac output even if stroke volume is normal, .but with bradycardia and wide pulse pressure

=Diastolic dysfunction.

Constrictive pericarditis:Marked fluid retention and peripheral oedema, ascites, pleural effusions and elevated jugular veins

Restrictive cardiomyopathy(rare) :Bi-atrial enlargement (restrictive filling pattern and high atrial pressures). Atrial fibrillation may cause deterioration

Left ventricular hypertrophy and fibrosis: Good systolic function but poor diastolic filling

Cardiac tamponade Hypotension, elevated jugular veins, pulsus paradoxus, poor urine

.(output(emergency

===:Pathophysiology***

Heart failure carries a very poor prognosis; approximately 50% of patients with severe heart _failure due to left ventricular dysfunc

tion will die within 2 years. Many patients die suddenly from malignant .ventriculaarrhythmias or MI

Cardiac output is a function of the preload (the volume and pressure of blood in the ventricle at the end of diastole), the afterload (the volume and pressure of blood in the .ventricle during systole) and myocardial contractility; this is the basis of Starling's Law

Fall in cardiac output activates counter_ regulatory neurohumoral mechanisms that in normal physiological circumstances would support cardiac function, but in the

setting of impaired ventricular function can lead to a deleterious increase in both afterload .and preload

Stimulation of the renin – angiotensin – aldosterone system leads to vasoconstriction, salt and water retention, and sympathetic nervous system activation. This is mediated by angiotensin II, a potent constrictor of arterioles in both the kidney and the systemic circulation . Activation of the sympathetic nervous system may initially maintain cardiac output through an increase in myocardial contractility, heart rate and peripheral vasoconstriction. However, prolonged sympathetic stimulation leads to salt and water retention , promoted by the release of aldosterone, endothelin-1 (a potent vasoconstrictor peptide with marked effects on the renal vasculature) and, in severe heart failure, antidiuretic hormone (ADH). Natriuretic peptides are released from the atria in response to

atrial stretch, and act as physiological antagonists to the fluid conserving effect of .aldosterone

After MI.: hypertrophy/ hyperstimulation/ overloading of non-infarcted segments, with thinning, dilatation and expansion of the

.infarcted segment leads to remodelling

.This leads to further deterioration in ventricular function and worsening heart failure

.The onset of pulmonary and peripheral oedema is secondary hyperaldosteronism

===:Types of heart failure***

=Left-sided heart failure. There is a reduction in the left ventricular output and an increase ... ,in the left atrial or pulmonary venous pressure,causes pulmonary oedema; later

leads to reflex pulmonary vasoconstriction, which protects the patient from pulmonary .(oedema at the cost of increasing pulmonary hypertension(as a complication

= Right-sided heart failure: include chronic...

.lung disease (cor pulmonale), multiple pulmonary emboli and pulmonary valvular stenosis

=Biventricular heart failure: Failure of the left then right heart may develop because the ... disease process, such as dilated cardiomyopathy or ischaemic heart

(disease(complicated by pulm. hypertension

=Diastolic Heart failure may develop as a result of impaired ventricular filling and high ... filling pressures caused by abnormal ventricular relaxation, caused by a stiff non-compliant ventricle and is commonly found in patients with left ventricular hypertrophy(as in (.HT./AS./HCMP

Systolic and diastolic dysfunction often coexist, particularly in patients with coronary artery .disease

Conditions such as large arteriovenous shunt, beri-beri , severe =High-output failure... anaemia or thyrotoxicosis can occasionally cause heart failure due to an excessively high .cardiac output , in a presence of structurally normal heart and valves

=Acute left heart failure occurs either de novo(as in extensive STMI.), or as an acute decompensated episode on a background of chronic heart failure(as in chronic severe .(valvular diseases), so-called acute-on-chronic heart failure (due to decompensating factors

===Factors that may precipitate or aggravate heart failure in patients with pre-existing *** :heart disease

Myocardial ischaemia or infarction .

Intercurrent illness, e.g. infection •

- Arrhythmia, e.g. atrial fibrillation
- Inappropriate reduction of therapy
- Administration of a drug with negative inotropic properties (e.g. β-blocker) or fluid--retaining properties (e.g. non
- (steroidal anti-inflammatory drugs (NSAIDs), corticosteroids
- Pulmonary embolism
- Conditions associated with increased metabolic demand, e.g. pregnancy, thyrotoxicosis, anaemia
- .I.v. fluid overload, e.g. post-operative i.v. infusion

===Clinical assessment***

=Acute left heart failure...

Acute de novo left ventricular failure presents with a acute onset of dyspnoea at rest that rapidly progresses to acute respiratory distress, orthopnoea . The precipitant, such as acute . MI, is often apparent

The patient appears agitated, pale and clammy. The peripheries are cool to the touch and .the pulse is rapid

.Inappropriate bradycardia or excessive tachycardia should be identified

The BP is usually high because of sympathetic nervous system activation, (low if the patient is in cardiogenic shock). In acute de novo heart failure, there has been no time for ventricular dilatation and the apex is not displaced./ no cardiomegaly by CXR. or Echo. Auscultation occasionally identifies the murmur of a catastrophic valvular (although, faint , difficult to identify),or septal rupture, or reveals a triple 'gallop' rhythm. Crepitations are .heard at both lung bases, consistent with pulmonary oedema

=Acute-on-chronic heart failure will have additional features of long-standing heart failure . Potential precipitants, such as an upper respiratory tract infection or inappropriate cessation .of diuretic medication, should be identified

=Chronic heart failure...

Patients with chronic heart failure commonly follow a relapsing and remitting course, with periods of stability and episodes of decompensation leading to worsening symptoms that .may necessitate hospitalisation

A low cardiac output causes fatigue, and a poor effort tolerance; the peripheries are cold and low radial pulse volume /BP is low/ low pulse pressure/ diminished MV. excurtion. Poor .renal perfusion leads to azotemia

Pulmonary oedema due to left heart failure presents as inspiratory crepitations over the .lung bases

In contrast, right heart failure produces a high JVP with hepatic congestion and dependent

peripheral oedema. In ambulant patients, the oedema affects the ankles, whereas in bed-bound patients it collects around the thighs and sacrum. Ascites or pleural effusion occurs in .chronic constrictive pericarditis. Heart failure is not the only cause of oedema

. (Chronic heart failure is sometimes associated with marked weight loss (cardiac cachexia

===:DDx. of peripheral oedema***

Cardiac failure: right or combined left then right heart failure, pericardial constriction, . .cardiomyopathy

- Chronic venous insufficiency: varicose veins: unilateral/ hyperpigmented skin / non .healing bleeding venous ulcer/ past hx. of DVT
- Hypoalbuminaemia: nephrotic syndrome, liver disease, protein-losing enteropathy(rare); often widespread, can affect arms and face/ rapid refilling
- Drugs:Sodium retention: fludrocortisone, NSAIDs/Increasing capillary permeability: nifedipine, amlodipine
- Chronic lymphatic obstruction
- Idiopathic: women > men.

===Complications***

- =Renal failure is caused by poor renal perfusion due : to a low cardiac output and may be .. exacerbated by diuretic therapy, angiotensin-converting enzyme (ACE) inhibitors and .angiotensin receptor blockers, esp. in elderlies
- =Hypokalaemia may be the result of treatment with potassium-losing diuretics or .. hyperaldosteronism caused by activation of the renin–angiotensin system, leads to fatigue muscle weakness, constipation,and U wave in ECG. with prolonge QT, ST depression T inversion. Most of the body’s potassium is intracellular and there may be substantial depletion of potassium stores, even when the plasma potassium concentration is in the .normal range

=Hyperkalaemia may be due to the effects of drug treatment, particularly the .. • combination of ACE inhibitors and spironolactone (which both promote potassium .retention), and renal dysfunction.Causes short QT. , hyperacute T., widened QRS. , and VT

=Hyponatraemia is a feature of severe heart failure and is a poor prognostic sign. It may .. • be caused by diuretic therapy, inappropriate water retention due to high ADH .secretion.Causes hypotension, syncope, and signs of dehydration

=Impaired liver function is caused by hepatic venous congestion and poor arterial .. • perfusion, which frequently cause mild jaundice and abnormal liver function tests.Causes no .symptoms

=Thromboembolism. Deep vein thrombosis and pulmonary embolism may occur due to ..• the effects of a low cardiac output and enforced immobility, whereas systemic emboli may be related to arrhythmias, atrial flutter or fibrillation, or intracardiac thrombus complicating .conditions such as mitral stenosis, MI or left ventricular aneurysm

=Atrial and ventricular arrhythmias are very common and may be related to electrolyte ... changes (e.g. hypokalaemia), the underlying structural heart disease, and the pro-arrhythmic effects of increased circulating catecholamines or drugs. Sudden death occurs in up to 50% of patients with heart failure and is often due to a ventricular arrhythmia. Frequent ventricular ectopic beats and runs of non-sustained ventricular tachycardia are common .findings in patients with heart failure and are associated with an adverse prognosis

===Investigations***

Serum urea and electrolytes, *haemoglobin, *thyroid function,* ECG and* chest X-ray help * .to establish the cause and severity of the HF

and detect any complications. *Brain natriuretic peptide (BNP) is elevated in heart failure and is a marker of risk; (it is useful in the investigation of patients with breathlessness or .(peripheral oedema

Echocardiography is very useful and should be considered in all patients with heart failure * in order to:• determine the aetiology

,detect unsuspected valvular heartdisease •

identify patients who will benefit from long-term therapy with drugs, such as ACE • . inhibitors

Chest X-ray :A rise in pulmonary venous pressure from left-sided heart failure first shows * y as an abnormal distension of the upper lobe pulmonary veins (with the lon the chest X-ra patient in the erect position). The vascularity of the lung fields becomes more prominent, .and the right and left pulmonary arteries dilate

Subsequently, interstitial oedema causes thickened interlobular septa and dilated lymphatics. These are evident

as horizontal lines in the costophrenic angles (septal or 'Kerley B' lines). More advanced changes due to alveolar oedema cause a hazy opacification spreading from the hilar regions, .and pleural effusions

=== Acute circulatory failure (cardiogenic***

Shock)

Shock' is clinical picture of critical impairment of tissue perfusion due to acute circulatory ' .failure

.Signs: agitated/ pale/sweaty/tachycardia/weak pulse/tachypnea/sbp. below 90/oligurea

===:causes***

=:Myocardial infarction.

Shock in acute MI is due to left ventricular dysfunction . It may be due

to infarction of the RV and a variety of mechanical complications, including an acquired ventricular septal defect (due to infarction and rupture of the septum) and acute mitral .(regurgitation (due to infarction or rupture of the papillary muscles

Hypotension, oliguria, confusion and cold clammy peripheries are the manifestations of a low cardiac output, whereas breathlessness, hypoxaemia, cyanosis and inspiratory crackles .at the lung bases are typical features of pulmonary oedema

Those with cardiogenic shock should be considered for immediate intra-aortic balloon .counterpulsation and revascularisation

:=Acute massive pulmonary embolism.

This may complicate leg or pelvic vein thrombosis and usually presents with sudden collapse./ elevated jvp. / murmure of TR. Bedside echocardiography demonstrates a dilated RV;D. dimer. assay is high.(sensitive / not specific).CT pulmonary angiography

.usually provides a definitive diagnosis

:=Cardiac tamponade.

This is due to a collection of fluid or blood in the pericardial sac, compressing the heart; . Tamponade may complicate any form of pericarditis but can be due to malignant disease. Other causes include trauma and rupture of the free wall of the myocardium following MI

=:features*

Gross elevation of the JVP Hypotension• Tachycardia• Collapse• Dyspnoea•.

Pulsus paradoxus (a large fall in BP during unaudable heart sounds • • Kussmaul's sign (a paradoxical rise in the inspiration when the pulse may be impalpable)• JVP during inspiration

When there is a large pericardial effusion, the ECG complexes are small and there

may be electrical alternans: a changing axis with alternate beats caused by the heart swinging from side to side in the pericardial fluid. A chest X-ray shows an enlarged globular heart but can look normal/ with normal lung shadow. Echocardiography

is the best way of confirming the diagnosis and helps to identify the optimum site for .aspiration of the fluid

:.=Valvular HD.

may be due to the sudden onset of aortic regurgitation, mitral regurgitation or prosthetic valve dysfunction.Murmurs are often unimpressive . Transthoracic echocardiography will establish the diagnosis in most cases; however, transoesophageal echo. is helpful esp. in .prosthetic HD

:=Tachyarrythmias .

.VT.: unconscious/no breathing/pulseless / no heart sounds

AF./SVT./AT.: rapid rhythem/hemodynamically unstablIn all: DC._synchronised is always .indicated