MEDICINE

432 Team

4

Heart Failure, Etiology & Diagnosis

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COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

Objectives

1. Definition of heart failure (HF).
2. Know the Pathophysiology of HF.
3. Diagnosis of HF.
4. Describe the Causes of HF.
5. Know the Classification of HF.

Helpful Video

http://www.youtube.com/watch?v=mhYeO2fwSps

Definition:

Heart Failure is a **complex syndrome** that can result from any **structural** (from the heart) or **functional** (from outside the heart) cardiac disorder that impairs the ability of the heart to either **FILL** or **EJECT** blood. Output **doesn't meet demand = HF.**

2 types: 1- HF with Reduced ejection fraction= systolic HF (below 50%)

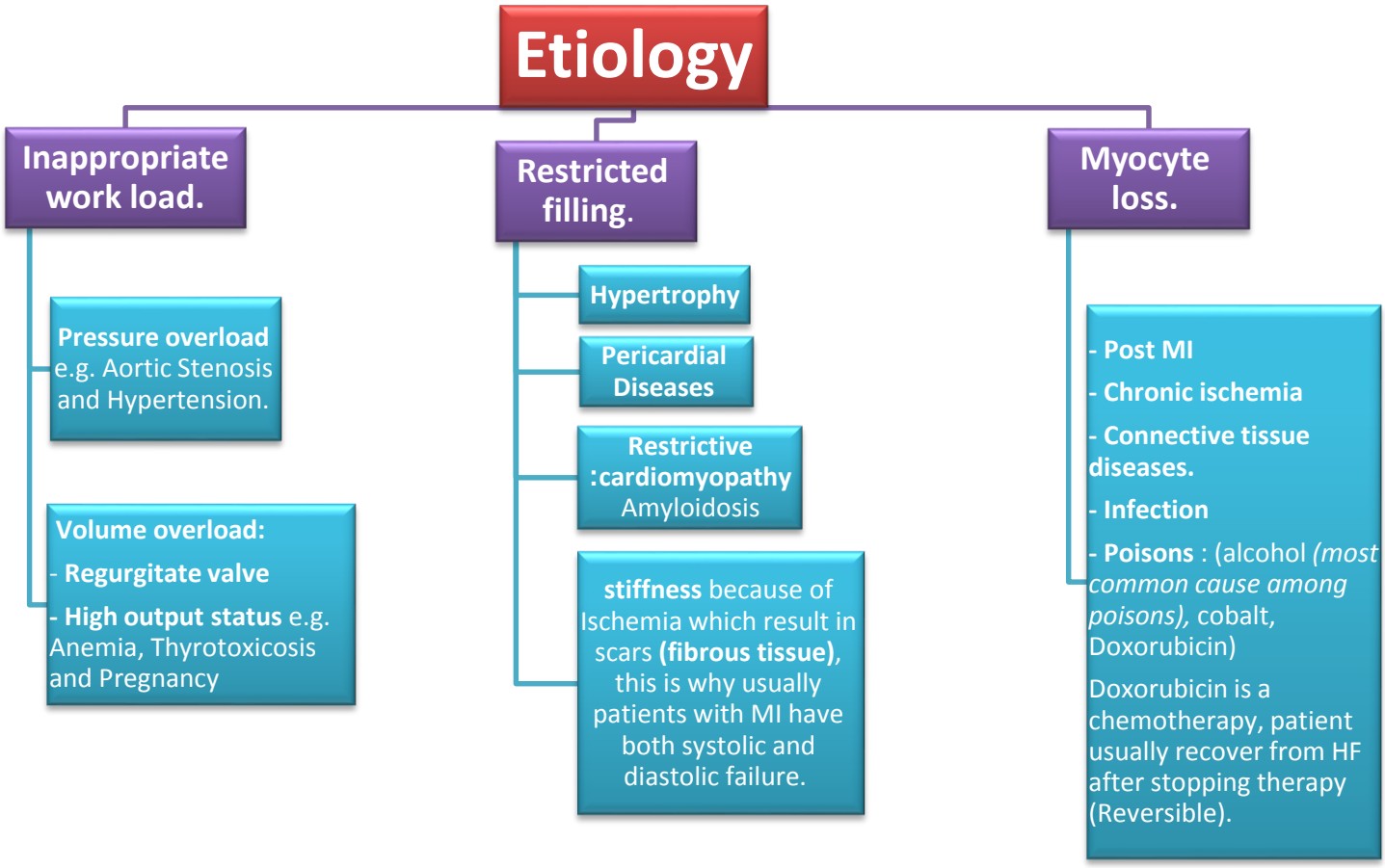
2- HF with Preserved ejection fraction= diastolic HF (above 50%)

So it's either a problem in the pre-load, after-load or pumping function.

Prevalence:

- 0.4-2% overall, 3-5 % in over 65s, 10% of over 80s
- Commonest medical reason for admission
- Annual mortality of 60% over 80s
- 10% also have AF
- Progressive condition - median survival 5 years after diagnosis

REMEMBER LEFT VENTRICULAR FAILURE IS A TRUE LIFE THREATENING EMERGENCY.



Main Causes:

1. Ischemic heart disease (35-40%)
2. Dilated cardiomyopathy (30-34%)
3. Hypertension (15-20%)

Other:

- **Undilated cardiomyopathy:** Hypertrophic, restrictive (amyloidosis, sarcoidosis)
- **Valvular heart disease**
- **Congenital heart disease (ASD, VSD)**
- **Alcohol and drugs** (chemotherapy, trastuzumab, imatinib)
- **Hyperdynamic circulation** (Anemia, thyrotoxicosis, hemochromatosis, Paget's)
- **Right heart failure** (RV infarct, pulmonary htn, pulmonary embolism, COPD)
- **Arrhythmias: Atrial Fibrillation (most common arrhythmia to cause HF),** bradycardia (Complete heart block, the sick sinus syndrome)
- **Pericardial disease** (constrictive pericarditis, pericardial effusion)
- Infections

> ***Note(s):***
>
> *Heart Failure is not a disease by itself, when a patient presents with HF it is the result of an underlying cause(disease), and it has to be established in all patients!*

Classification:

- ***Systolic & Diastolic:*** there's no much known treatment for diastolic HF yet, just treat the underlying disease.
  - **LV systolic dysfunction:** commonly caused by ischemic heart disease, but can also occur with valvular heart disease and HTN.
  - **RV systolic dysfunction**: secondary to LVSD but can occur with pulmonary hypertension, RV infarction, Mitral stenosis, arrhythmogenic RV cardiomyopathy, adult congenital heart disease.
    Usually presents with: LL edema, ascites, hepatic congestion and cardiac cirrhosis (on the long run).
  - **Diastolic failure:** symptoms and signs of HF with preserved ejection fraction above 45-50%, and abnormal LV relaxation assessed by

echocardiography. It is more commonly in elderly hypertensive patients but may occur with primary cardiomyopathies.

- ***High Output Failure/ Low Output Failure:***

  High output causes: Pregnancy, anemia, thyrotoxicosis, A/V fistula, Beriberi, Paget's disease.

- ***Chronic (most common) / Acute:***

  Acute causes: large MI, aortic valve dysfunction, hypertensive emergency.

Symptoms and signs of HF:

Symptoms:

- Exertional dyspnea, Orthopnea, Paroxysmal nocturnal dyspnea.
- **Low cardiac output symptoms:** Fatigue and tiered.
- **Abdominal symptoms:** *Anorexia, nausea, abdominal fullness, Right hypochondrial pain* (*because of congestive liver, and edema).*

Signs:

- **Cardiomegaly** (Displaced and sustained apical impulses)
- **High diastolic BP & occasional decrease in systolic BP (decapitated BP).**

HTN because of sympathetic system activation.

- **3rd and 4th heart sounds:**

  Third heart sound: **one of the most common finding in HF** (like a horse running) *low pitched sound that is heard during rapid filling of ventricle.*

  Mechanism of S_3:

  - *sudden deceleration of blood as elastic limits of the ventricles are reached*
  - *Vibration of the ventricular wall by blood filling.*
  - *Common in children*

  Fourth heart Sound (S_4) stiff V heart.

  Mechanism: *It occurs just after atrial contraction and immediately before the systolic S_1 and is caused by the atria contracting forcefully in an effort to overcome an abnormally stiff or hypertrophic ventricle.* (Wikipedia)

  - Usually at the end of diastole.
  - Exact mechanism is not known.
  - Could be due to contraction of atrium against stiff ventricle.

  The only patient we can't hear S_4 in is ***Atrial Fibrillation*** patient.

- **Elevated JVP.**

- **Tachycardia**
- **Bi-basal crackles.** (due to edema)
- **Pleural effusion.**
- **Peripheral ankle edema.**
- **Ascites.**
- **Tender hepatomegaly.**

NYHA CLASSIFICATION OF HF:

Class I: No limitation of activities; no symptoms with ordinary activities.
Class II: Mild limitation of activity; comfortable with rest or mild exertion.
Class III: Marked limitation of activities; comfortable at rest only.
Class IV: Symptoms of HF occur at rest, any physical activity brings discomfort.

Diagnosis of HF:

The diagnosis of HF should not be based on history and clinical findings; it requires evidence of cardiac dysfunction with appropriate investigations using objective measures of LV structure and function (Echocardiography).

European Society of HF guidelines:

Essential features:

1- Symptoms and signs of HF
2- Objective evidence of cardiac dysfunction (at rest, echo)

Non-essential features:

3- Response to treatment (in cases where the diagnosis is in doubt)

Framingham Criteria for Dx of Heart Failure:

Major Criteria	Minor Criteria
Cardiomegaly: if there's so no structural changes that means the reason is outside the heart e.g. Renal failure	Lower Limb edema
JVD	Night cough
Rales (crackles)	Dyspnea on exertion
S_3 Gallop	Hepatomegaly
PND	Pleural effusion
Acute Pulmonary Edema	Tachycardia 120 bpm
Positive hepatic Jugular reflex if you press on the abdomen or the liver you will find that the JV will go up and stay up, normally JV will go back to its normal position after few sec.	Weight loss 4.5 kg over 5 days management
↑ venous pressure > 16 cm H_2O Normal 5	

Differential diagnosis:

- Pericardial diseases.
- Liver diseases.
- Nephrotic syndrome.
- Protein losing enteropathy. not common

Laboratory Findings:

- Anemia
- Hyperthyroid
- Chronic renal insuffiency, electrolytes abnormality
- Pre-renal azotemia.
- Hemochromatosis one of the cause of amylydosis.

Electrocardiogram:

- Old MI or recent MI
- Arrhythmia AFB most common, once patient with HF develop AF they become unstable.
- Some forms of Cardiomyopathy are tachycardia related
- LBBB left bundle branch block (wide QRS)→*may help in management*

Chest X-ray:

- Cardiomegaly.
- Size and shape of heart.
- Evidence of pulmonary venous congestion (white lung) (dilated or upper lobe veins → perivascular edema).
- Pleural effusion.

Echocardiogram:

- Function of both ventricles
- Wall motion abnormality that may signify CAD
- Valvular abnormality
- Intra-cardiac shunts
- **CAD cardio angiogram** – we don't do it acutely- contras will do fluid over load, may cause HF/Renal Failure.

Cardiac Catheterization:

- When CAD or valvular is suspected.
- If heart transplant is indicated.

HF suspected because of signs & symptoms

↓

Assess presence of cardiac disease by ECG, CXR, and natriuretic

↓

Normal: unlikely HF

Abnormal: Imaging by echocardiography

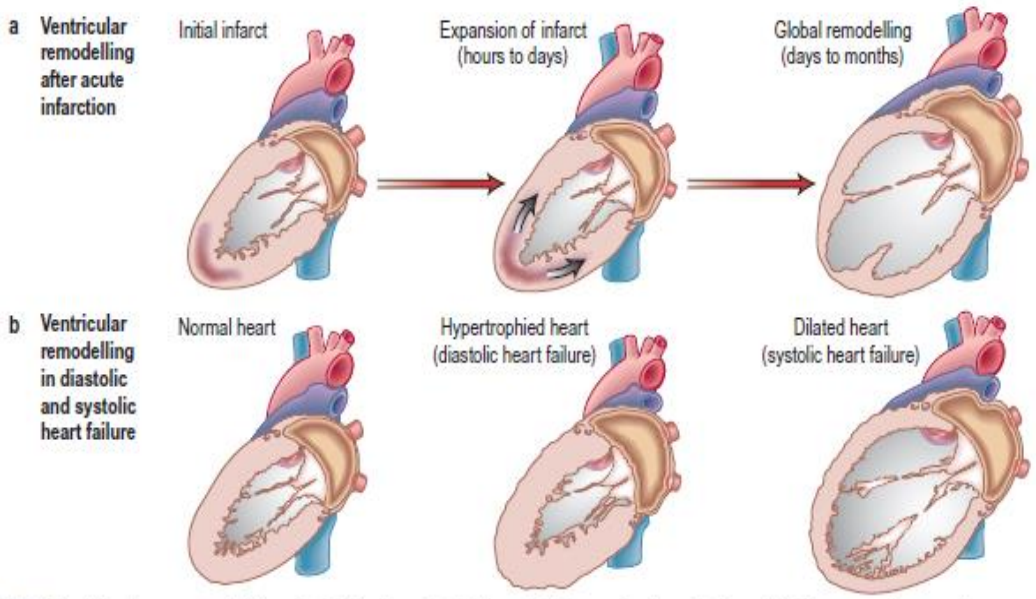
Normal: unlikely HF

↓

Annormal: Assess etiology, degree, precipitating factors, type of cardiac dysfunction

↓

Choose Treatment

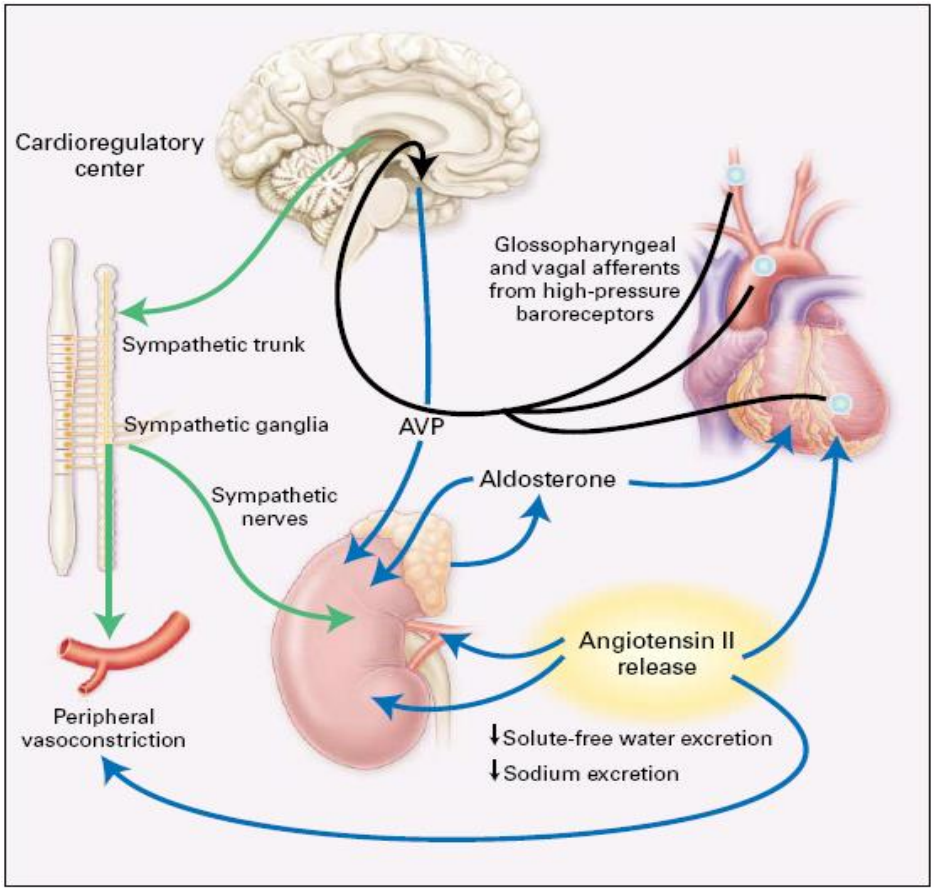


14.52 **Ventricular remodelling** in (a) ischaemic (MI) and (b) non-ischaemic heart failure, e.g. in cardiomyopathy.

Investigations to consider in all patients
Transthoracic echocardiography is recommended to evaluate cardiac structure and function, including diastolic function (Section 4.1.2), and to measure LVEF to make the diagnosis of HF, assist in planning and monitoring of treatment, and to obtain prognostic information.
A 12-lead ECG is recommended to determine heart rhythm, heart rate, QRS morphology, and QRS duration, and to detect other relevant abnormalities (*Table 5*). This information also assists in planning treatment and is of prognostic importance. A completely normal ECG makes systolic HF unlikely.
Measurement of blood chemistry (including sodium, potassium, calcium, urea/blood urea nitrogen, creatinine/estimated glomerular filtration rate, liver enzymes and bilirubin, ferritin/TIBC) and thyroid function is recommended to: (i) Evaluate patient suitability for diuretic, renin–angiotensin–aldosterone antagonist, and anticoagulant therapy (and monitor treatment) (ii) Detect reversible/treatable causes of HF (e.g. hypocalcaemia, thyroid dysfunction) and co-morbidities (e.g. iron deficiency) (iii) Obtain prognostic information.
A complete blood count is recommended to: (i) Detect anaemia, which may be an alternative cause of the patient's symptoms and signs and may cause worsening of HF (ii) Obtain prognostic information.
Measurement of natriuretic peptide (BNP, NT-proBNP, or MR-proANP) should be considered to: (i) Exclude alternative causes of dyspnoea (if the level is below the exclusion cut-point–*see Figure 1*–HF is very unlikely) (ii) Obtain prognostic information.
A chest radiograph (X-ray) should be considered to detect/exclude certain types of lung disease, e.g. cancer (does not exclude asthma/COPD). It may also identify pulmonary congestion/oedema and is more useful in patients with suspected HF in the acute setting.

Investigations to consider in selected patients
CMR imaging is recommended to evaluate cardiac structure and function, to measure LVEF, and to characterize cardiac tissue, especially in subjects with inadequate echocardiographic images or where the echocardiographic findings are inconclusive or incomplete (but taking account of cautions/contraindications to CMR).
Coronary angiography is recommended in patients with angina pectoris, who are considered suitable for coronary revascularization, to evaluate the coronary anatomy.
Myocardial perfusion/ischaemia imaging (echocardiography, CMR, SPECT, or PET) should be considered in patients thought to have CAD, and who are considered suitable for coronary revascularization, to determine whether there is reversible myocardial ischaemia and viable myocardium.
Left and right heart catheterization is recommended in patients being evaluated for heart transplantation or mechanical circulatory support, to evaluate right and left heart function and pulmonary arterial resistance.
Exercise testing should be considered: (i) To detect reversible myocardial ischaemia (ii) As part of the evaluation of patients for heart transplantation and mechanical circulatory support (iii) To aid in the prescription of exercise training (iv) To obtain prognostic information.

Pathophysiology of HF:



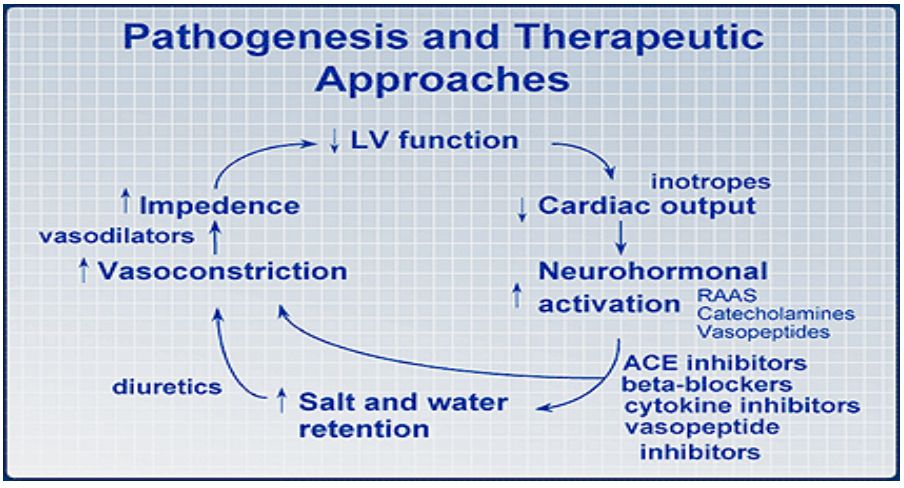
This stimulates a variety of interdependent compensatory responses involving the **cardiovascular system**, **neurohormonal systems**, and **alterations in renal physiology.**

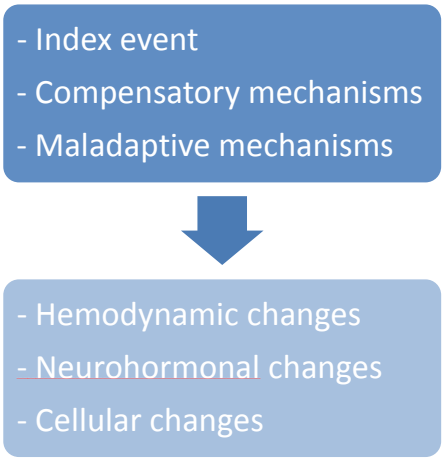
Cardiac Output = Stroke volume x Heart rate

Stroke Volume = The Amount of blood coming from the heart in a single beat.

Stroke volume → ↑ Heart rate ↓

In patient with acute HF, we want to improve the stroke volume not to reduce the heart rate with a patient of 100 beats/min. and we need to treat the underlying cause.





- ***Hemodynamic:*** The initial manifestations of **hemodynamic dysfunction** are a ***reduction in stroke volume*** and a ***rise in ventricular filling pressures*** under conditions of increased systemic demand for blood flow.

- ***Neurohormonal changes:***

N/H changes	Favorable effect	Unfavor. effect
↑Sympathetic activity	↑ HR,↑ contractility, vasoconst. → ↑ V return,↑ filling	Arteriolar constriction → After load →↑ workload →↑ O_2 consumption
↑ Renin-Angiotensin-Aldosterone	Salt & water retention→↑ VR	Vasoconstriction → ↑ after load
↑Vasopressin	Same effect	Same effect
↑interleukins &TNFα	May have roles in myocyte hypertrophy	Apoptosis
↑Endothelin	Vasoconstriction→↑VR	↑ After load

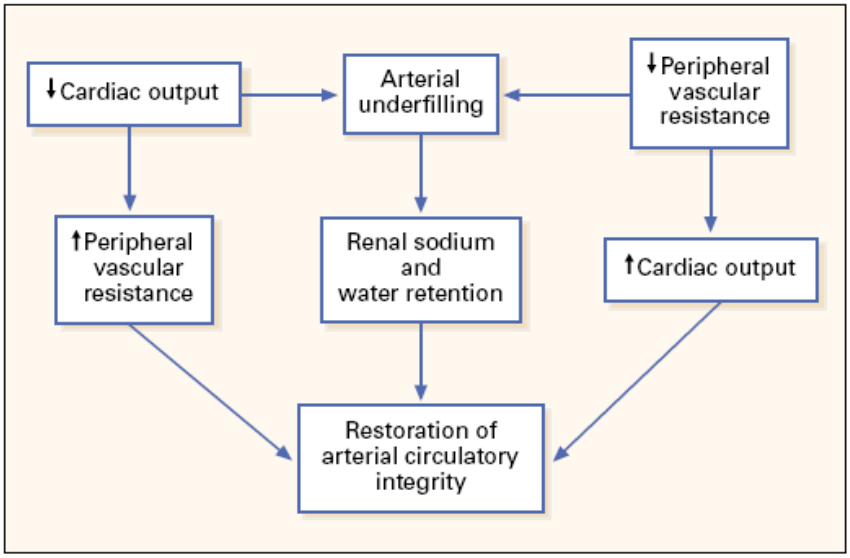
- ***Cellular changes:*** ***(Irreversible)***
  - Changes in $Ca+^{2}$ handling.
  - Changes in adrenergic receptors:
    - Slight ↑ in α_1 receptors.
    - β_1 receptors desensitization → followed by down regulation.
  - Changes in contractile proteins.
  - Program cell death (Apoptosis).
  - Increase amount of fibrous tissue.

Body-Fluid Volume:

- Renal Na and water excretion:
  - Dependent on arterial circulation
  - Cardiac output and peripheral resistance
- Decrease in circulation leads to ***arterial underfilling*** .
- Decreased effective circulating volume.
- ***Neurohormonal*** reflexes are triggered.

Arterial Underfilling:

- Causes and consequences.
- Counter-regulation.



Pathophysiological changes:

- ventricular dilatation
- Myocyte hypertrophy
- Increased collagen synthesis
- Altered myosin gene expression
- Altered sarcoplasmic ca-ATPase density
- Increased ANP secretion
- NA-water retention
- Sympathetic stimulation
- Peripheral vasoconstriction

Compensatory physiological changes to maintain cardiac output and perfusion, however these mechanisms are overwhelmed and become pathophysiological
When the heart fails, the body tries to maintain the perfusion to the vital organs by: Sympathetic nervous system activation, RAAS.

HF= Neurohormonal syndrome

Factors That May Precipitate Acute Decompensation of Chronic Heart Failure:

Discontinuation of therapy (patient noncompliance or physician initiated)25%	Myocardial ischemia or infarction
Initiation of medications that worsen heart failure (calcium antagonists, β-blockers, nonsteroidal anti-inflammatory drugs, antiarrhythmic agents)	Worsening hypertension
Iatrogenic volume overload (transfusion, fluid administration)	Worsening mitral or tricuspid regurgitation
Dietary indiscretion	Fever or infection
Pregnancy	Anemia
Exposure to high altitude	Arrhythmias

Acute Heart Failure:

Acute heart failure (AHF) occurs with the rapid onset of symptoms and signs of heart failure secondary to abnormal cardiac function, causing elevated cardiac filling pressures. This causes severe dyspnoea and fluid accumulates in the interstitium and alveolar spaces of the lung (pulmonary edema).

Etiology:

* People with ***ischemic heart disease*** present with an acute coronary syndrome or develop a complication of a myocardial infarct, e.g. papillary muscle rupture or Ventricular septal defect requiring surgical intervention.

*People with ***valvular heart disease*** also present with AHF due to valvular regurgitation in endocarditis or prosthetic valve thrombosis. A thoracic aortic dissection may produce severe aortic regurgitation.

* People with ***hypertension*** present with episodes of 'flash' pulmonary oedema despite preserved left ventricular systolic function.

*In both ***acute and chronic kidney disease*** fluid overload and a reduced renal excretion will produce pulmonary oedema.

*Atrial fibrillation is frequently associated with AHF and may require emergency cardioversion.

Diagnosis:

Initial investigations performed in the emergency room should include the following:

*__A 12-lead ECG__ for acute coronary syndromes, left ventricular hypertrophy, atrial fibrillation, valvular heart disease, left bundle branch block

*__A chest X-ray__ (cardiomegaly, pulmonary oedema, pleural effusion, non-cardiac disease)

*__Blood investigations__ (serum creatinine and electrolytes, full blood count, blood glucose, cardiac enzymes and troponin, CRP and D-dimer)

*__Plasma BNP__ or __NT-proBNP__ (BNP >100 pg/mL or NT-proBNP>300 pg/mL) indicates heart failure

*__Transthoracic echocardiography__ should be performed without delay to confirm the diagnosis of heart failure and possibly identify the cause.

SUMMARY

1. HF is a neurohormonal syndrome that happens when output doesn't meet demand
2. HF results from an underlying cause that needs to be identified.
3. The diagnosis of HF should not be based on history and clinical findings; it requires evidence of cardiac dysfunction with appropriate investegations using objective measures of LV structure and function (Echocardiography).
4. When the heart fails the body tries to maintain the perfusion to the vital organs by: Sympathetic nervous system activation, RAAS.

IMPORTANT NOTES FROM EXTERNAL RESOURCES

	Notes
Kumar&Clarks clinical medicine	**Natriuretic peptides (ANP, BNP and C-type):** ***Atrial natriuretic peptide (ANP)*** is released from atrial myocytes in response to stretch. ANP induces diuresis, natriuresis, vasodilatation and suppression of the renin-angiotensin system. Levels of circulating ANP are increased in congestive cardiac failure and correlate with functional class, prognosis and haemodynamic state. **The renal response to ANP is attenuated in heart failure**, probably secondarily to reduced renal perfusion, receptor downregulation, increased peptide breakdown, renal sympathetic activation and excessive Renin-angiotensin activity. ***Brain natriuretic peptide (BNP)*** (so called because it was first discovered in brain) is predominantly secreted by the ventricles, and has an action similar to that of ANP but greater diagnostic and prognostic value. ***C-type peptide***, which is limited to vascular endothelium and the central nervous system, has similar effects to those of ANP and BNP.

Questions

Case: a 22 yo firefighter presents with dyspnea on exertion, he reports that he has trouble keeping up with his squad and can no longer carry his 40lp pack. He reports 2 months of nocturnal cough, 10lp weight loss, and fatigue. He denies any history of smoking, and he drinks 2-3 six-packs of beer daily on weekends.

He presented 3 weeks ago to a primary clinic, he was told he has pneumonia and asthma, he was started on Beta agonists and antibiotics, but he did not feel well.

1- Which one of the following findings is the least specific in making a diagnosis?

a) Wheezing
b) S3 gallop
c) Cephalization on chest radiograph
d) Pulsusalternas
e) Elevated JVP

2- He is diagnosed with CHF, what laboratory test would have been helpful in confirming the etiology of this patient's symptoms?

a) Total bilirubin
b) C-reactive protein
c) Troponin I
d) Creatinine
e) B-type natriuretic Peptide

3- Which test would provide the most information to assess his condition?

a) Electrocardiogram
b) Arterial blood gas analysis
c) Echocardiogram
d) Chestradiograph
e) Pulmonary function tests

432 Medicine Team Leaders

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For mistakes or feedback: *medicine341@gmail.com*

Answers:

1st Question: A

2nd Question: E

3rd Questions: C