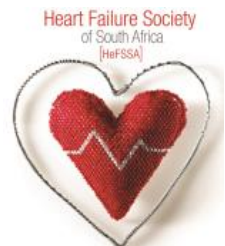


# HeFSSA Practitioners Program 2019

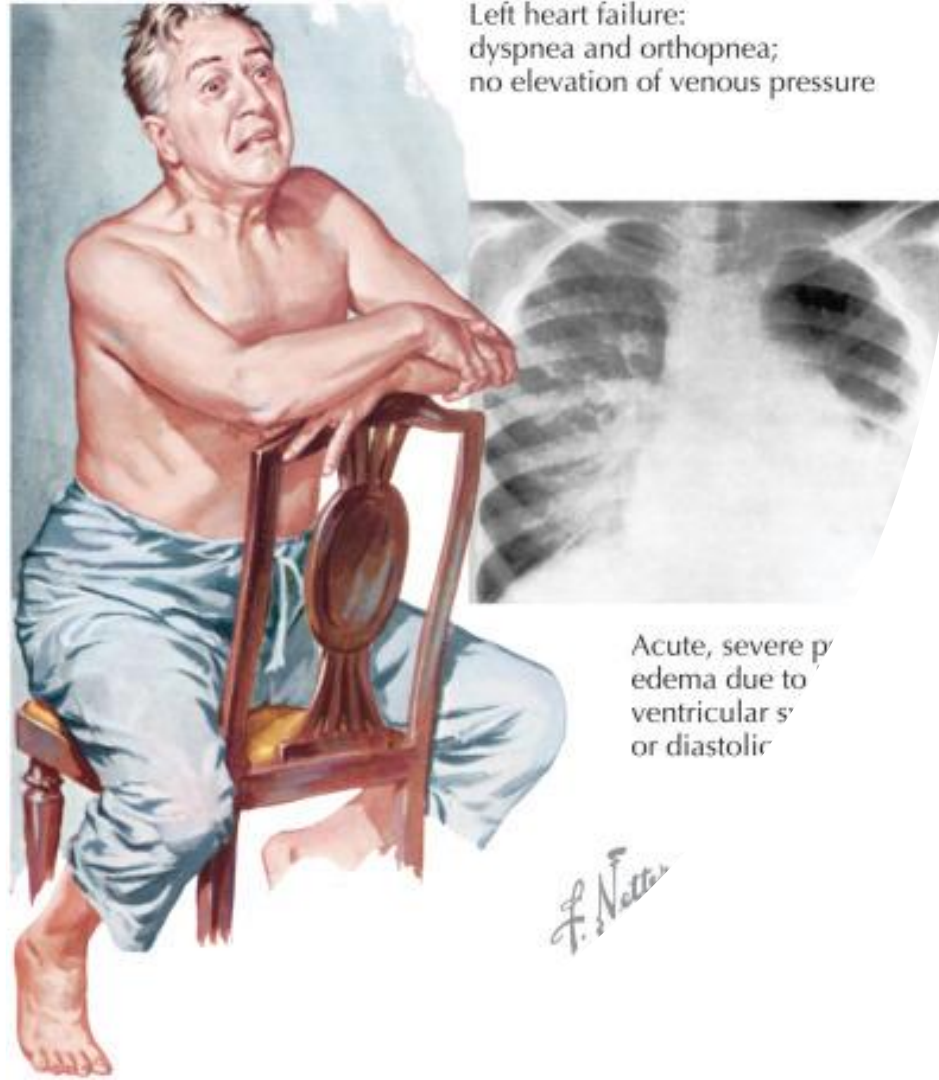
## “Challenges in Heart Failure Management”

- Dyspnoea and leg swelling, when is it heart failure?
- Management of acute decompensated heart failure
- Heart failure during pregnancy
- Refractory oedema in heart failure patient



# CASE STUDY:

## Dyspnoea and leg swelling, when is it heart failure?



# What is dyspnoea?

Baseline level of exercise/physical activity and patient perceptions must be taken into account when evaluating patients with the symptoms of “dyspnoea”

“smothering feeling/tightness/tiredness in the chest”

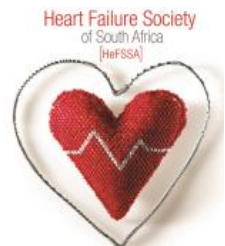
“cannot get enough air”

**Dyspnoea = Abnormally uncomfortable awareness of breathing**

“air does not go all the way down

“choking sensation”

“fatigue/tiredness during exercise



# Pathophysiology

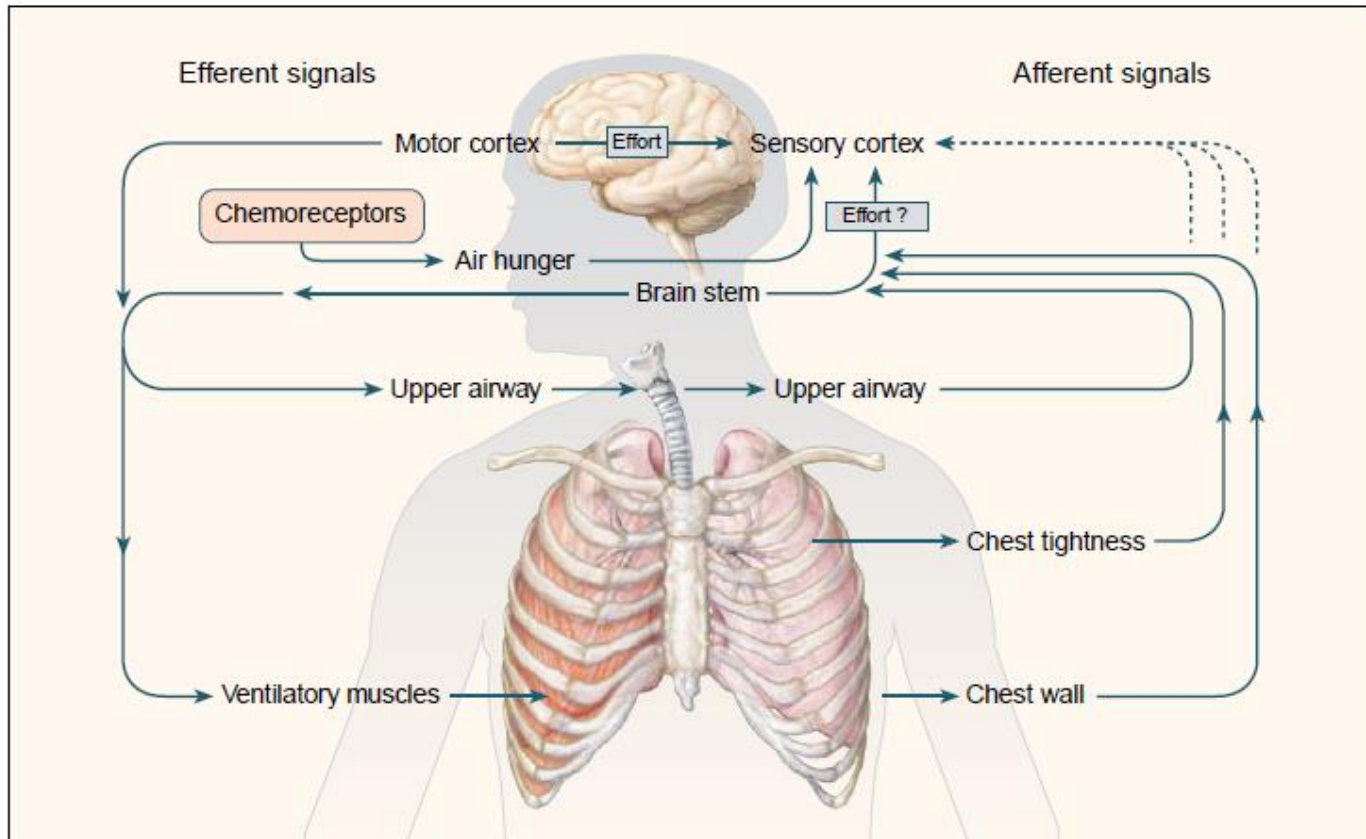
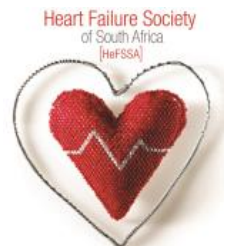


Figure 1. Efferent and Afferent Signals That Contribute to the Sensation of Dyspnea.

The sense of respiratory effort is believed to arise from a signal transmitted from the motor cortex to the sensory cortex coincidentally with the outgoing motor command to the ventilatory muscles. The arrow from the brain stem to the sensory cortex indicates that the motor output of the brain stem may also contribute to the sense of effort. The sense of air hunger is believed to arise, in part, from increased respiratory activity within the brain stem, and the sensation of chest tightness probably results from stimulation of vagal-irritant receptors. Although afferent information from airway, lung, and chest-wall receptors most likely passes through the brain stem before reaching the sensory cortex, the dashed lines indicate uncertainty about whether some afferents bypass the brain stem and project directly to the sensory cortex.

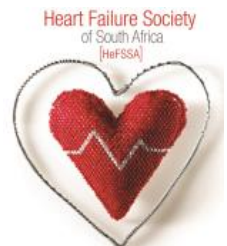
# Differential diagnosis

1. Obstructive airways disease e. g. asthma/COPD
2. Diffuse parenchymal lung disease e.g. interstitial lung disease
3. Pulmonary vascular occlusive disease e. g. thromboembolism
4. Diseases of the chest wall/weakness of respiratory muscle e. g. Guillain-Barre
- 5. Heart Disease**
6. Anemia
7. [Anxiety]



# Classification

NYHA Class	Symptoms
I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea (shortness of breath).
II	Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea (shortness of breath).
III	Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, or dyspnea.
IV	Unable to carry on any physical activity with discomfort. Symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases.



# Patterns of dyspnoea may be helpful

Sudden unexpected episodes of dyspnoea

Consider: PE, spontaneous pneumothorax

Trepopnoea [dyspnoea in the lateral decubitus position]

Consider: CCF

Parosymal nocturnal dyspnoea [sudden episodes of dyspnoea at usually at night]

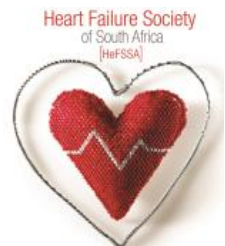
Consider: CCF, asthma [due to circadian rhythm of airway muscle tone], chronic bronchitis [hypersecretion of mucus]

Orthopnoea [dyspnoea in supine position]

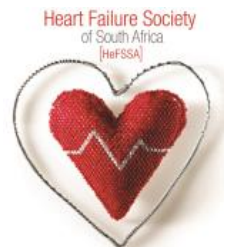
Consider: CCF but may occur in asthma/COPD and bilateral diaphragmatic paralysis [rare]

Platypnoea: dyspnoea in the upright position

Consider: positional changes in V/Q relationships usually account for this



“Cardiac” Dyspnoea usually diagnosed when underlying heart disease is diagnosed based on history and clinical examination supplemented by non-invasive testing.





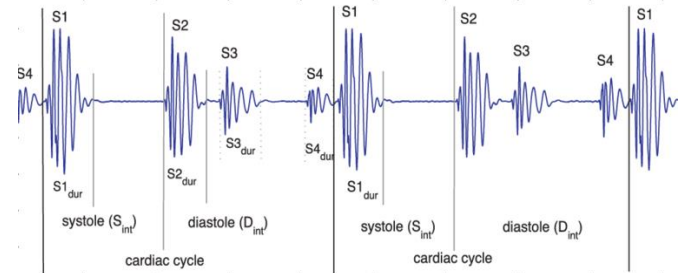
# Clinical signs suggestive of cardiac etiology

Peripheral Oedema



Previous History  
of Heart Disease

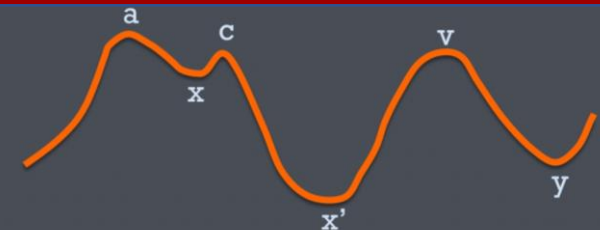
Presence of S3 or S4



LV enlargement



Elevated JVP



- a – atrial contraction
  - x – atrial relaxation
  - c – bulging of tricuspid valve with ventricular contraction
  - x' – downward movement of tricuspid valve with ventricular contraction
  - v – passive atrial filling
  - y – atrial emptying with opening of the tricuspid valve
- a-c-v waves  
x-y descents

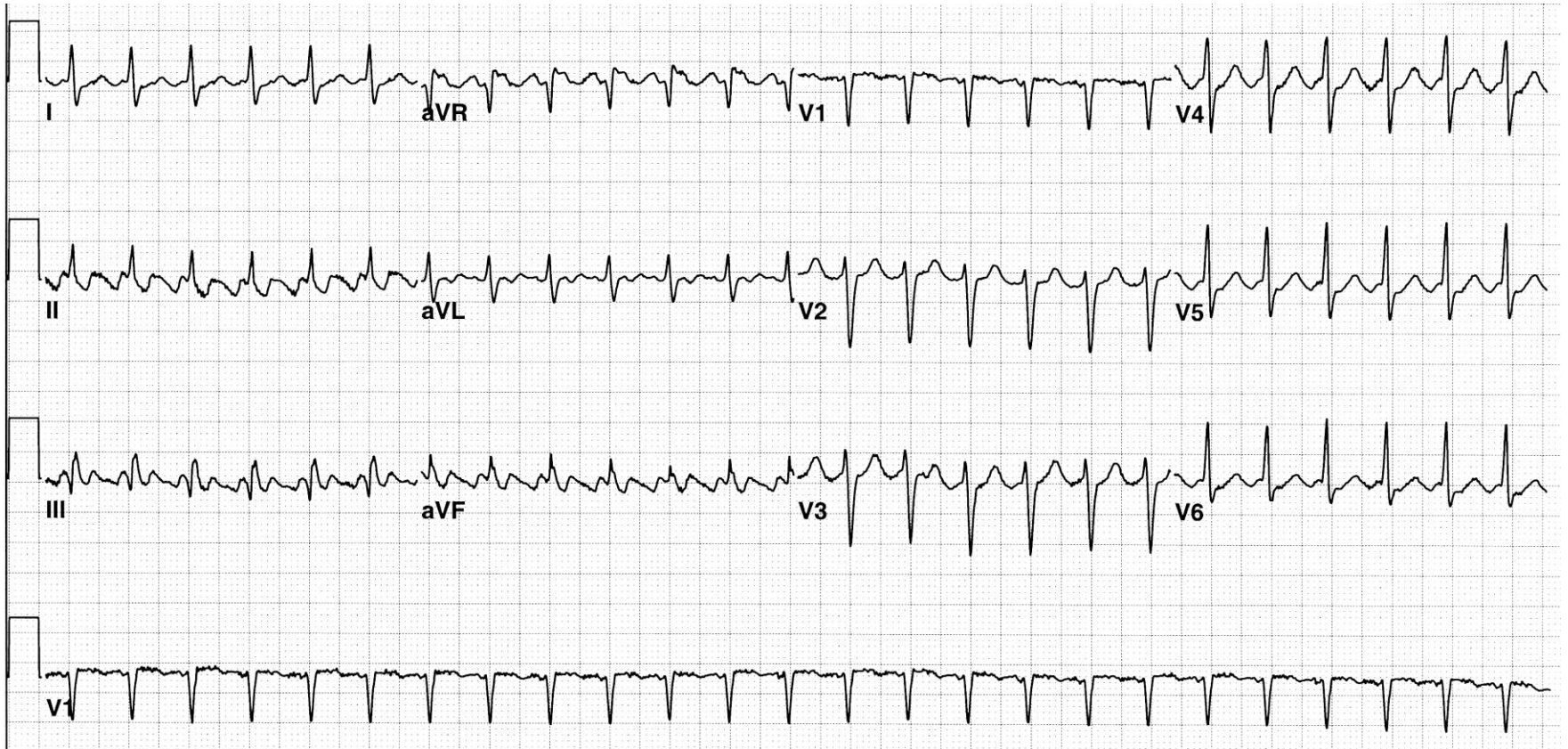
# How useful are clinical signs?

Sensitivity and specificity of clinical symptoms and signs in HF

Clinical Features	Sensitivity (%)	Specificity (%)
Breathlessness	66	52
Orthopnoea	21	81
PND	33	76
History of oedema	23	80
Tachycardia	7	99
Pulmonary crackles	13	91
Oedema on examination	10	93
3 <sup>rd</sup> heart sound	31	95
Raised JVP	10	97

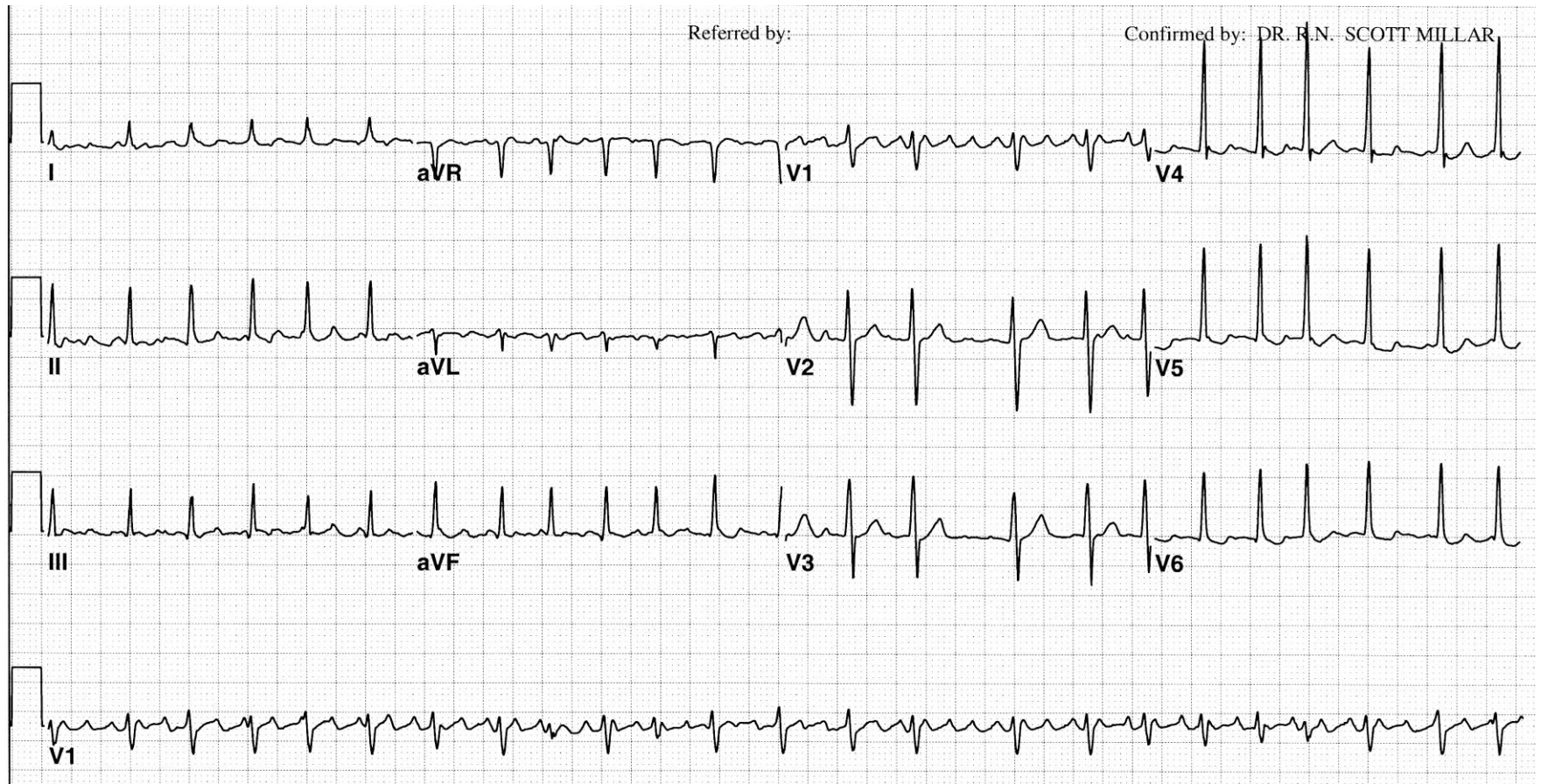
Sosin M et al, Manson Publishing 2006

# ECG – Arrhythmias – AF/Atrial Flutter



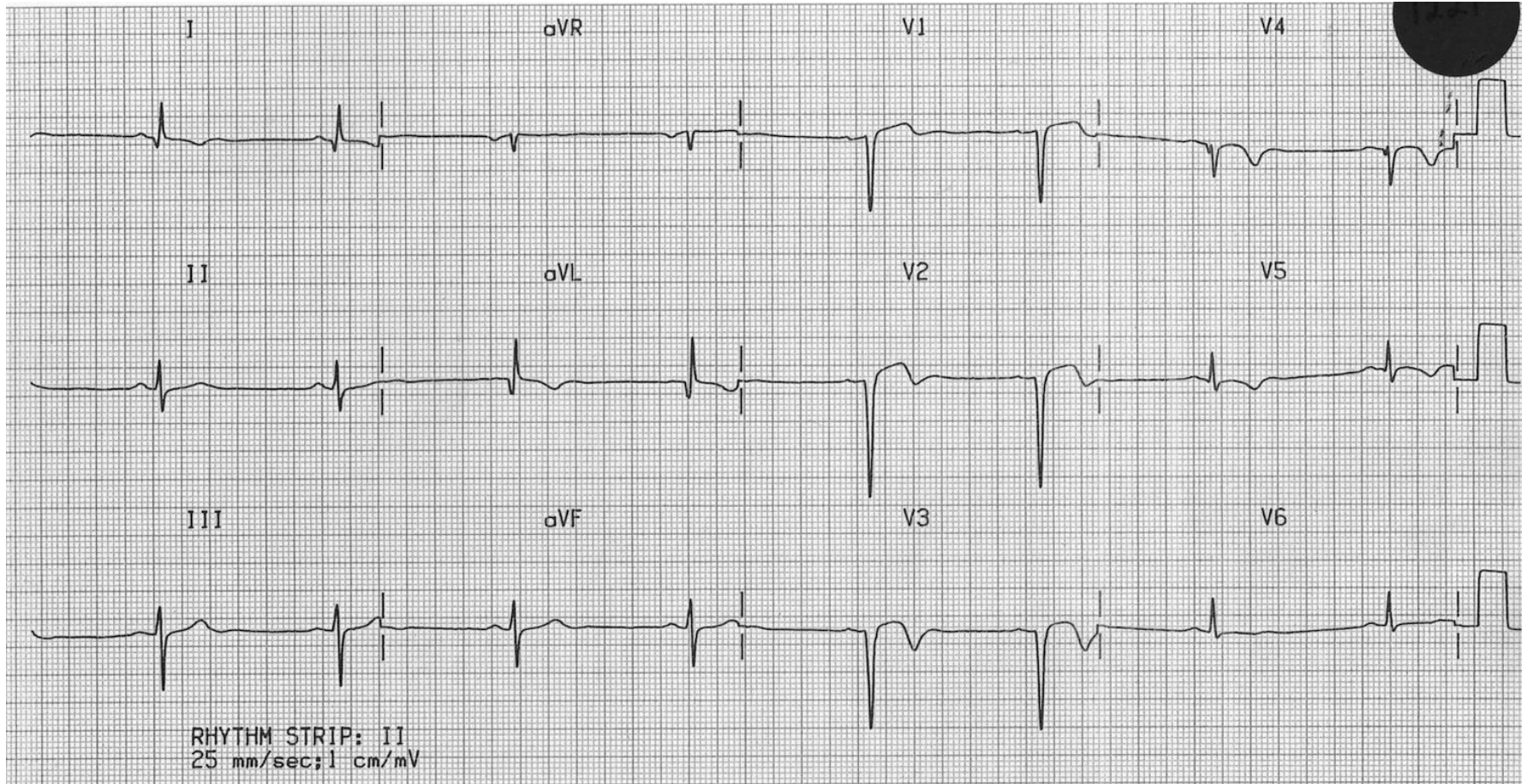


# ECG – Arrhythmias – AF/Atrial Flutter



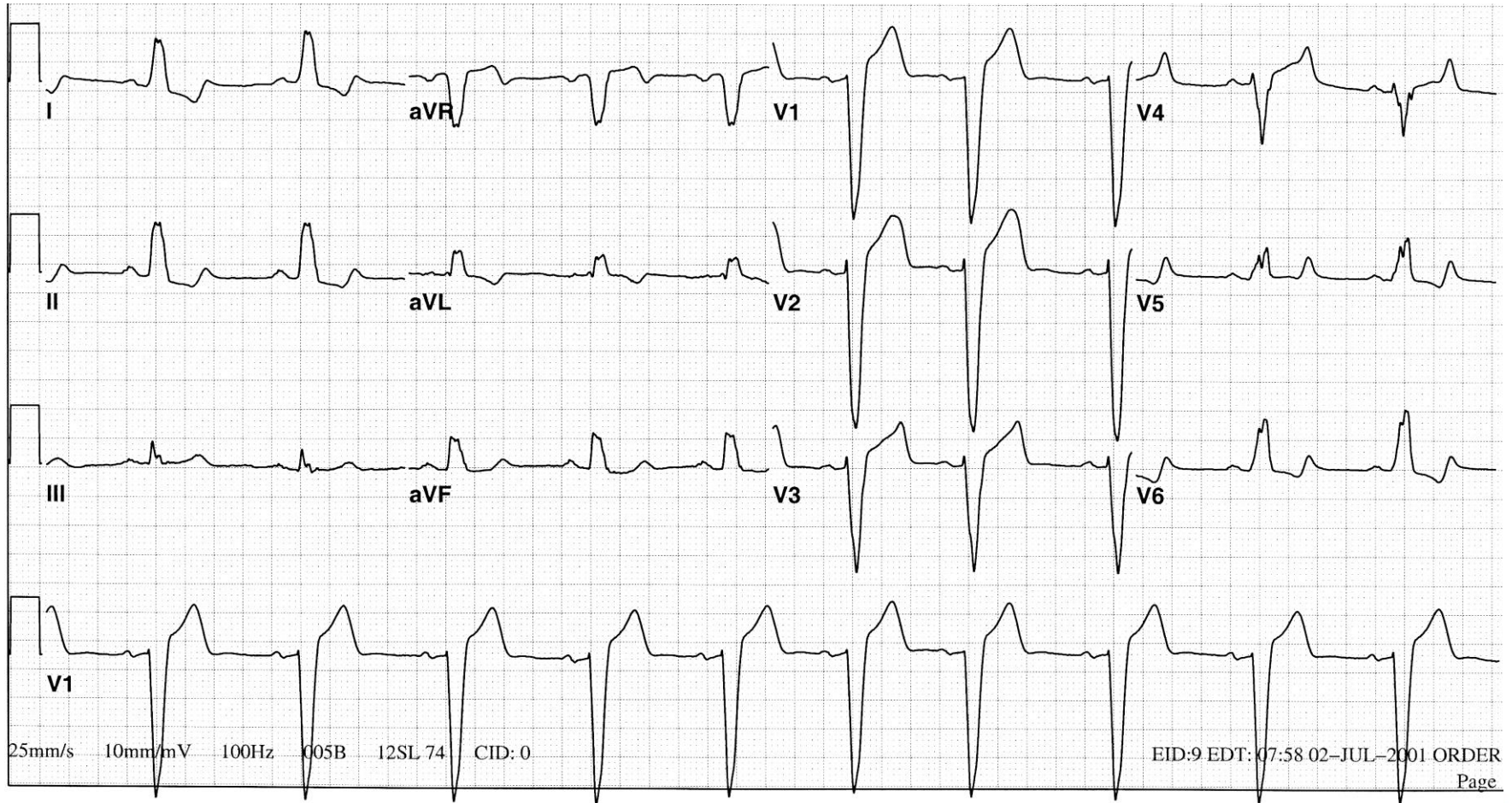


# ECG – previous infarct



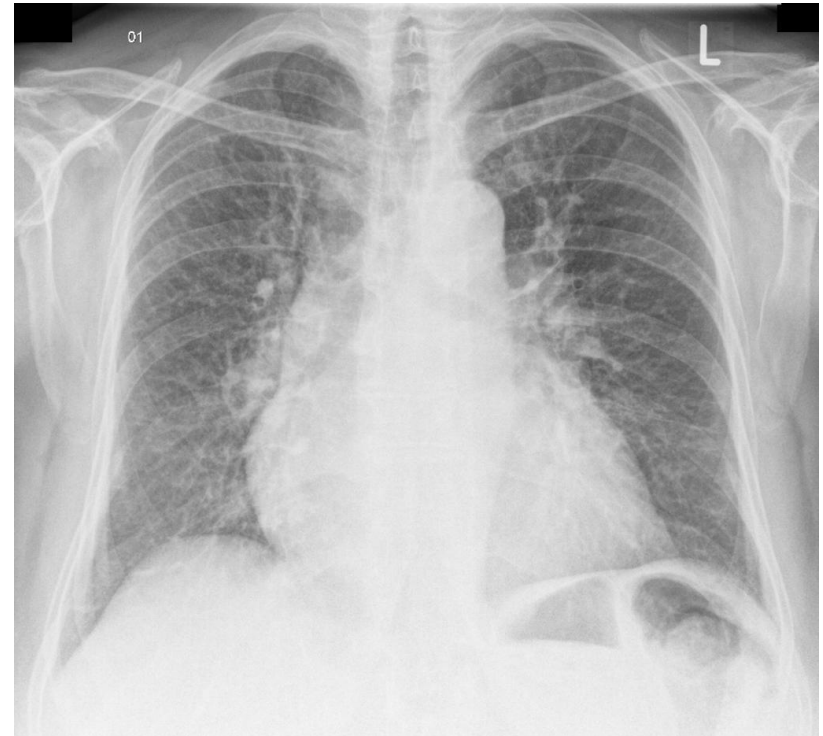
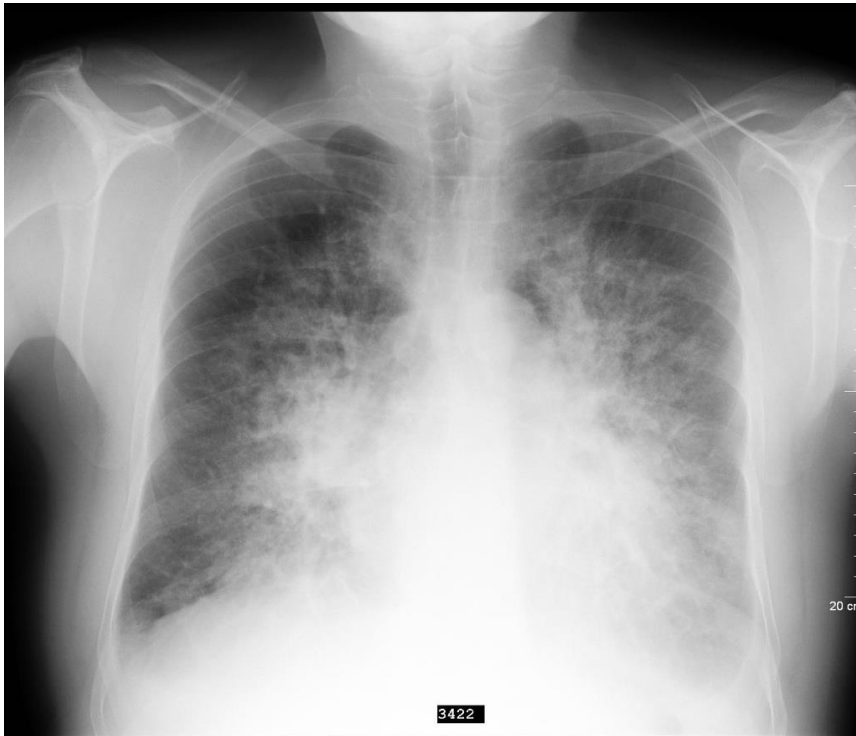


# ECG - LBBB

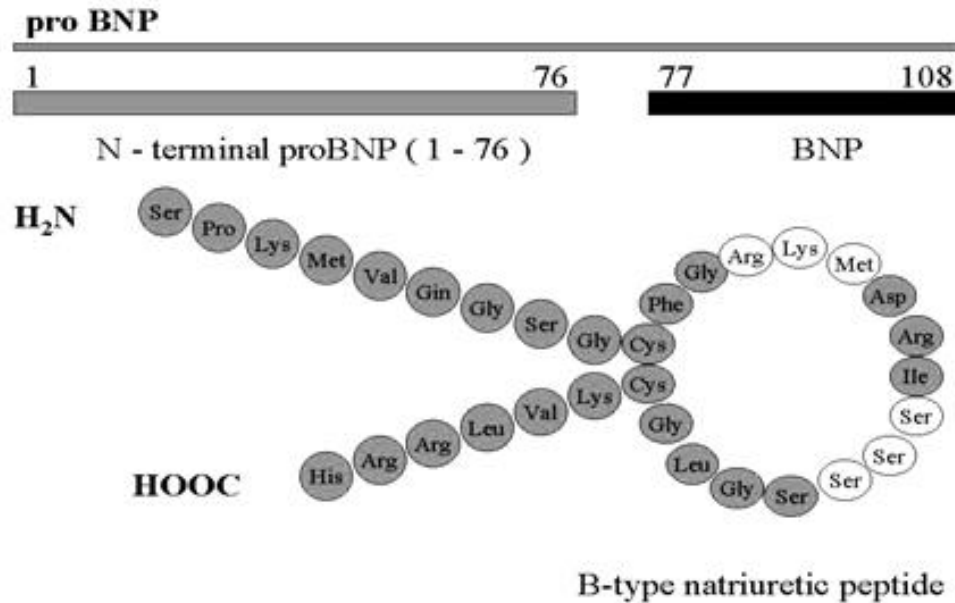


# CHEST X-RAYS:

Interstitial oedema, pulmonary vascular redistribution, accumulation of fluid in septal planes, pleural effusion, cardiomegaly



# Natriuretic Peptides



NT-proBNP <125 pg/mL has a **negative predictive value** of 0.94 – 0.98 (i. e. if the NT-proBNP is this low >95% of the time the patient does NOT have heart failure)

This cut-off is also valid to exclude HFpEF.

It is therefore useful to **exclude** heart failure as a cause of dyspnea.



# Triggers of elevated BNP

## Cardiac disease

- Heart failure with reduced ejection fraction
- Heart failure with preserved ejection fraction
- RV failure
- Valvular heart disease
- Myocarditis
- LVH
- Coronary artery disease
- Myocardial trauma
- Arrhythmias
- Pericardial disease

Obese patients may have relatively lower BNP levels compared to non-obese individuals

## Pulmonary disease

- Acute PE
- Pulmonary hypertension
- OSA
- Infection
- COAD

## Neurologic disorders

- Stroke

## Critical illness

- Sepsis
- Burns
- Transfusion associated circulatory overload

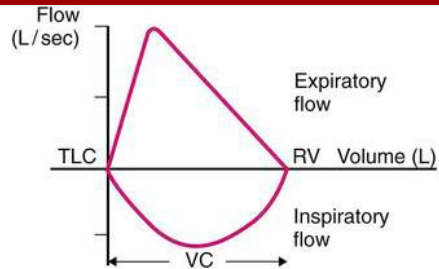
## Toxins

- Chemotherapy
- Snake bites

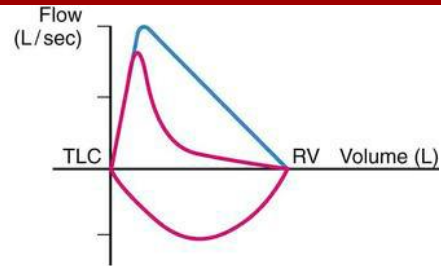
## Other

- Renal insufficiency
- Anaemia
- Cirrhosis
- Hypertension
- Hyperaldosteronism

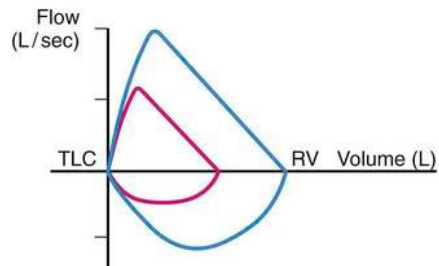
# Lung function Tests



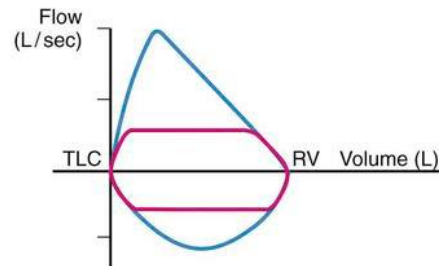
A. Normal



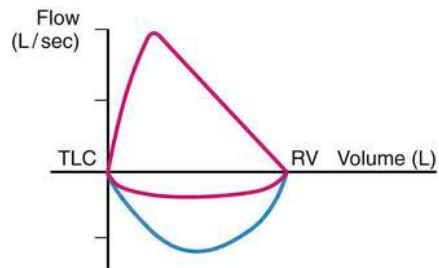
B. Emphysema



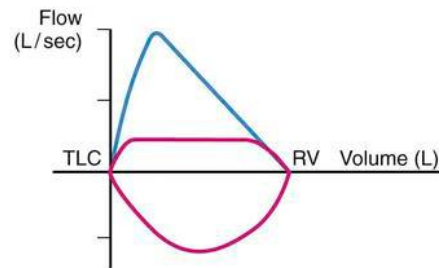
C. Unilateral main-stem bronchial obstruction



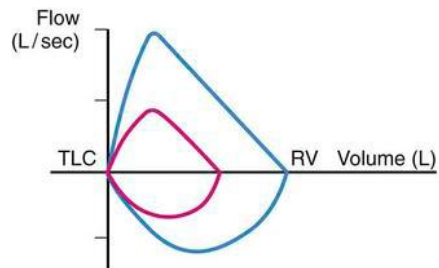
D. Fixed UAO



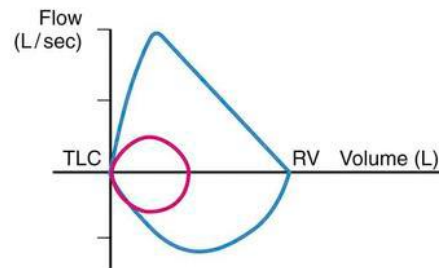
E. Variable extrathoracic UAO



F. Variable intrathoracic UAO

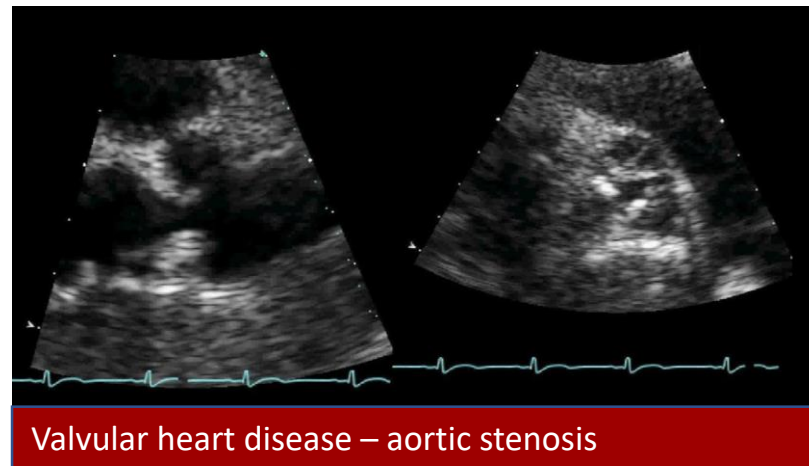
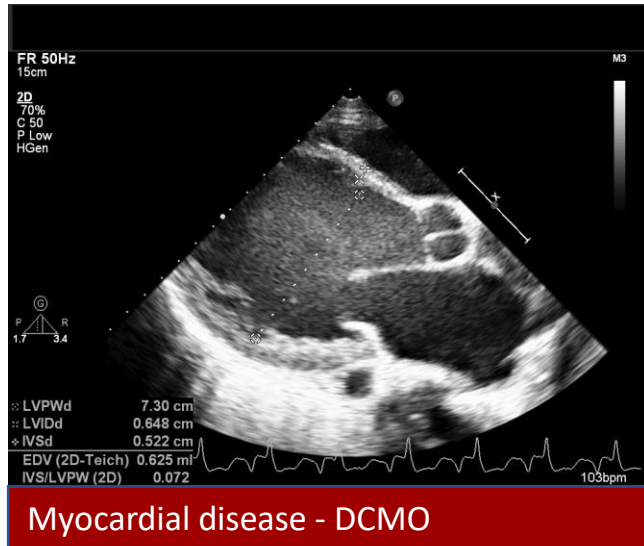


G. Restrictive parenchymal lung disease

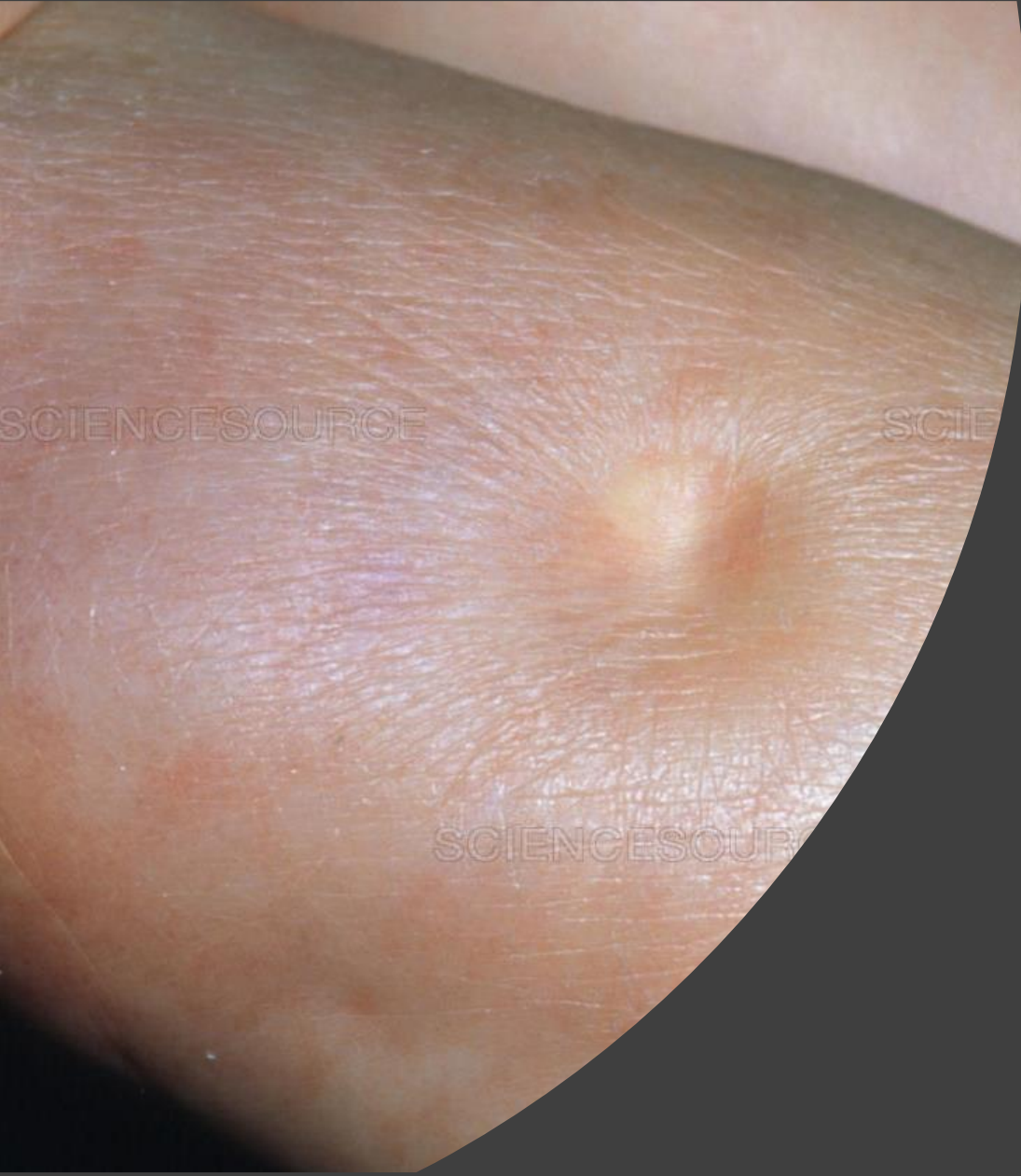


H. Neuromuscular weakness

# Echocardiogram



# Leg swelling



# Oedema

Oedema=clinically apparent increase in interstitial fluid volume – usually manifests with swelling of the extremities

Anasarca: gross, generalised oedema

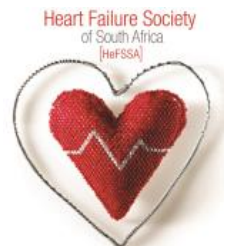
Ascites: accumulation of excess fluid in the peritoneal cavity

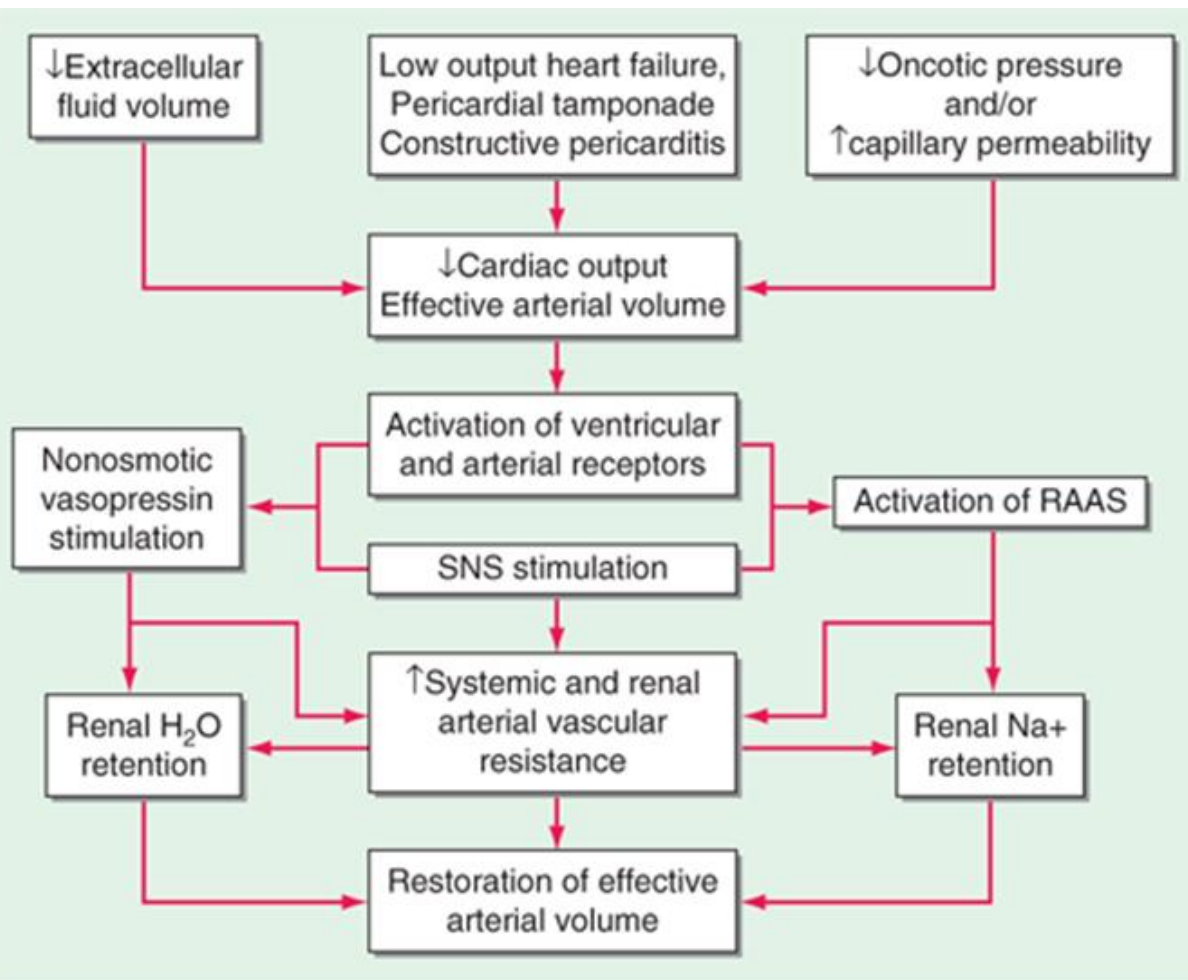
## Localised

Usually due to lymphatic or venous obstruction:  
thrombophlebitis,  
lymphangitis, lymphnode resection, tumour, DVT

## Generalised

Heart failure  
Kidney disease  
Nephrotic syndrome  
Cirrhosis  
Malnutrition  
Hypothyroidism  
Pregnancy  
Drugs

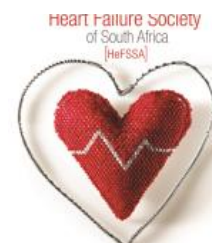




A

Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition  
[www.accessmedicine.com](http://www.accessmedicine.com)

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Organ System	History	Physical Exam	Lab Findings
Cardiac	Dyspnoea/ortho- pnoea/PND	Elevated JVP S3 Small pulse pressure	Increased Urea:Crea ratio Elevated uric acid Na often lower LFTs may be abnormal due to hepatic congestion
Hepatic	Dyspnoea infrequent Ethanol abuse	Often associated with ascites JVP normal BP usually lower Other signs of chronic liver disease (jaundice, palmar erythema, Dupuytren, spider angiomas, gynecomastia)	Reductions in serum albumin and cholesterol LFTs increased Raised INR Hypokalemia Macrocytosis
Renal	Dyspnoea less prominent Uraemic symptoms (LOA, restless legs, reduced concentration, altered taste)	Usually hypertensive Retinopathy (diabetic/hypertensive) Periorbital oedema may be prominent Pericardial friction rub	Albuminuria Hypoalbuminaemia Raised creatinine Hyperkalemia Hyperphosphatemia and hypocalcemia Normocytic anaemia



# Drugs associated with oedema

NSAIDs

Antihypertensive agents

Vasodilators

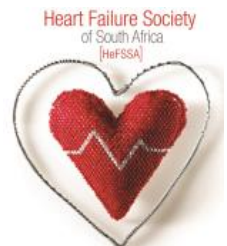
- Minoxidil
- Clonidine
- Hydralazine
- Methyldopa
- CCBs
- Alpha-adrenergic blockers

Steroids

Cyclosporine

Growth hormone

Certain immunotherapies





# What catches us out?



Anaemia

Anxiety

Liver  
cirrhosis

Pregnancy

Drugs

Patient does actually  
not have dyspnoea,  
symptoms wrongly  
interpreted

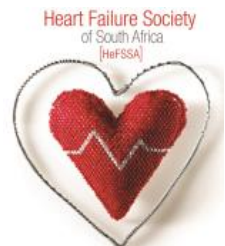
Pulmonary  
emboli

Kidney  
disease

Co-existing  
pathology – e. g.  
COAD and severe  
aortic stenosis

# Summary

1. Confirm the symptom – does this patient have dyspnoea? If so, how bad is it (NYHA classification)
2. What is the cause – recall the differential diagnosis
3. Distinguishing a cardiac cause from noncardiac causes requires integration of:
  - History and clinical signs
  - ECG
  - CXR
  - NT-proBNP
  - +/-Echo and lung function tests



THANK YOU

