

Right Ventricle In Heart Failure

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- **Cardiologists have become “LV centric” though circulation is a closed system and the RV plays an integral part in it.**

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- Many a time the right ventricle (RV) is regarded as the “younger brother” of the left ventricle (LV) and is treated as a less important member of the contractile apparatus. This view stemmed from the concept that the RV functions is not great as it pumps blood only to the lungs.
- However, the circulatory system is a closed one and both ventricles are interdependent, working together in an orchestrated complex pattern in health and disease.
- The failure of one ventricle deleteriously affects the performance of the other



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- The RV contracts in three ways: the inward motion of the RV free wall, shortening of longitudinal fibres pulling the apex towards the base of the heart, and traction by LV contraction .the LV traction component contributes about 20-40% of RV cardiac output .

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- **The RV ejects the same stroke volume as the LV but against a much lower resistance of the pulmonary vasculature. This results in an RV stroke work which is almost one fourth that of the LV,**



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- **There is important ventricular interdependence between the RV and the LV. The components of this are the sharing the interventricular septum, the encircling fibres and the pericardium.**

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- **Acute dilatation of the RV, for example in RV infarction or significant pulmonary embolism, shifts the septum to the left. This shift impairs LV diastolic filling as well as its contractility. The constraining effect of the pericardial sac comes into play in diastole when a dilated ventricle restricts the filling of the other.**

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- **Some of the mechanisms as to why RHF follows LHF are: 1) they may both be affected by the same pathology whether it is ischaemia, cardiomyopathy or myocarditis; 2) development of pulmonary hypertension in (LVF); 3) severe LVF may result in decreased coronary perfusion for the RV; 4) LV dilatation can impair RV diastolic function by increasing pericardial constraint.**

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- Nowadays, with the increasing use of ACE inhibitors and BB, more frequently patients with LVF survive to develop pulmonary hypertension and RVF. This is the reason why RV failure is considered “ the common final pathway” it has been shown to be the most important indicator of poor prognosis in HF.

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Prevalence

- RVF as the primary presentation of acute decompensated HF and hospitalisation accounted for 2.2% of HF admissions in the CHARITEM registry ; however, it was present as secondary to acute LV failure in more than one fifth of the cases.
- In our Egyptian HF registry, 4.5% of patients with acute HF presented with RHF as opposed to 3% in other ESC regions .
- This could be attributed to the even higher incidence of rheumatic heart disease.

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Causes of right heart failure

- **The causes of RHF can be divided broadly into three categories: secondary to pulmonary hypertension, RV and tricuspid valve pathology, and diseases of the pericardium.**
- **Pulmonary hypertension (PH) is the most common cause of RHF. The commonest cause of PH is left-sided heart failure. This is termed PH type 2 as it is associated with high wedge pressure**

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- **A particular type of PH results from acute pulmonary embolism, and can result in acute RVF. Recurrent showers of smaller pulmonary emboli can end in chronic thromboembolic pulmonary hypertension (CTEPH).**

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- **Less commonly, RVF could result from direct affection of myocardial disease by myocarditis, cardiomyopathy, ischaemia, or arrhythmia. RV infarction complicates 30 to 50% of inferior myocardial infarction**

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- **Gradual accumulation of pericardial effusion can compress the thin-walled RV and prevent its filling, presenting as RHF. Constrictive pericarditis is one of these diagnoses which can be easily missed. The commonest cause used to be prior tuberculosis infection, but nowadays it is mostly secondary to chest radiotherapy or previous cardiac surgery.**

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Clinical diagnosis

- Symptoms of RVF are mainly due to systemic venous congestion and/or low cardiac output. This includes exertional dyspnoea, fatigue, dizziness, ankle swelling, epigastric fullness and right upper abdominal discomfort or pain.



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- Signs: raised jugular venous pulse (JVP), right ventricular gallop, usually a pansystolic murmur over the tricuspid area which increases with inspiration, also, an enlarged tender liver, ascites ankle oedema.

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- **to highlight the importance of raised JVP as a clinical sign. It is a specific sign of RVF . Raised JVP is a prognostic marker. Analysis of the SOLVD study has shown that it correlates with mortality and a risk of heart failure.**
- **RV infarction should be suspected in the context of inferior MI by the triad of raised JVP, hypotension and clear lung fields.**

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Technical clues to diagnosis

- **Manifest HF is not difficult to diagnose if careful attention is paid to clinical signs. However, the underlying aetiology can sometimes be elusive. On the one hand, if there is a long history of cardiomyopathy or chronic obstructive airway disease, usually history plus simple investigations can easily determine the diagnosis. However, reaching the diagnoses of other less common causes such as PAH, CTEPH or constrictive pericarditis can be a challenge.**

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- **Electrocardiography in patients with pulmonary hypertension shows signs of RV hypertrophy**
- **Echocardiography can give a rapid estimate of the RV size, shape and shift of the IVS.**
- **Because of the RV geometry and the complex 3D shape, measurement of RV function is a challenge.**

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- **Estimation of pulmonary hypertension is an integral part of evaluation of a patient with suspected RVF.**
- **Special attention should be paid to IVC diameter and distensibility in relation to respiration**
- **Examination of the PA and the flow across it gives a further clue for PH.**

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- **Contrast echo is useful in the detection of intracardiac shunts.**
- **A lung function test is needed to confirm the presence and severity of obstructive airway disease.**
- **High-resolution CT of the chest is helpful when underlying lung fibrosis is a possible diagnosis.**
- **Cardiac MRI is the gold standard nowadays for measurement of RV volumes and function.**
- **CT pulmonary angiography is essential if CTEPH is suspected.**

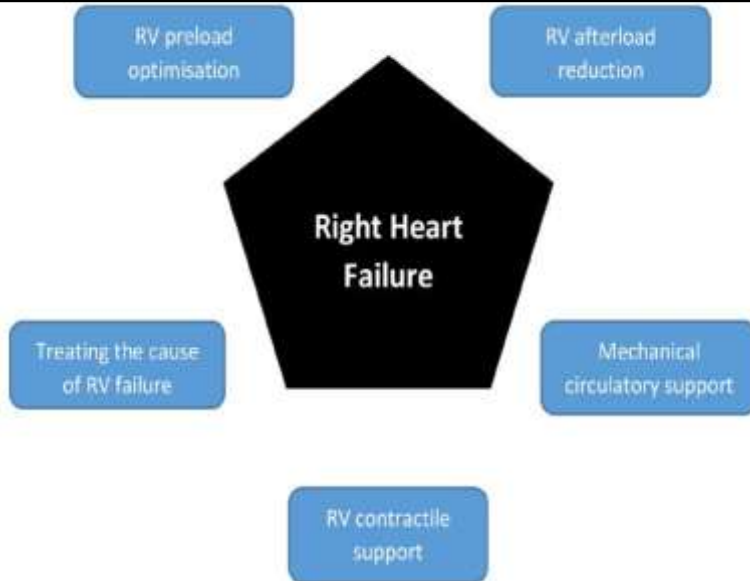
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Biochemical markers

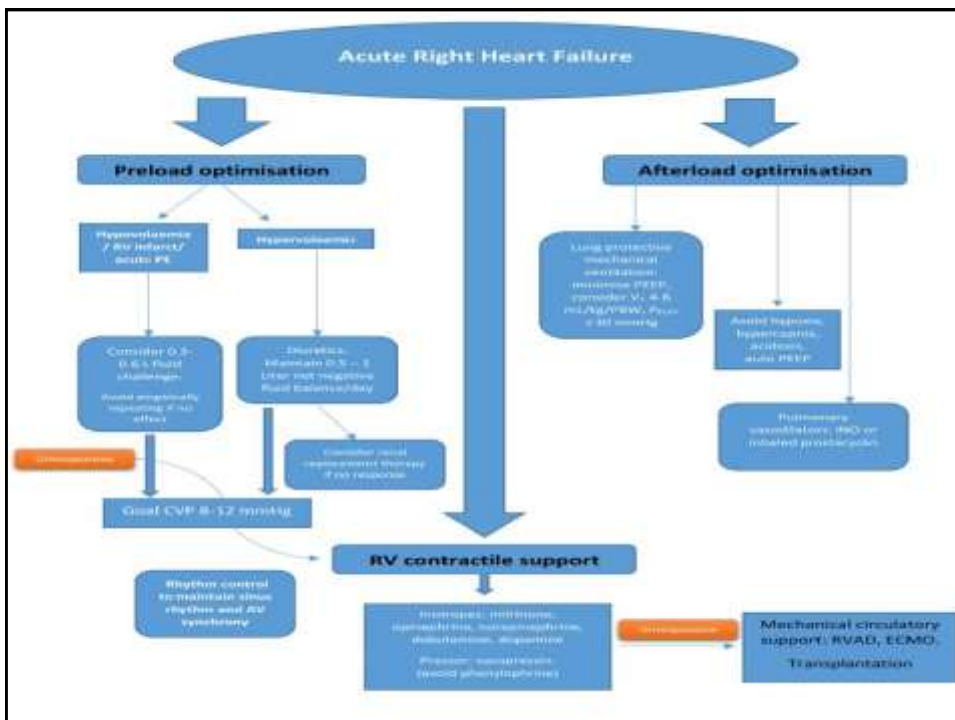
- **Systemic venous congestion affects the liver and kidney and results in derangement of their function.**
- **There are no specific biomarkers for RVF, but raised BNP and troponins reflect stress and injury . Their rise reflects the severity and poor prognosis.**

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Treatment of right heart failure: is there a solution to the problem?



Principles of managing right heart failure.



Optimisation of RV preload, afterload and contractility

(a) Preload optimisation

- **Critically ill patients may have reduced right heart preload due to volume loss, reduced venous tone from medications, sepsis .**
- **In conditions where RV output is impaired due to contractile dysfunction but the afterload is normal (as can be seen in acute RV infarction), a higher preload is needed to maintain forward flow.**

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- **However, the majority of conditions leading to RHF are characterised by high RV afterload. In these scenarios, reducing excessive RV preload with diuretics or haemofiltration is key to reducing RV dilatation and free wall tension,**

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(b) Afterload reduction

- **Strategies for optimising RV afterload can be divided into general measures and pharmacotherapy, including pulmonary vasodilators. General measures are aimed at correcting conditions that can increase pulmonary vascular resistance as hypoxia , and hypercapnia.**

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- **Pulmonary vasodilators include several classes of drug that are approved for use in pulmonary arterial hypertension (PAH) . However, vasodilators have the potential to cause systemic hypotension.**

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- **inhaled nitric oxide is the pulmonary vasodilator of choice in the critically ill and has been shown to improve pulmonary haemodynamics in RHF**

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- **Inhaled prostacyclin analogues have been shown to be safe and effective for cardiothoracic surgical patients with pulmonary hypertension, refractory hypoxaemia, or right heart dysfunction,**

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- **Phosphodiesterase 5 (PDE5) inhibitors** They decrease pulmonary arterial pressure and increase cardiac output in both acute and chronic pulmonary hypertension.



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(c) Improving contractility

- Strategies to improve RV contractility can also be divided into general and pharmacologic measures. General measures target the avoidance of overstressing the RV free wall by optimising preload and afterload
- Maintenance of sinus rhythm is important in RHF

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- **Concerning inotropes, low-dose dobutamine (5-10 $\mu\text{g}/\text{kg}/\text{min}$)**
- **Dopamine would be preferable for hypotensive, non-tachycardia patients.**

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- **Milrinone, a selective PDE3 inhibitor, It is an attractive agent in pulmonary hypertension resulting from biventricular failure**
- **Both epinephrine and norepinephrine would be preferable in hypotensive patients.**

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- **Mechanical circulatory support devices and treating the underlying cause of right heart failure**
- **In cases refractory to medical therapy, mechanical circulatory support may offer a bridge to recovery or to the definitive management of the underlying cause.**
- **Percutaneous support options include extracorporeal membrane oxygenation (ECMO), which offers right- and left-sided circulatory support.**

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Conclusion



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- **Even though RVF does not take centre stage in the field of heart failure research and clinical trials, it is actually the final pathway of LVF.**
- **Pulmonary hypertension is the commonest cause of RVF.**
- **Due to the unusual anatomy of RV, assessment of its function is a challenge. However, technical advances, especially in echocardiography and cardiac MRI, are helping to evaluate RV function and volumes as well as measurement of pulmonary artery pressure. Careful history taking, clinical examination and the targeted use of investigations can elucidate the underlying pathology.**

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- **The management of isolated acute RVF remains more of an art than a science**
- **In addition to treating the specific cause, RV preload optimisation, the use of selective pulmonary vasodilators, RV inotropic support and mechanical circulatory device therapy form integral components of a comprehensive strategy to support the failing right heart.**

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Thank you