Right heart failure is characterized by a low cardiac output, hypotension, hepatic enlargement and raised JVP.

- The appearance of a dilated right ventricle with a reduced ejection fraction, however, should prompt a reduction in preload in a patient who is not volume responsive (as defined by lack of alteration in heart rate, blood pressure, cardiac and urine output).
- The therapeutic effect can be monitored by sequential echocardiography or by using right heart catheterization and, ideally, continuous measurement. The converse is true since sequential volume challenges monitored by pulmonary artery pressure changes in the absence of reversed right ventricular interdependence will increase cardiac output, up to the individualized optimal filling point.

- No selective right heart inotrope exists.
- Augmentation of contractility can be achieved by b-1-mimetics, calcium sensitizer phosphodiesterase inhibitors. The problem is that without afterload manipulation, increasing right heart contractility and hence output, increases myocardial oxygen consumption but without a systemic benefit.
- The calcium-sensitizing, isotropic agent, levosimendan has been shown to provide a survival advantage in heart failure trials. In a pilot study of levesimendan in early ARDS, Morelli et al. demonstrated that a reduction in the pulmonary vascular resistance by levosimendan improved right ventricular function.
- Inotropes may provide a benefit in instances where septicemia related to hypoxemia is a problem.
- They elevate the mean arterial pressure, coronary artery perfusion and may, consequently, reduce myocardial work. Other vasoactive agents such as norepinephrine, phentolamine and phenoxyamine may elevate pulmonary arterial pressures and thus improve myocardial oxygenation. The benefit is lost once the right ventricle consumes more oxygen, to maintain output, in the face of the elevated afterload.

Prostaglandins
- Prostaglandins can be given by inhalation, systemically or subcutaneously.
- The vasoactive effects are mediated by nitric oxide release and interaction at a local level with the vascular endothelial smooth muscle. Prostacyclin I1 systemically undergoes significant first pass pulmonary metabolism but with lower systemic pressures and resistance, adversely altering the ventilation-perfusion matching and subsequently arterial oxygenation.
- When infused or as an aerosolized agent it is less effective than nitric oxide or aerosolized prostaglandin I2. Iloprost is a stable lipophilic analog of natural prostaglandin I2, a short acting natural prostaglandin. The vasoactive of iloprost, in combination with its prolonged plasma half-life, results in its systemic actions of lowered mean arterial pressure and reduced systemic vascular resistance.
- Nebulized prostaglandins are attractive in that they have limited systemic effects, are cheap and do not require specialized delivery systems. The particle size, however, cannot be easily controlled and hence inefficient of delivery may be significant resulting in higher doses with potential systemic side effects.

Nitric oxide
- Inhaled nitric oxide (NO) by virtue of its localized vascular endothelial action, through cGMP generation and its interaction with calcium gated potassium channels and protein kinase G as well as cGMP independent paths, acts as pulmonary vasodilator. Its effects are limited to the ventilated areas of the lung, with minimal systemic effects due to its rapid hepatic metabolism.
- No benefit outcome has yet been demonstrated in responders, however, although oxygenation and pulmonary resistance do improve.
- Withdrawal of nitric oxide has been shown to result in rebound pulmonary hypertension.
- Intravenous nitric oxide requires specialized delivery systems and the side effect profile is significant, with platelet dysfunction, myocardial depression, renal failure and the formation of toxic compounds such as peroxynitrites. The side effects are dose dependent and although recommended doses are under 10 ppm quantities to 80 ppm have been used.

Sildenafil
- Sildenafil is a phosphodiesterase V enzyme, whose inhibition prolongs the action of cGMP, with the overall effect of reducing pulmonary vascular tone.
- Sildenafil and vardenafin, members of the same class of phosphodiesterase inhibitors, have similar effects but of different magnitude and duration. Sildenafil has been evaluated in compensated right ventricular dysfunction, but its lack of an intravenous preparation limits its use.

Systemic vasodilators
- Systemic vasodilators such as sodium nitroprusside, glyceryl trinitrate and hydralazine all reduce pulmonary arterial afterload but at the expense of systemic hypotension, decreasing coronary collateral pressures and potentially leading to a deleterious preload reduction, exacerbating the dysfunction of the right ventricle, already compromised because of high right ventricular and diastolic pressure, through ischemia.
- Hence selective pulmonary vasodilators are more desirable in reducing afterload than global agents.

Recombinant BNP
- Natriuretic peptides induced by myocardial stress and dilatation are an attractive means to detect heart failure and to monitor response to therapy. They have been used to stratify outcome in acute pulmonary edema in long-term follow-up for patients with congestive heart failure, and also as predictors of mortality in dilatation cardiac failure, renal failure, amyloidosis, sepsis and diabetes.
- Plasma levels of natriuretic peptides have been shown to be proportional to the magnitude of right ventricular dysfunction and correlate negatively with the ejection fraction.
- Levels vary in populations, sex, age groups and by disease state. In the critically ill patient population natriuretic peptides may be elevated due to underlying or coexisting heart disease or lung disease.

- The aim in the management of right ventricular dysfunction is to disrupt the cycle of auto-aggravation.
- For right ventricular dysfunction, reducing afterload is critical to achieve the desired result.
- Similarly in a normal afterload state, augmentation of contractility raises the right ventricular ejection fraction.
- Ventricular systolic function is difficult to judge. In a dilated compensated systolic ventricle, volume reduction is most likely to improve the right ventricular ejection fraction. In the absence of elevated right atrial pressure then monitored volume changes are justified.
- Right ventricular systolic or diastolic dysfunction must be considered.
- Hypoxаemia and hypercapnia worsen pulmonary artery pressures as does positive end-expiratory pressure (PEEP), intracranial pressure and high tidal volumes. Optimization of these variables needs to occur before pharmacological manipulation is undertaken.

- The management of acute right ventricular infection should follow standard guidelines for the prevention of occulted coronary arteries.
- The pathophysiology of right ventricular support

Clinical diagnosis
- Right ventricular function:
- In systole, because of the constraints imposed by the pericardium, the high pressure in the left ventricle and the heart's anatomical configuration, the septum intrudes into the right ventricular cavity.
- The right ventricle is better suited to volume overload than the left, but increased afterload is more detrimental.
- The low pressures in the right side of the heart arise as a result of the thin walled ventricle and highly compliant pulmonary circulation.
- In pulmonary hypertension, dilatation occurs as a compensating mechanism.

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