

general

- Right heart failure is characterized by a low cardiac output, hypotension, hepatic enlargement and raised JVP.
- Cardiogenic shock due to right ventricle failure has a mortality rate comparable to left ventricle failure

Primary causes	Decompensated chronic disease states
Volume/pressure overload Left ventricular failure Acute respiratory distress syndrome Massive pulmonary embolus Anemic fluid embolus Mechanical Mechanical ventilation Other Sepsis Cardiac Cardiomyopathies - dilated ischaemic Valvular regurgitation (tricuspid/pulmonary) Tricuspid valve rupture (traumatic/infectious)	Respiratory disease Cor pulmonale - interstitial lung disease Chronic obstructive pulmonary disease Sleep apnoea and alveolar hypoventilation Muscle weakness Neuromuscular disease Polymyositis Amyotrophic lateral sclerosis Muscular dystrophy Connective tissue disease Mixed connective tissue disease Systemic lupus Primary Sjogrens Rheumatoid arthritis Hepatic porto-pulmonary syndrome Cardiac Decompensated adult congenital heart disease Intra-cardiac shunt Cardiomyopathies - dilated ischaemic

causes

- problems with clinical diagnosis:
- (i) right ventricular failure may exist in the absence of peripheral oedema.
 - (ii) peripheral oedema is not discriminatory for right heart failure.
 - (iii) elevated jugular venous pressures and abnormal waveforms may be distorted by mechanical ventilation, body habitus and lung hyperinflation in COPD patients.
 - (iv) signs such as hypotension, tachycardia, tachypnoea & hepatomegaly are nonspecific.

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.
 - The lack of myocardial bulk means that contractility cannot be maintained in the face of increased pulmonary resistance.
 - In pulmonary hypertension, dilatation occurs as a compensating mechanism.

pathophysiology

- Right heart failure:
- In all cases, there is a critical point at which ventricular dilatation cannot compensate.
 - Consequently, there is reversal of the ventricular septal pressure gradient, abnormal septal movement, rising atrial pressures and TR.
 - The abnormal volume and pressure loading stress the right side of the heart, resulting in increased oxygen demand, decreased coronary driving pressures and worsening right ventricle output. The global reduction in left sided preload contributes to systemic hypotension exacerbated by septal dyskinesia and reversal of the interventricular dependence pressures. This in turn further lowers coronary perfusion pressures. This vicious cycle has been termed auto-aggravation.

imaging

- CXR
- Changes in the pulmonary vasculature and the cardiac shadow may allow the diagnosis of underlying pathology potentially associated with pulmonary hypertension and, by inference, right ventricular involvement
- Echocardiography
- Echocardiography can show structural change, dynamic responses to intervention and allows quantitative and qualitative measurements to refine the significance of findings.
 - With transthoracic echocardiography (TTE) multiple measurements, ratios and estimates have been used to assess quantitative and qualitative parameters including:
 - (i) tricuspid regurgitation;
 - (ii) long axis cavity size,
 - (iii) short axis septal kinetics,
 - (iv) apex loses triangle shape;
 - (v) right ventricular end-diastolic area/left ventricular end-diastolic area (>0.6 or >1);
 - (vi) left inferior hypokinesis;
 - (vii) right ventricle size in comparison to left ventricle;
 - (viii) right ventricular end diastolic volume diameter >30mm at level of mitral valve from left precordial view;
 - (ix) loss of inspiratory collapse of inferior vena cava
 - (x) dilation of pulmonary artery,
 - (xi) tricuspid regurgitation;
- Right heart catheterisation
- Right heart catheterisation and thermodilution are invasive but can provide nearly continuous values, in contrast to other modalities, for right heart cardiac output and continuous right heart pressures.

biochemistry

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

general treatment aims

- The aim in the management of right ventricular dysfunction is to disrupt the cycle of auto-aggravation.
- For a given contractile state, reducing afterload will increase the ejection fraction.
- Similarly in a normal afterload state, augmentation of contractility raises the right ventricular ejection fraction.
- Volume status is difficult to judge. In a dilated decompensated ventricle with elevated atrial pressures, volume reduction is most likely to improve the right ventricular ejection fraction. In the absence of elevated right atrial pressure then monitored volume challenges are justified.
- Reduction in myocardial oxygen demand or improvements in coronary perfusion must also be considered.
- Hypoxaemia and hypercarbia worsen pulmonary artery pressures as does positive end expiratory pressure (PEEP), intrinsic PEEP and high tidal volumes. Optimization of these variables needs to occur before pharmacological manipulation is undertaken.

Surgical, interventional and right ventricular support

- The management of acute right ventricular infarction should follow standard guidelines for the reperfusion of occluded coronary arteries.
- Pacing, where indicated, has been shown to reduce mortality in biventricular failure and should be considered in order to restore atrioventricular synchrony to ensure adequate preload
- Right ventricular assist devices may be appropriate in particular circumstances

acute right ventricular dysfunction [created by Paul Young 22/10/07]

volume optimisation

- Failure can be defined as the point at which the right ventricle fails to compensate for an increased ventricular volume, as each fibre has an optimal stretch to allow maximal pressure generation, which, when exceeded, results in dilatation and eventually ventricular failure.
- Determination of preload is problematical but the presence of high right atrial filling pressures is indicative of elevated right ventricular pressures, which extrapolates to a raised ventricular volume. This may not necessarily be true in all cases and depends on the compliance of the ventricle.
- In chronic elevation of right atrial pressures the pressure may be high, but this is a poor predictor of volume response, the patient may therefore still be volume recruitable and sequential monitored fluid challenges are justified.
- 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 effect of therapy can be monitored by sequential echocardiography or by using right heart catheterization and, ideally, continuous measurement.
- The converse is true though: sequential volume challenges monitored by pulmonary artery pressure changes in the absence of reversed right ventricle interdependence will increase cardiac output, up to the individualized optimal filling point.

inotropes & vasopressors

- No selective right heart inotrope exists.
- Augmentation of contractility can be achieved by b-mimetics, calcium sensitizers and 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, lusitropic agent, levosimendan has been shown to provide a survival advantage in heart failure trials. In a pilot study of levosimendan 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 ischaemia related to hypotension is a problem.
- They elevate the mean arterial pressure, coronary artery perfusion and may, consequently, reduce myocardial work.
- Vasotropic agents such as noradrenaline, phenylephrine and vasopressin may elevate diastolic 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.
- Phosphodiesterase inhibitors such as milrinone and amrinone inhibit the phosphodiesterase enzymes responsible for cAMP/cGMP breakdown, augmenting myocardial contractility.
- The advantage of this drug class is that the mechanism is independent of b-adrenoceptor states and does not increase myocardial oxygen demand. Nebulized milrinone, interestingly, has been shown to have an additive effect with prostaglandin I2 in terms of pulmonary vasodilatation.

afterload reduction

- General
- The poor contractile reserve of the right ventricle means that the primary modality for treatment of acute right ventricle dysfunction secondary to elevated pulmonary artery pressures is by means of selective pulmonary vasodilatation.
 - Acutely, afterload reduction may be effected by using localized (inhaled) or systemic vasodilators. The consequences of selected pulmonary vasodilatation are of decreased resistance (and consequently afterload), improved VQ matching and decreased arterial hypoxaemia.
- Prostaglandins
- Prostaglandins can be given by inhalation, systemically or subcutaneously.
 - The vasodilatory effects are mediated by nitric oxide release and interaction at a local level with the vascular endothelial smooth muscle. Prostaglandin E1 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 carbocyclic analogue of prostaglandin I2, a short acting natural prostaglandin. The vascular spillover of inhaled iloprost, in combination with its prolonged plasma half-life, results in its systemic actions of lowered mean arterial pressure and 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 easily be controlled and hence inefficiency of delivery may be significant resulting in higher doses with potential systemic spillover.
- 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 overspill because of its rapid inactivation by haemoglobin
 - No outcome benefit 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
 - Inhaled 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 up 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.
 - Tadalafil and vardenafil, members of the same class of phosphodiesterase inhibitors, have similar effects but of different magnitude and duration. Sildenafil has been evaluated in decompensated 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 afterload but at the expense of systemic hypotension, decreasing coronary ostial perfusion pressures and potentially leading to a deleterious preload reduction, exacerbating the dysfunction of the right ventricle, already compromised because of high right ventricular end diastolic pressure, through ischaemia. Hence selective pulmonary vasodilators are more desirable in reducing afterload than global agents.

Mechanical ventilation & PEEP

- Recombinant BNP
- Neseritide is a recombinant version of BNP. Its actions, when infused, are identical to the in-vivo effects of BNP (natriuresis, sympathetic dampening and suppression of the renin- angiotensin axis by increasing cGMP). It reduces both preload and afterload, consequently improving cardiac output without inotropy. Concerns exist over the decreased 30-day survival and its adverse impact on renal function.
 - In addition the systemic side effect can be that of hypotension and subsequently, decreased coronary perfusion pressures.
 - To date it has not been evaluated in pure right heart failure.
- Mechanical ventilation & PEEP
- The distending alveolar pressure, when transmitted through the pulmonary capillary bed, determines the opening pressure of the pulmonary artery valve.
 - The greater the tidal volume the greater the impedance and hence the myocardial power generation has to be increased.
 - Pleural pressure is transmitted to the myocardium because of the constricting pericardium, which limits the extent of ventricular distension. Thus for an increase in pleural pressures a consequently higher preload is required to maintain the right ventricular end diastolic volume
 - A right ventricular friendly strategy is to set PEEP to limit gas trapping with prolonged expiratory times and to utilize as low a tidal volume and respiratory rate as possible without deleterious ventilatory consequences