Cor Pulmonale

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Risk

- RV failure is the third most common cardiac Dx after age 50 y.
- Of all CHF admissions, 10–20% have some aspect of right heart failure.
- Gender predominance is male > female.

Perioperative Risks

- Increased risk for respiratory failure, severe right heart failure (≥10% if cor pulmonale Dx made preop)
- Risk of prolonged postop ventilatory support

Worry About

- Increased PVR may cause systemic hypotension due to RV dysfunction, resulting in decreased LV filling
- Hypoxia, hypoxemia, hypercarbia, and acidosis intraop or in early postop period, which increase PVR
- Underlying CAD and LV dysfunction

Overview

- Alteration in RV structure (hypertrophy) and function (decreased)
- Most common cause: Long-standing LV dysfunction leading to RV failure, with other common causes including chronic pulmonary emboli and end-stage COPD resulting in increased PVR (secondary to chronic hypoxia and structural changes)
- Any disease that increases PVR chronically, which can induce RV changes, including idiopathic and toxin-induced pulm Htn, pulm fibrosis, severe obstructive sleep apnea, CHD with chronic RV overload, or RV outflow obstruction
- Prognosis: Favorable for those who can maintain a near-normal PaO_2; unfavorable for those with structural changes

Etiology

- LV heart failure
- COPD: Smoking or severe asthma
- Long-standing untreated OSA
- Acute or chronic pulm embolus
- CHD with RV volume overload (L-to-R shunt and long-standing pulmonic insufficiency) or afterload increase (pulm outflow obstruction)
- Primary pulm Htn or severe pulm fibrosis

Usual Treatment

- Use a lynchpin to optimize RV function by decreasing its afterload and PVR.
- Always optimize conditions to decrease PVR toward normal levels by treating hypoxia and hypercarbia.
- Vasodilators (only one-third of pts improve). Chronic treatments include oral medications such as calcium channel blockers; phosphodiesterase-5 inhibitors (sildenafil, and tadalafil); endothelian receptor antagonists (bosentan); inhaled or IV prostacyclin analogues such as epoprostenol, iloprost, and treprostenil; and IV or inhaled phosphodiesterase-3 inhibitors (milrinone). Acute treatments include inhaled nitric oxide, prostacyclin analogues iloprost, epoprostenol, or IV milrinone.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	RV failure Increased PVR Tricuspid regurgitation	DOE Effort-related syncope Chest pain	Accentuated pulm S_2 Diastolic or systolic murmur Dependent edema	CXR ECHO Right heart cath
RESP	COPD	DOEChronic cough, sputum	Hyperinflated lungsWheezing, rhonchi	CXR PFTs
GI	Passive congestion of liver or spleen		Hepatosplenomegaly	LFTs Albumin PT
RENAL	Impaired ability to excrete Na^+ and H_2O	Edema	Edema	Urinary Osm Urine-specific gravity
CNS	Stimulation of sympathetic nervous system secondary to hypoxia		Tachycardia	

Key References: Hosseinian L: Pulmonary hypertension and noncardiac surgery: implications for the anesthesiologist, *J Cardiothorac Vasc Anes* 28(4):1064–1074, 2014; Fox DL, Stream AR, Bullt T: Perioperative management of the patient with pulmonary hypertension, *Semin Cardiothorac Vasc Anest* 18(4):310–318, 2014.

Perioperative Implications

Preoperative Preparation

- Mortality risk in pts with primary pulm Htn and cor pulmonale high (2–20%); morbidity as high as 20–40%.
- Treat underlying infections.
- Maximize treatment of reversible airway disease.
- Avoid preop medications that will depress ventilation.
- Consider baseline ABG to assess PaO_2 and $PaCO_2$.

Monitoring

- Consider arterial line for beat-beat arterial pressure monitoring, noninvasive cardiac output measurement, and ABG collection.
- Consider intraop TEE to monitor RV function and RV dilation.
- Consider pulm arterial catheter to monitor PA pressures and CVP monitoring for evaluation of RV function for large fluid shift reoperations; remember that pts with pulm Htn have increased risk of pulm artery rupture.

Airway

- Potential for bronchospasm

Induction

- Try to increase SVR if pt has fixed increased PVR.
- Deep anesthesia for intubation may decrease incidence of bronchospasm and sympathetic stimulation, which increases PVR; however, caution must be used to avoid hypercarbia.

Maintenance

- Potent inhalational agents for bronchodilation.
- Consider avoiding nitrous oxide (which may increase PVR) and large doses of narcotics (which may cause postop hypoventilation and hypercarbia).
- Although positive pressure ventilation may increase PVR secondary to alveolar expansion (optimal TV around FRC), it can decrease PVR secondary to better oxygenation.
- Aggressively prevent hypercarbia, hypoxemia, and hypothermia, all of which may cause an increase in PVR.
- Hypovolemia and hypervolemia will influence RV function; TEE and CVP may be useful.
- Consider the use of inhaled NO or iloprost to treat increased PVR.
- Consider use of IV milrinone to decrease PVR (but beware of decreased SVR, which may require treatment with an α-adrenergic agonist).
- Consider the use of beta-adrenergic agents such as dobutamine or epinephrine to support RV cardiac output if faced with hemodynamic instability.

Extubation

- Bronchospasm may occur during emergence.
- Avoid hypoventilation and resultant hypercarbia.

Adjuvants

- Regional anesthesia is an option, but high level may decrease SVR in pts with a fixed increased PVR, leading to CV collapse.
- Inhaled NO or iloprost.
- Preop phosphodiesterase-5 inhibitors may accentuate effects of intraop vasodilators.

Postoperative Period

- Postop pain management with either low-dose epidural local anesthetics with low-dose opioids or low-dose intrathecal opioids can minimize resp depression.

Anticipated Problems/Concerns

- Increased PVR and RV dysfunction from hypoxia/hypercarbia or hypothermia