Editorial

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Management Strategies for Patients with Pulmonary Hypertension

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Description

Pulmonary hypertension encompasses a spectrum of pathologies best characterized by their anatomical location: Precapillary arteries and arterioles, alveoli and capillary beds and post capillary pulmonary veins and venules. Idiopathic pulmonary arterial hypertension is the result of increased vasoconstriction, pulmonary vascular remodeling and in situ thrombosis provoked by endothelial dysfunction, smooth muscle proliferation and neointimal formation in the precapillary arteries and arterioles.

Pulmonary venous hypertension is typically a result of left ventricular diastolic dysfunction, valvular heart disease, or pulmonary venous disorders. Compared with the left ventricle, the right ventricle demonstrates a heightened sensitivity to changes in afterload. Right ventricular stroke volume decreases proportionately to acute increases in afterload. In addition, a normal right ventricle cannot acutely increase the mean PAP to more than 40 mm Hg. Right ventricular systolic dysfunction, severe tricuspid regurgitation, arrhythmias, and left ventricular dysfunction caused by ventricular interdependence may contribute to low cardiac output and hypotension in patients with pulmonary hypertension.

Pulmonary hypertension may first be recognized when an echocardiogram is obtained or a pulmonary artery catheter is placed for hemodynamic monitoring. Determining the cause and significance of the elevated PAP then dictates appropriate therapy. A comprehensive work-up is then necessary to determine the cause and hemodynamic consequence of pulmonary hypertension.

Physical examination of patients with right ventricular failure classically reveals an elevated jugular venous pulse with a large v wave. An early finding is a prominent pulmonic component of the second heart sound. Other findings may include a palpable right ventricular heave, and the holosystolic blowing murmur of tricuspid regurgitation murmur along the left lower sternal border.

Laboratory evaluation is undertaken to identify reversible causes of pulmonary hypertension. In the ICU however, many laboratory derangements result from critical illness itself. Nonetheless, clues may be provided as to the underlying cause of pulmonary hypertension by polycythemia.

Complications of PA catheterization are particularly dangerous in patients with pulmonary hypertension and right ventricular strain. Tachyarrhythmia's have potentially life-threatening consequences of decreased stroke volume due to shortened filling time or deterioration into fatal arrhythmias. Obtaining a pulmonary capillary wedge pressure also may be difficult in patients with markedly elevated pulmonary pressures.

In patients with pulmonary hypertension and respiratory failure mechanical ventilation may have untoward hemodynamic effects. Increases in lung volume and decreases in functional residual capacity can increase PVR and right ventricular afterload.

Pulmonary vasodilators can be classified into two main categories: those that increase production of cyclic guanidine monophosphate and cyclic adenosine monophosphate, such as nitric oxide and prostanoids, respectively and those that decrease the breakdown of cyclic guanosine monophosphate such as sildenafil and zaprinast and of cyclic adenosine monophosphate such as milrinone.

Some causes of pulmonary hypertension and hemodynamic instability encountered in the ICU require urgent therapy of the underlying disorder. Even in these cases, the aforementioned drugs are sometimes useful in the short term.

Cardiac and thoracic surgery may be complicated by postoperative pulmonary hypertension. Pulmonary hypertension is recognized as a major risk factor for morbidity and mortality in cardiothoracic surgery. Although the ethology of postoperative pulmonary hypertension is unclear, pulmonary parenchymal and endothelial injury due to cardiopulmonary bypass.