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Pulmonary Hypertension in the Critical Care Setting: Classification, Pathophysiology, Diagnosis, and Management

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Pulmonary hypertension (PH) is common in the critical care setting, and may be a target for specific therapy. Moderate degrees of PH are most often the consequence of acute or chronic heart failure, hypoxemia, or acute pulmonary embolism (PE), and may be relatively rapidly reversible. The consequences of more severe forms of PH, both acute and chronic, can include hypotension; low cardiac output; right heart failure with congestion of the liver, gut, and kidneys; and varying degrees of hypoxemia, each of which can lead to death or severe disability. We will review the physiology, definitions, classification, pathogenesis, diagnostic tools and algorithms for the diagnosis and specific treatments for the various causes of PH as seen in the critical care setting.

Physiology, definition, and classification

Normally the pulmonary artery pressure is about one fifth of systemic pressure. The pulmonary vasculature in adults and children has excellent vasodilator reserve and accommodates increases in flow. However, the pulmonary vasculature, like the systemic vasculature, can respond in varying degrees and pathologically to several triggers including pressure, flow, hypoxemia, toxins, and emboli, which can induce endothelial dysfunction, loss of elastance, smooth muscle vasoconstriction, and cellular hypertrophy resulting in decreased luminal diameter of the resistance vessels, the pulmonary arterioles.

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Significant PH is most commonly defined as an sPA or right ventricular systolic pressure (RVSP) >40 mm Hg or mean PA (mPA) >25 mm Hg at rest or $>$ 30 mm Hg during exercise. Additional criteria for PAH are a PCW $\leq$ 15 mm Hg, and pulmonary vascular resistance (PVR) $\geq$ 3 Wood units (also known as RU or resistance units) [2]. This definition has been used to characterize PAH for epidemiologic studies and new drug evaluation. Lesser degrees of PH with lower pulmonary vascular resistance can be found in mildly symptomatic persons with early PAH. With an increase in stroke volume from obesity, anemia, or sepsis, the sPA or right ventricular systolic pressure (RVSP) estimated from the echo-Doppler may exceed 40 mm Hg.

In the intensive care unit (ICU) patient, PH may be suspected due to characteristic signs and symptoms, discovered incidentally on an echo-Doppler, or its presence may be known at the time of ICU admission. Of course, multifactorial pulmonary hypertension is common and more often the rule in the ICU. The Venn diagram in Fig. 1 depicts the potential relationship between the various etiologies of PH. For example, pulmonary emboli, both acute and chronic, could result in PH from hypoxemia, pulmonary vasoconstriction, decrease in the pulmonary vascular volume, and often occurs in the setting of congestive heart failure (CHF). Chronic left heart failure or mitral valve disease can result in pulmonary congestion and interstitial lung disease, hypoxemia, hypertrophy of the pulmonary arterioles, and noncompliance of the major pulmonary arteries.

The determinants of the systolic pulmonary artery pressure (sPA) include the right ventricular stroke volume and compliance of the main pulmonary artery and its branches. The diastolic pulmonary artery pressure (dPA) determinants include the tone of the pulmonary arterioles, the size of the pulmonary vascular bed ($>100{,}000$ pulmonary arterioles), the pulmonary capillary wedge

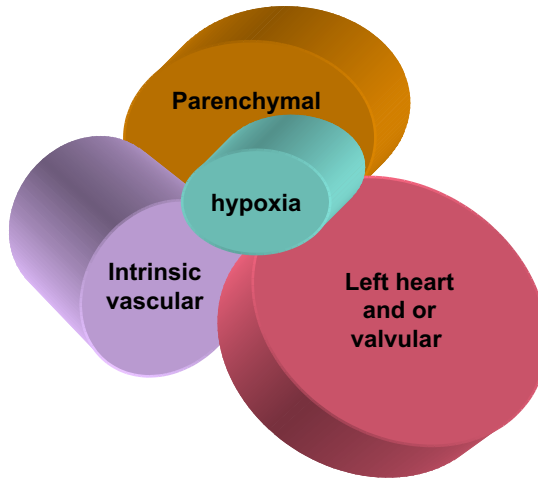

Fig. 1. Paradigm of multifactorial pulmonary hypertension.