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Pulmonary Arterial Hypertension in Young Patients

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Perspective

Pulmonary arterial hypertension, also known as group 1 pulmonary hypertension according to the World Health Organization categorization system, is a serious consequence of systemic sclerosis caused by pulmonary vascular involvement. The high mortality seen in systemic sclerosis-associated pulmonary arterial hypertension is likely due to impairment of right ventricular systolic function and the presence of other non-group-1 pulmonary hypertension phenotypes that may impede clinical response to pulmonary arterial hypertension-targeted therapy. This article outlines two areas of recent progress in the therapy of individuals with systemic sclerosis and pulmonary hypertension: the tolerability of pulmonary arterial hypertension-targeted therapy in patients with mild to severe interstitial lung disease, as well as the therapeutic importance of the antifibrotic impact of soluble guanylate cyclase stimulators seen in preclinical studies.

Pulmonary hypertension is linked to a higher risk of maternal mortality and morbidity during pregnancy. Our systematic review of the literature on the use of targeted treatments for pulmonary arterial hypertension during pregnancy found that mortality has decreased significantly since a previous review in 1998 (16 percent *vs*. 38 percent), with a further non-significant decrease since the most recent review in 2009 (16 percent *vs*. 25 percent). Aside from the use of targeted treatments, the prompt implementation of these treatments, as well as their early planned delivery, may help to improve the outcome. Furthermore, research suggests that women with mild pulmonary hypertension or a favourable functional class may have a better prognosis, but there is no evidence that these women have a lower mortality rate.

Despite the improved prognosis, pregnancy is not recommended in women with pulmonary hypertension, and termination should be considered if one occurs. If the pregnancy persists, a multidisciplinary team in a specialty centre should be consulted. Pulmonary vasoconstriction is caused by hypoxia. Regional hypoxic vasoconstriction enhances perfusion-alveolar ventilation matching. Right ventricular afterload is increased by global hypoxic vasoconstriction. In mammals and birds, the hypoxic pulmonary pressor response is ubiquitous, but there is considerable interspecies and interindividual diversity. In proportion to the initial vasoconstriction, chronic hypoxia causes pulmonary hypertension. Long-term hypoxia is linked to an increase in red blood cell bulk, which exacerbates pulmonary hypertension by increasing blood viscosity.

Humans have mild to moderate hypoxic pulmonary hypertension, but the pulmonary vascular pressure-flow relationships are steep, resulting in a significant afterload on the right ventricle after exercise. With intake-specific pulmonary vasodilating therapies, a partial recovery of 10-25 percent of the hypoxia-induced decrease in maximum oxygen uptake has been described. Hypoxia has been shown to reduce the contractility of cardiac fibres *in vitro*. In intact animals, however, the acutely hypoxic right ventricle can maintain the coupling of its contractility to increased afterload. In healthy hypoxic human participants, echocardiographic examinations of the right ventricle demonstrate decreased diastolic function, but intact or even augmented systolic function initially and modestly lowered chronically. In patients with chronic mountain sickness, these findings are more prominent. The clinical significance of these findings is still unknown. In a small number of people who develop severe pulmonary hypertension and clinical right ventricular failure in hypoxia, almost no imaging studies of right ventricular function have been documented. There are currently no imaging studies of right ventricular function during hypoxic exercise in normal persons. While it's possible that the right ventricle affects exercise capacity in hypoxia, more research is needed to confirm this.

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