2. Nitric oxide therapy

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Nitric oxide is endogenously produced by a variety of cells including pulmonary artery endothelial cells. It has been shown to represent endothelium dependent relaxation factor.

Nitric oxide, when inhaled as a gas has been shown to have pulmonary vasodilating properties. Importantly, inhaled NO has no systemic hemodynamic effects ; it is a completely selective pulmonary vasodilator.

Inhaled NO has been examined in a variety of animal models of pulmonary hypertension as well as in several human diseases characterized by pulmonary hypertension and/or arterial hypoxemia.

It is important to note that all reports of inhaled NO to date have been essentially anecdontal uncontrolled case series. NO randomized controlled trial of inhaled NO has been completed showing efficacy.

The following applications of inhaled NO will be discussed :

Animal models

1. Hypoxic pulmonary vasoconstriction
2. Acute pulmonary embolism
3. Thromboxane-analogue induced pulmonary hypertension
4. Oleic acid induced lung injury

Human studies

HYPOXEMIC STATES :

ARDS

Obstructive lung disease

Pulmonary fibrosis

PULMONARY HYPERTENSIVE STATES

Congenital heart disease

Cardiomyopathy

Thromboembolic disease

Primary pulmonary hypertension

Despite clear evidence that inhaled nitric oxide can have dramatic hemodynamic and gas exchange effects, several unanswered questions remain :

1. Does inhaled NO lead to improved outcome in hypoxemic or pulmonary hypertensive states ?
2. Are there any currently unrecognized toxicities of long term NO administration ?

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