The Basic Problem

Respiratory therapists are expert at the aerosol delivery of medication. We recognize the many advantages of aerosol delivery, including decreased cost, less systemic exposure, fewer systemic side effects, faster onset of action at the site of disease, faster delivery, and "user friendliness" compared with intravenous administration.[1] However, for an aerosolized medication to be safe and effective, it needs to be able to be delivered as an aerosol, which often means it needs to be soluble or solubilized in a carrier, it must not be inactivated at the airway surface, it must not be harmful to the airway or the lung, and it must have demonstrated efficacy. Clearly not every drug is a candidate for aerosol delivery, and drugs that are administered by aerosol often require special formulations different from those used in intravenous preparations.[2]

Intravenous formations of epoprostenol have been delivered via aerosol and are effective pulmonary vasodilators in very sick patients with pulmonary hypertension. Aerosolized intravenous epoprostenol is similar in effectiveness to inhaled nitric oxide for improving oxygenation, decreasing right-ventricular afterload, and decreasing pulmonary artery pressure.[3] However, inhaled nitric oxide is far more expensive.

Despite its effectiveness, there are very few data related to the safety of intravenous epoprostenol when given as an aerosol. In this issue of RESPIRATORY CARE, Kuch and colleagues[4] evaluated the effect of aerosolized intravenous epoprostenol in either a glycine buffer or a sucrose/L-arginine buffer. Human airway cells were grown and differentiated at an air-liquid interface to form an organotypic airway to test the effects of these aerosols in vitro. They demonstrated that epoprostenol in either of these buffers rapidly induced ciliostasis and airway cell death. Given the very high pH (ie, 11–13) of the buffers commonly used for epoprostenol aerosols, it was not surprising to see adverse effects on the airways, but these results were dramatic.[4]

These studies were conducted in vitro on an organotypic airway model; however these adverse effects may not be as severe when administered to persons with pulmonary hypertension.[5] On the other hand, patients receiving aerosolized epoprostenol are often acutely ill with severe cardiopulmonary disease and are at greatest risk for additional insults to their lungs. Given these results, it is prudent that, if epoprostenol is to be administered to critically ill patients via aerosol, the administration time be minimized. There is also an opportunity for developing inhaled vasodilators in a buffer solution with a more neutral pH. Although it is critically important to improve oxygenation and decrease pulmonary artery pressure in severely ill patients, this should not be at the cost of airway damage and thus increasing the risk of mucostasis and infection.

SEE THE ORIGINAL STUDY ON PAGE 1427

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