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## Cardiopulmonary support and physiology

# Inhaled prostacyclin reduces cardiopulmonary bypass–induced pulmonary endothelial dysfunction via increased cyclic adenosine monophosphate levels

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**OBJECTIVE:** Cardiopulmonary bypass triggers a systemic inflammatory response that alters pulmonary endothelial function, which can contribute to pulmonary hypertension. This study was designed to demonstrate that inhaled prostacyclin, a selective pulmonary vasodilator prostaglandin, prevents pulmonary arterial endothelial dysfunction induced by cardiopulmonary bypass.

**METHODS:** Three groups of Landrace swine were compared: control without cardiopulmonary bypass (control group); 90 minutes of normothermic cardiopulmonary bypass (bypass group); 90 minutes of cardiopulmonary bypass and treated with prostacyclin during cardiopulmonary bypass (continuous nebulization with continuous positive airway pressure until the end of the cardiopulmonary bypass; prostacyclin group). After 60 minutes of reperfusion, swine were put to death and pulmonary arteries harvested. After contraction to phenylephrine, endothelium-dependent relaxation to bradykinin and acetylcholine was studied in standard organ chamber experiments. The pulmonary artery intravascular cyclic adenosine monophosphate content was compared between the 3 groups (post-cardiopulmonary bypass).

**RESULTS:** There was a statistically significant improvement of the endothelium-dependent relaxation to bradykinin in the prostacyclin group when compared with the bypass group ( $P < .05$ ). There was no statistically significant difference for endothelium-dependent relaxation to acetylcholine ( $P > .05$ ) between the prostacyclin and the bypass groups. There was a statistically significant decrease in the cyclic adenosine monophosphate content and a statistically significant increase of the mean pulmonary artery pressure in the bypass group only ( $P < .05$ ).

**CONCLUSION:** Prophylactic use of inhaled prostacyclin has a favorable impact on the pulmonary endothelial dysfunction induced by cardiopulmonary bypass associated with preservation of pulmonary intravascular cyclic adenosine monophosphate content and the pulmonary vascular tone.

