

**Adams JA. Endothelium and cardiopulmonary resuscitation. Crit Care Med. 2006 Dec;34(12 Suppl):S458-65. Review.**

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The endothelium is a viable target for injury, repair and cellular modulation. Because of its vast extension and active metabolic status of producing mediators for vasomotor tone, coagulation, and inflammation, it is a key target for therapy during ischemia/reperfusion injury. Cardiopulmonary resuscitation is a model of whole-body ischemia/reperfusion injury. It has become apparent that the endothelium participates in a host of responses elicited by ischemia/reperfusion. This review examines the role of the endothelium during and after ischemia/reperfusion and the participation by its mediators and evidence for endothelial involvement during and after cardiopulmonary resuscitation. The strategic location of the endothelium makes it an excellent signal transduction mechanism for a host of disease processes. In addition to biochemical stimuli, mechanical stimulation of the endothelium elicits production of several mediators, including endothelium-derived nitric oxide, prostaglandins, and antithrombotics and anticoagulants. Whole-body, periodic acceleration is a novel method of stimulating the endothelium via pulsatile shear stress. Periodic acceleration has been shown to be an effective experimental method of cardiopulmonary resuscitation, with evidence of postresuscitation cardioprotective effects. This review indicates that understanding endothelial modulation during and after ischemia/reperfusion will significantly improve therapeutic choices.