TACSM Abstract

Hypoxic Preconditioning Attenuates Ischemia-reperfusion Injury in Older Adults

STEN STRAY-GUNDERSEN, SAHAR D. MASSOUDIAN, FRANK WOJAN, HIROFUMI TANAKA, and SOPHIE LALANDE

Clinical Exercise Physiology Laboratory; Department of Kinesiology and Health Education; The University of Texas at Austin; Austin, TX

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Lalande, Sophie (sophie.lalande@austin.utexas.edu)

ABSTRACT

Sudden restoration of blood flow to an ischemic vessel paradoxically damages endothelial cells. In young healthy adults, ischemic preconditioning, caused by repeated periods of brief ischemia induced by local cuff inflation prior to reperfusion, attenuates endothelial dysfunction following an ischemia-reperfusion injury. However, ischemic preconditioning does not consistently protect against ischemia-reperfusion injury in older adults. Intermittent systemic hypoxemia, induced via brief bouts of breathing low levels of oxygen, attenuates endothelial dysfunction following an ischemia-reperfusion injury in young adults. **PURPOSE**: To determine whether intermittent hypoxia protects against ischemia-reperfusion injury in older adults. **METHODS**: Twelve older adults (5 women, age: 57 ± 9 years, height: 173 ± 8 cm, body weight: 75.8 ± 13.4 kg) visited the laboratory on two separate occasions. Endothelium-dependent vasodilation was assessed by brachial artery flow-mediated dilation using a semiautomated diagnostic ultrasound system before and after 20 minutes of upper arm blood flow occlusion to induce an ischemia-reperfusion injury. Blood flow occlusion was preceded by either intermittent hypoxia, consisting of three 4-minute hypoxic cycles at a targeted arterial oxygen saturation of 80% interspersed with 4-minute room air cycles, or intermittent normoxia, consisting of three 4-minute normoxic cycles separated by 4-minute room air cycles. **RESULTS**: Intermittent hypoxia resulted in an arterial oxygen saturation of 80 ± 2%, which corresponded to oxygen levels of 11.4 ± 0.7%. When preceded by intermittent normoxia, blood flow occlusion reduced flow-mediated dilation by 4.1 ± 2.6% (6.5 ± 1.7 to 2.4 ± 1.7%). In contrast, flow-mediated dilation was reduced by 2.0 ± 1.5% when blood flow occlusion was preceded by intermittent hypoxia (5.6 ± 1.7 to 3.6 ± 2.3%, $P = 0.03$). When compared to intermittent normoxia, intermittent hypoxia resulted in a greater heart rate (60 ± 10 vs. 68 ± 10 bpm, $P < 0.01$) but did not affect cardiac output (5.1 ± 1.4 vs. 5.8 ± 1.8 L/min, $P = 0.11$). **CONCLUSION**: Hypoxic preconditioning attenuated the reduction in flow-mediated dilation induced by a 20-minute blood flow occlusion in older adults. Thus, exposure to intermittent hypoxia represents a promising strategy to protect against ischemia-reperfusion injury in populations at risk for ischemic events.