Abstract

Objective: Examine the endothelial function of conduit arteries above and below the level of spinal cord lesion using a method independent of arterial dimensions. Endothelial dysfunction is considered a highly sensitive marker of cardiovascular disease progression. Reports on endothelial function (using flow mediated dilation) after spinal cord injury (SCI) has been contentious, due to a lack of appropriate adjustment for differences in resting arterial dimensions as well as shear rate between SCI and able bodied controls. Moreover, exercise has been linked with improved vascular function through increases in blood flow and shear stress. This study also aims to assess the effect of passive exercise on the endothelial function of conduit arteries of SCI animals.

Design: Experimental cross sectional study.
Participants/methods: We examined endothelial function in Wistar rats with complete T3 spinal cord transection (SCI), T3 transection and passive exercise (PE), and uninjured controls. In vitro wire myography was used to examine endothelial mediated vasodilation (acetylcholine, ACh) in the BA and FA of each rat.

Results: Femoral arteries from SCI animals exhibited impaired reactivity to ACh (i.e., requiring 5x greater ACh to reach 50% of maximal dilation; p<.01) compared to those of the controls. Passive exercise after SCI improved the sensitivity of FA (p<.01) to be similar to that of uninjured animals. However, brachial arteries from all groups showed similar responses to ACh (p=0.789).

Conclusion: We have shown, for the first time, the expected endothelial dysfunction in the inactive/supraspinally disconnected femoral artery after SCI. Furthermore, passive exercise of the hind legs was effective in preventing endothelial dysfunction. Together, this study provides mechanistic insight into cardiovascular disease progression after SCI, as well as a potential therapeutic intervention.

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Objective 1. Describe the endothelial function of conduit arteries above and below the level of spinal cord lesion.

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