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The NO-cGMP axis in endothelial ischemia and ischemic preconditioning

Tommaso Gori*[1] and John Parker[2]

Address: [1]Department of Internal Cardiovascular and Geriatric Medicine, University of Siena, Italy and [2]Department of Cardiology, Mount Sinai and University Health Network Hospitals Toronto, Canada

Email: Tommaso Gori* - tommaso.gori@utoronto.ca

* Corresponding author

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The biology of cardiac and peripheral ischemia and reperfusion (IR) injury is extremely complex, and the vascular endothelium plays a central role both in protecting from ischemic damage as well as in mediating this damage. Due to its strategic location and its intense biosynthetic activity, the vascular endothelium is particularly sensitive to IR: the endothelium is the first tissue damaged by IR, and human in vivo models of isolated endothelial IR injury have been developed that allow investigating the mechanisms of this phenomenon. Particularly during reperfusion, the rapid formation of superoxide anion and other reactive oxygen species causes endothelial damage, leading to the so-called no-reflow phenomenon. In this way, the endothelium determines permanent impairment to tissue reperfusion, extending the ischemic damage. At the same time, the endothelium, like any other tissue, can be preconditioned against IR damage, i.e., it is able to develop a protective phenotype that defends itself and the tissues from IR. We will discuss how the endothelium is the first casualty in the setting of IR, and at the same time how this tissue can be protected by physical and pharmacological stimuli, which opens new therapeutic possibilities.