Importance of Mitochondrial Proteins for Myocardial Ischemia/Reperfusion Injury and Protection From It

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Acute myocardial infarction with subsequent left ventricular dysfunction and heart failure remains a major cause of morbidity and mortality. Timely reperfusion improves outcomes, although reperfusion itself contributes to ultimate infarct size. Brief episodes of nonlethal ischemia/reperfusion can reduce injury by sustained ischemia/reperfusion. This so called “ischemic conditioning” recruits complex signal cascades of activation of sarcolemmal receptors and intracellular enzymes, reactive oxygen/nitrosative species, mitochondrial stabilization and finally inhibition of death signaling.

In this context Prof. Schulz will go into detail on the role of connexin 43 as a regulator of gap junctions but also of mitochondrial respiration, ATP generation and mitochondrial potassium influx. Prof. Schulz will discuss current experimental and clinical concepts to reduce ischemia/reperfusion injury.

12:00 – 13:00h
Kursraum 6
(on campus: Mittelallee 10, 1. UG)
Augustenburger Platz 1, Berlin