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Abstracts



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Ratlar: Microcirculation showed greater heterogeneity in both shape and size in the SHR-C group, and duration of centre spaces were observed. These alterations were less pronounced in the normotensive group. We found fragmented fragmentation of microcirculation in the SHR-C group ($p < 0.005$ vs. WKY), which was significantly attenuated by the L-22566 treatment. Western blot analysis showed decreased translocation of the p38/JNK1/2 protein into the microcirculation in the SHR-C group compared to the SHR-C group. No significant differences were observed in the level of p38/JNK1/2 protein.

Discussion: In a hypertensive animal model, the oxidative stress-induced "arterial" microcirculation fragmentation was significantly attenuated by L-22566 treatment. This may be due to the (assumed) opposing effect of p38/JNK1/2 inhibition towards well-known effect of increased oxidative stress resistance and on increased heterogeneity in stability of the heart.

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CARDIOPROTECTION IN METABOLIC DISEASES

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Ischemic pre- and postconditioning are well described endogenous adaptive responses of the heart to withstand ischemia-reperfusion injury. Most experimental studies on cardioprotection have been performed in animal models, in which ischemia-reperfusion is induced in the absence of other disease processes. However, ischemia-reperfusion is a human condition, which is caused by or associated with various cardiovascular risk factors, including hypertension, hyperlipidemia, diabetes, insulin resistance, obesity, and heart failure. In this review, we will discuss the role of these factors in the development of ischemia-reperfusion injury per se and in the development of cardioprotection. Among many other possible mechanisms, for example, in hyperlipidemia and diabetes, the increased increase in reactive oxygen and nitrogen species and the decreased ATP-sensitive potassium channel conductance in the ischemic heart may play a role in the reduced expression of cardioprotective factors and in aging, the reduced expression of ATP may direct more sympathetic signaling pathways and potentially interacting with the cardioprotective effect of preconditioning. The aim of this presentation is to review the mechanisms that are involved in the development of ischemia-reperfusion injury and the role of cardioprotection in the context of metabolic diseases such as hyperlipidemia, diabetes, obesity, and aging, and to discuss the potential role of these factors in the development of cardioprotection.

Keywords: preconditioning and postconditioning

ADENOSINE KINASE ACTIVATION CONTRIBUTES TO HYPERTENSION IN RAT MODEL

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We aim to characterize the spontaneously hypertensive stroke-prone (SHR-SP) and the normotensive Wistar-Kyoto (WKY) rat model of hypertension and its possible difference in their blood pressure and body weight. We observed the diameter changes of cerebral arteries, and the contractile force of the femoral artery. We found that the body and the heart weight, and also the blood

The body weight of the SHR-SP rats (255±22 g) was smaller than the weight of the WKY rats (361±23 g, $p < 0.001$). The heart body weight (0.0147 g) was smaller (230±12 mg) and aortic (161±10 mg) and blood pressure of the SHR-SP was significantly higher (140±10 mmHg) than the WKY rats (107±7 mmHg, 105±7 mmHg). The myogenic tone of the aorta was smaller in the two groups. After pretreatment with flow-chance inhibitor furosemide (10 µM), the myogenic tone decreased more in the SHR-SP group (82.5±5.2%) than in the WKY group (34.4±5.8%). Pretreatment with other substances, such as acetylcholine, norepinephrine, serotonin, and L-22566, we did not find any difference, not even after adding furosemide to the solution. In the case of the femoral artery, treatment with 100 mM KCl resulted in higher contractile force in SHR-SP (0.31±0.17 mN) than in WKY (0.25±0.13 mN). Pretreatment with furosemide resulted in the normalization of the higher contractile force in the SHR-SP group (decreased to 0.24±0.18 mN), but had no effect on the contractile force of the WKY group (0.25±0.13 mN). In case of more contractile force, we detected similar reaction, contraction for SHR-SP (0.71±0.16 mN), which decreased to 0.31±0.15 mN in the presence of furosemide, contraction for WKY: 0.71±0.16 mN, which was 0.31±0.15 mN after using furosemide.

According to our results, the RAS system probably plays an important role in the pathogenesis of high blood pressure and hypertension. Understanding of the pathogenesis may lead to new and better, fully novel effective drug development.

HOW I SUPPORT THE FAILING HEART IN SEPSIS

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Introduction: Sepsis and septic shock are the leading cause of death of patients admitted to the intensive care units. Sepsis presents a complex pathophysiological state with multiple organ dysfunction (MOD) and multiple organ failure (MOF). The failure presents the speed is multiorgan and clinical consequences of sepsis induced myocardial dysfunction, thus providing a better understanding of present and possible future therapeutic possibilities.

Discussion: Heart failure in sepsis is a not adequately enhanced cardiac output, which might be accompanied by systemic dysfunction, reduced heart rate variability and impaired baroreflex sensitivity. The extent of sepsis cardiomyopathy can be more or less quantified by taking the afterload into consideration, thus measuring the afterload-induced cardiac performance.

Reversible nature of myocardial dysfunction in sepsis supports the rather functional than anatomical character of the underlying pathophysiology. While initial hypotheses suggested the role of global myocardial ischemia, present findings implicate the role of cellular myocardial depression factors, such as, impaired cellular calcium homeostasis, mitochondrial dysfunction and apoptosis. Studies focusing on blocking myocardial depression factors like TNF or IL-1, the same as attempts to inhibit nitric oxide production could not prove any benefit. Studies of blocking the process of apoptosis through their phenotypic effects might turn out to be a potential therapy for sepsis-induced myocardial dysfunction, yet their applicability for this purpose in clinical practice still has to be proved. Considerable progress in understanding the complex mechanism of sepsis-induced myocardial dysfunction is needed to develop therapeutic targets. Nevertheless further research is needed to provide enough evidence for the application of these experimental developments in clinical setting. At present, the best treatment for sepsis-induced cardiac dysfunction still remains the administration of the sepsis focus and adequate hemodynamic support. Hemodynamic support should be goal-directed and should include