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RESEARCH AREA

Sepsis remains one of the leading causes of death in the intensive care units which necessitates the development of new diagnostic tools and novel, more efficient therapeutic possibilities. The basic problem in sepsis is the discrepancy between oxygen delivery and oxygen consumption which can lead to irreversible oxygen extraction deficit and energy shortage. The cornerstone of acute care should be to prevent, assess and treat oxygen debt globally. We propose that causative factors and signs of oxygen deficit have to be examined together on microcirculatory, cellular (endothelial) and subcellular (mitochondrial) levels in different shock-affected organs (e.g. the intestine and lung) by employing sufficiently long-term, clinically relevant experimental models. With this theoretical background, the major goal of our study is to find optimal, clinically applicable manoeuvres for microcirculatory recruitment and mitochondrial resuscitation to minimize the energy deficit of organs during the septic response.

TECHNIQUES AVAILABLE IN THE LAB

Our research laboratories are equipped with instruments to identify macro- and microcirculatory changes (hemodynamic computerized data-acquisition and analysis systems, laser-Doppler flowmetry, fluorescence-based intravital microscopy, orthogonal polarisation spectral imaging). Fluorescence confocal laser scanning endomicroscopy technique offers the possibility of acquiring precise *in vivo* data for histological analysis. A high resolution respirometer is available for examination of mitochondrial function (activities of the components of electron transport chain) and additional laboratory facilities (ELISA) to study inflammatory biomarkers. Animal house and fully-equipped operating theatres are available for surgical intervention of small (rats) and larger animals (minipigs).

SELECTED PUBLICATIONS

Poles, M.Z., Bódi, N., Bagyánszki, M., Fekete, É., Mészáros, A.T., Varga, G., Szűcs S., Nászai, A., Kiss, L., Kozlov, A.V., Boros, M., **Kaszaki, J.** (2018) Reduction of nitrosative stress by methane: Neuroprotection through xanthine oxidoreductase inhibition in a rat model of mesenteric ischemia- reperfusion. **Free Radic Biol Med 120:** 160-169.

Érces, D., Nógrády, M., Varga, G., Szűcs, S., Mészáros, A.T., Fischer-Szatmári, T., Cao, C., Okada, N., Okada, H., Boros, M., **Kaszaki, J.** (2016) Complement C5a inhibition improves late hemodynamic and inflammatory changes in a rat model of nonocclusive mesenteric ischemia. **Surgery 159:** 960-971.

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Boros, M., Ghyczy, M., Érces, D., Varga, G., Tőkés, T., Kupai, K., Torday, Cs., **Kaszaki, J.** (2012) The anti-inflammatory effects of methane. **Crit Care Med 40:** 1269-1278.

Kaszaki, J., Érces, D., Varga, G., Szabó, A., Vécsei, L., Boros, M. (2012) Kynurenines and intestinal neurotransmission – the role of N-methyl-D-aspartate receptors. **J Neural Transm 119:** 211-223.