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Non-invasive in vivo human model of the involvement of human epidermal mitochondria in the early post-ischaemic preconditioning
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Funding Acknowledgements: Grant “Nowelniejs z najlepszych 2.0” funded by the Ministry of Science and Higher Education.

Background: The repetitive ischemia and reperfusion may induce cell damage and tissue protection against the reperfusion-related injuries. This process, known as the post-ischaemic preconditioning (IPC), can be observed in different tissues and organs, including heart, muscles, or skin. During ischemia leading to cellular hypoxia, the amount of NADH gradually increases. During the reperfusion and restoration of oxygen, the amount of NADH drops down as it turns to NAD+ in the process of passing both hydrogen and electrons to oxygen within mitochondria. NADH can emit fluorescence light at the length of 460 nm; thus, by measuring such fluorescence, it is possible to quantify the amount of NADH. This study aimed to assess non-invasively the presence of IPC in the human skin by quantifying the flow-mediated skin fluorescence at the length of 460 nm (FMSF) at rest, during repeated ischemia and reperfusion episodes.

Methods: We studied 99 healthy people (23.6 ± 7.8 years old, 55% women) who underwent a non-invasive and in vivo measurement of the FMSF at rest, and then three times during 100-second brachial artery occlusions (blood pressure cuff inflated to the pressure 60 mmHg above each subject’s systolic blood pressure) producing forearm ischemia (isch), and the subsequent 10-minute reperfusion (Rep). To study IPC effects on the NADH, we compared the reperfusion-related changes in FMSF after the first and third episode of ischemia by measuring: (1) the reperfusion magnitude (RepM), (2) the contribution of the reperfusion to the total change in FMSF during ischemia and reperfusion (RepCont), and (3) the half-time of the recovery of FMSF to the baseline during the reperfusion (Rep). Results were compared by the paired nonparametric Wilcoxon test and presented as median and the 25th–75th percentile (IQR).

Results: Comparing with the first post-ischemic changes, the third ischemia caused a significant increase in the RepM from 14.6 (IQR: 12.3–18.2)% to 17.3 (IQR: 12.7–21.4)% (p < 0.0007), the RepCont from 64.0 (IQR: 55.1–80.8)% to 69.1 (IQR: 58.5–81.0)% (p = 0.0176), and a shortening of the 10-minute rep from 27.2 (IQR: 17.7–41.8) s to 22.1 (IQR: 15.3–40.0) s (p < 0.0087).

Conclusions: We show in an in vivo human model that the short repetitive ischemia and reperfusion episodes may produce acute changes in FMSF corresponding to the post-ischemic preconditioning. In more details, the IPC is accompanied by the significantly stronger and faster post-ischemic recovery of the skin fluorescence to the baseline level. Further physiological and clinical studies are required to explore and better understand this model, including the effects of non-pharmacological and pharmacological treatment.

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Evaluation of anti-arrhythmic efficacy of the IKI inhibitor PA-6 in domestic dogs with chronic atrial fibrillation
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Funding Acknowledgements: Supported by an Advances in Veterinary Research grant (AVR-15-05) from the dept of Clinical Sciences of Companion Animals, Utrecht University.

Background: The inward rectifier inhibitor pentamidine analogue 6 (PA-6) is effective in cardioversion of anaesthetised goats with persistent rapid pacing induced atrial fibrillation (AF) and is not pro-arrhythmic in anaesthetised experimental dogs with chronic third-degree AV block. However, efficacy and safety in the clinical setting is unknown.

Purpose: To test efficacy and safety of PA-6 in converting AF to sinus rhythm (SR) in awake dogs with chronic atrial fibrillation.

Methods: To test efficacy and safety of PA-6 in converting AF to SR in awake dogs with chronic atrial fibrillation.

Results: Twenty-six dogs (modal 5, range 4–6) suffering from chronic atrial fibrillation were treated with oral oral L-arginine supplementation on endothelial function, insulin resistance, adiponectin level in hypertensive females with rheumatoid arthritis and its correlation with l-arginine aspartate
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Hypertension (HT) and rheumatoid arthritis (RA) are regarded as conditions associated with higher risk for cardiovascular disease. L-Arginine improves endothelial function and thereby is expected to be used in the prevention of cardiovascular disease. In addition, data exists that L-arginine can reduce insulin resistance and affects on the exchanger of adipose tissue. We aimed to evaluate the effects of oral L-arginine supplementation on endothelial function, insulin resistance, adiponectin level in HT females compared with RA. 112 females with mean age – 53.8 [49.6; 57.5] years were enrolled. Pts were randomized to study subgroup (n=58) received L-Arginine aspartate 30 mlday during 4 weeks in addition to standard treatment. Control subgroup pts (n=54) received only the standard treatment. The levels of adiponectin and insulin were measured in ELSA test. Insulin resistance was estimated using HOMA2 index. Endothelial-dependent flow mediated vasodilation (EDV) by D. Celemjer method was performed. All measuring were done at baseline and after 4 weeks. Statistical analysis included non-parametric methods with p value < 0.05.

EDV improved by 59.9% (p<0.001) in L-Arginine treatment group, in compare in control group – by 21.7% (p<0.05). Endothelial function had been normalized in 70,7% (p<0.03) and 18,5% (p<0.04) pts study and control group respectively. After 4 week L-Arginine supplementation the level of insulin resistance was significantly decreased by 10,4% (p<0.01) in the treatment group. Pts with EDV achieving ≥ 10% HOMA2 index decreased by 29.6% (p<0.02). The adiponectin level was significantly increased by 12.3% (p=0.001) in L-Arginine treatment group.