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Aims: Coronary perfusion decreases with aging and hypertension. Several effects of aging and hypertension on the resistance artery wall have been described, but their network remodeling is less described. Thus we aimed to quantitatively analyze the characteristic of aging- and hypertension-induced alterations of the coronary resistance artery network. **Methods:** Seven old and 8 young male rats, and 7 chronically Ang II infused hypertensive and 8 control female rats were investigated. The whole resistance artery network of the left anterior descendent coronary artery was prepared, in situ perfused with Krebs solution, and recorded by videomicroscopy. The network was divided into branching units and into segments, these latter were further divided into 50 micrometer ring units. A coordinate system was positioned above the heart, and the locations, directions and diameters of the ring units were determined. Their statistical distribution on the heart surface was statistically analyzed. **Results:** Broken course of the main branch and multiple branching are typical findings both in the aging and in the hypertensive networks. Large branches frequently originate at high angle, small branches at low angle, features rarely seen in normal, young networks. Dilation of the branches close to the orifice was accompanied by a reduction of the number of small diameter components (rarefaction). The diameter/frequency plot characteristically altered, with many similarities in the both groups. **Conclusion:** It seems to be characteristic, that in both situations originally thin branches thickened, and originally thick branches thinned, their courses in the network deviating from the typical young pattern. These morphological alterations may cause the decreased ventricular wall perfusion typical for the aged and for the hypertensive. Hypertension significantly reduced the network structure of these vessels. The observed alterations elevate the hemodynamic resistance and can be evaluated as an adaptive process to the hypertension.

P1

PROJECTION NEURONS WITH DISTINCT AXON-TRAJECTORIES IN THE LATERAL SPINAL NUCLEUS AND LAMINA I OF THE LUMBAR SPINAL DORSAL HORN OF RATS

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Aims: We intended to label and trace the axons of projection neurons in the lateral part of lamina I and in the lateral spinal nucleus (LSN). **Methods:** For patch-clamp recordings, we injected intracellular biocytin into lamina I and into LSN neurons of young adult rats. The recently described IR-LED illumination technique applied for the visual identification of neurons allowed us to use intact spinal cord preparations of the lumbar enlargement, preserving complete axonal trees within the specimen. Biocytin labelled neurons were detected with the avidin-biotin horseradish-peroxidase method and reconstructed in 3D by using Neurolucida. **Results:** Although projection neurons in the lateral part of lamina I and in the LSN show a remarkable morphological variability, on the basis of their axon-trajectories, the recovered neurons could be grouped into four distinct categories. The majority of labeled neurons gave rise to a single projection axon that originated from the

cell body or one of the primary dendrites, crossed the midline in the anterior commissure and ascended in the contralateral anterolateral tract (ALT). The main axon of a subset of these neurons gave rise to two long axon collaterals one of which ascended and the other descended in the ipsilateral dorsolateral funiculus. A second set of neurons sent their main axon to the contralateral ALT through the posterior commissure. The third set of neurons provided ipsilateral ascending projection. The main axon ran in the posterior commissure and after bypassing the central canal returned to the ipsilateral ALT through the anterior commissure, and gave short collaterals to the deeper laminae on both sides of the spinal cord. **Conclusion:** Axon trajectories of projection neurons in the lateral part of lamina I and in the LSN share common morphological features. Axon collaterals of these projection neurons may form propriospinal synaptic contacts contributing to the processing of nociceptive information.

L1

CB1 RECEPTOR ANTAGONISTS IN THE THERAPY: DEAD OR ALIVE?

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CB1 receptor (CB1R) antagonists were among the most promising molecules of pharmacological research in the last decade. They have been explored and found to be effective as therapeutic agents for obesity and related cardiometabolic problems, however, use of rimonabant, the first marketed CB1R antagonist, has been suspended due to its anxiogenic and depressive side effects with potential suicide risk. The development of several other CB1R antagonists, like otenabant, surinabant and taranabant, all of them already in clinical trials, were interrupted. Since almost all other antiobesity drugs, like dexfenfluramine or sibutramine which act through other mechanism of action were also suspended earlier, the unmet need for drugs that reduce body weight became enormous. The possibilities of the safe use of CB1R antagonists including also further developments will be presented here. One approach is the use of CB1R antagonists that poorly cross the blood brain barrier. These compounds do not affect behavioural responses mediated by CB1R in the brains of mice with genetic or diet-induced obesity, but do cause improvements in glucose homeostasis, fatty liver, and plasma lipid profile without sustained reduction in body weight (Tam et al, J Clin Investigation, 2010). The other approach is to use personalized medicine, namely genomics, to identify individuals who may benefit from treatment with centrally acting CB1R without psychiatric side effects (Lazary et al, Trends Pharmacol Sci *in press*, doi: 10.1016/j.tips.2011.02.013)