7. THE IMPACT OF THE 3 ENDOTHELIUM DERIVED GASSES (H2S, NO, CO) ON VASCULAR ENDOTHELIUM HOMEOSTASIS

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Introduction. Vascular endothelial fulfills a lot of functions having a decisive role in the control of circulatory and general homeostasis. Among the endothelium derived factors three gasses occupy a special position being tightly connected to the intrinsic system orchestrating vascular homeostasis.

Aim of study. To assess the role of nitric oxide (NO), hydrogen sulfide (H2S) and carbon monoxide (CO) in the regulation of native endothelial functions.

Methods and materials. It has been selected and analyzed 30 recent articles from Google discussed this approach.

Results. NO is synthesized from L-arginine under the action of endothelial nitric oxide synthase (NOS3). Its main role consists in vascular muscular media relaxation due to formation of cGMP in smooth myocytes. Thus, NO provides the vasorelaxant effect of acetylcholine, bradykinin, adenosine and also mediates the impact of hemodynamic stress in order to equilibrate the blood flowing in systole and diastole. NO decreases the expression of pro-inflammatory cytokines, and this effect is connected to antioxidant system boosting. Likewise, NO reduces the oxi-LDL passing in neointima and expression of MCP-1 (monocyte chemoattractant protein) resulting in atherosclerosis mitigation. Another important effect of NO is blunting of smooth myocytes hypertrophy and their migration when secretory phenotype appears. Endothelial H2S is produced from L-cysteine and also has a vasorelaxant effect provided by 2 mechanisms: hyperpolarization and decreased expression of phosphodiesterase. In addition to antiplatelet and antithrombotic effects H2S stimulates angiogenesis, reduces smooth vascular myocyte proliferation and damaging impact of hyperglycemia, hyperhomocysteinemia and hypercholesterolemia. Remarkably, NO augments the H2S production due stimulation of L-cysteine uptake, and the decrease of their circulating level is occurring in patients with arterial hypertension and diverse kinds of endothelial injury. Endothelial CO is generated by two heme-oxygenase enzymes and demonstrates many similar effects to NO and H2S. CO independently dilates small arteries, but also interplays with NO in the process of vascular relaxation, because of increased expression of NOS3. CO stimulates angiogenesis, confines inflammatory response due to nuclear factor expression inhibition and vascular cell apoptosis due to mitochondrial cytochrome c leakage reduction.

Conclusion. NO, H2S and CO have many common beneficial effects regarding the native endothelial homeostasis, such as: vasodilatation, antioxidant, anti-inflammatory, antiplatelet, antithrombotic, antiproliferation, anti-remodeling.