



2. CORONARY PERFUSION: REGULATION AND MECHANISMS OF DISORDERS

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Introduction. Coronary perfusion is a complex phenomenon ensuring the myocardial supply with oxygen and nutrients which is dependent on 2 major factors, such as myocardial compliance and feasibility of coronary arteries adequately to response on natural vasoconstricting and vasorelaxant agents. The coronary perfusion impairment could be either acute or chronic leading to development respectively of acute coronary syndrome (ACS) and chronic ischemic disease.

Aim of study. To reveal the main tools of coronary perfusion regulation and the key pathogenic mechanisms of coronary dysfunction.

Methods and materials. Was analyzed a material reflected in 20 recent articles found in Google.

Results. The crucial tool of coronary perfusion control is based on coronary Gregg phenomenon which embraces the capacity of coronary arteries to dilate on action of vasorelaxant factors mediated by endothelium, such as acetylcholine, bradykinin, adenosine, and prostacyclin. The main endothelium derived mediators are nitric oxide (NO), hydrogen sulfide, carbon oxide and endothelial hyperpolarizing factor which also confine the coronary constricting effect of norepinephrine, endothelin-1 and angiotensin II. Likewise, Vanhoutte coronary phenomenon providing coronary dilation due to hyperpolarization by epoxyeicosatrienoic acid (derivate of arachidonic acid) is also an important tool of coronary perfusion regulation. Moreover, it has a significant benefit in comparison with the Gregg phenomenon inasmuch does not depend on endothelium feasibility. Myocardium compliance or diastolic rigidity is tightly linked to its lusitropic function and quality of remodeling. Coronary atherosclerosis is considered as the main cause of coronary dysfunction, mainly based on endothelium dependent coronary reactivity impairment. When an atherosclerotic plaque obstructs an epicardial coronary artery more than 75% ACS develops and the worst clinical entity being acute myocardial infarction. Chronic coronary disease in the majority of cases is the result of decreased production of NO and negative coronary artery remodeling especially of subendocardial arterioles. Likewise, myocardial hypertrophy and increased fibrosis of extracellular matrix are significant pathogenic factors contributing to coronary perfusion diminution.

Conclusion. Endothelium dependent coronary perfusion is the physiological axis of normal hearts. Its disorder in association with coronary artery remodeling and myocardium diastolic rigidity represent main factors of coronary dysfunction. Meanwhile, the Vanhoutte coronary phenomenon dependent on hyperpolarization becomes a compensatory mechanism.