

The significance of coronary flow reserve in chest pain syndromes

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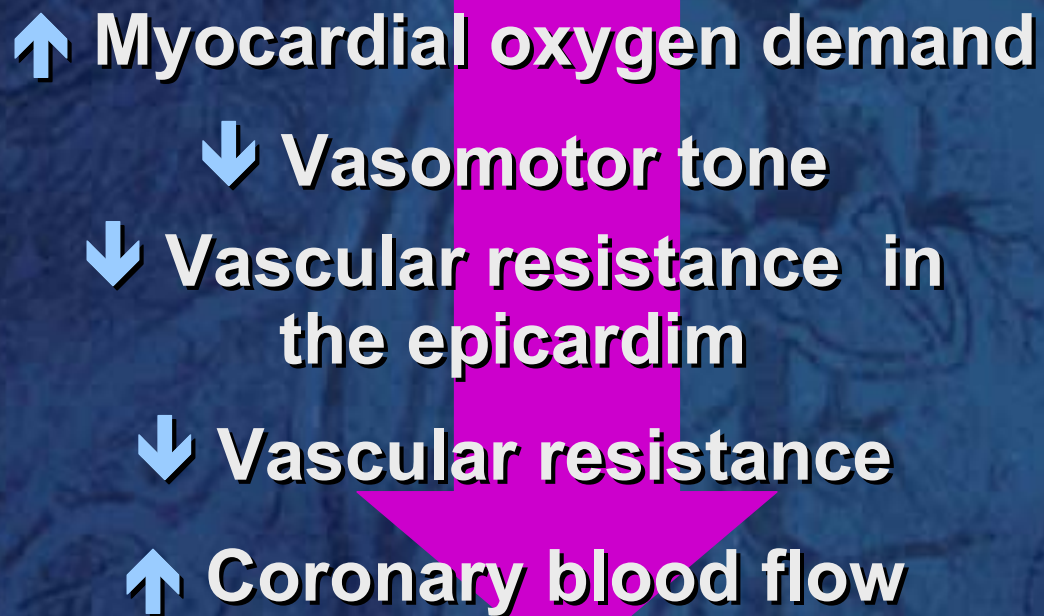
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Coronary blood flow regulation



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The increase in coronary blood flow occurring with augmented myocardial oxygen demands is regulated by changes in the vascular resistance of the coronary arteries.

The ability to increase coronary blood flow in response to vasoactive mechanisms is coronary flow reserve.

Coronary vascular resistance

**Epicardial coronary arteries contribute : 5 %
intramyocardial coronary arterioles:**

< 300 μm in diameter: 95%

< 100 μm : more than 50%

**Changes in microcirculation may result in
dramatic alterations in coronary flow and
coronary flow reserve, provoking ischemia**

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The major epicardial coronary arteries contribute only about 5% to the total vascular resistance. The intramyocardial coronary arterioles are responsible for the majority of coronary resistance

Major mechanisms of coronary flow regulation

Endothelial: arterioles 80-150 μm diam/epicardial arteries

vasoactive substances like nitric oxide, prostaglandins, endothelium-derived hyperpolarizing factor (EDHF), endothelin

Metabolic: arterioles 25-100 μm diam

adenosine, major metabolite that mediates metabolically induced coronary vasodilation during myocardial ischemia

Myogenic: arterioles 50-100 μm diam

intrinsic property of vascular muscle: vascular smooth muscle contraction or relaxation: vasodilation due to potassium ion efflux via ATP sensitive potassium channels – it is important, since myogenic constriction and dilation occur during autoregulation

Neurohumoral: arterioles, epicardial arteries 140-300 μm arteries

vasoactive substances like nitric oxide, prostaglandins, endothelium-derived hyperpolarizing factor (EDHF), endothelin

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Microvascular angina pectoris: Dysfunction of coronary arterioles.

Disturbance of coronary microcirculation based on impaired blood flow in small ($< 200\ \mu\text{m}$) intramural arteriolar resistance vessels or in coronary capillary system, or both

↑ coronary vascular resistance



coronary blood flow does not normally increase



↓↓ significant impairment in coronary flow reserve

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The term „microvascular angina pectoris” was proposed by Cannon and Epstein in 1985 for the symptoms „angina pectoris + positive ergometry test + epicardial coronary arteries without stenosis”

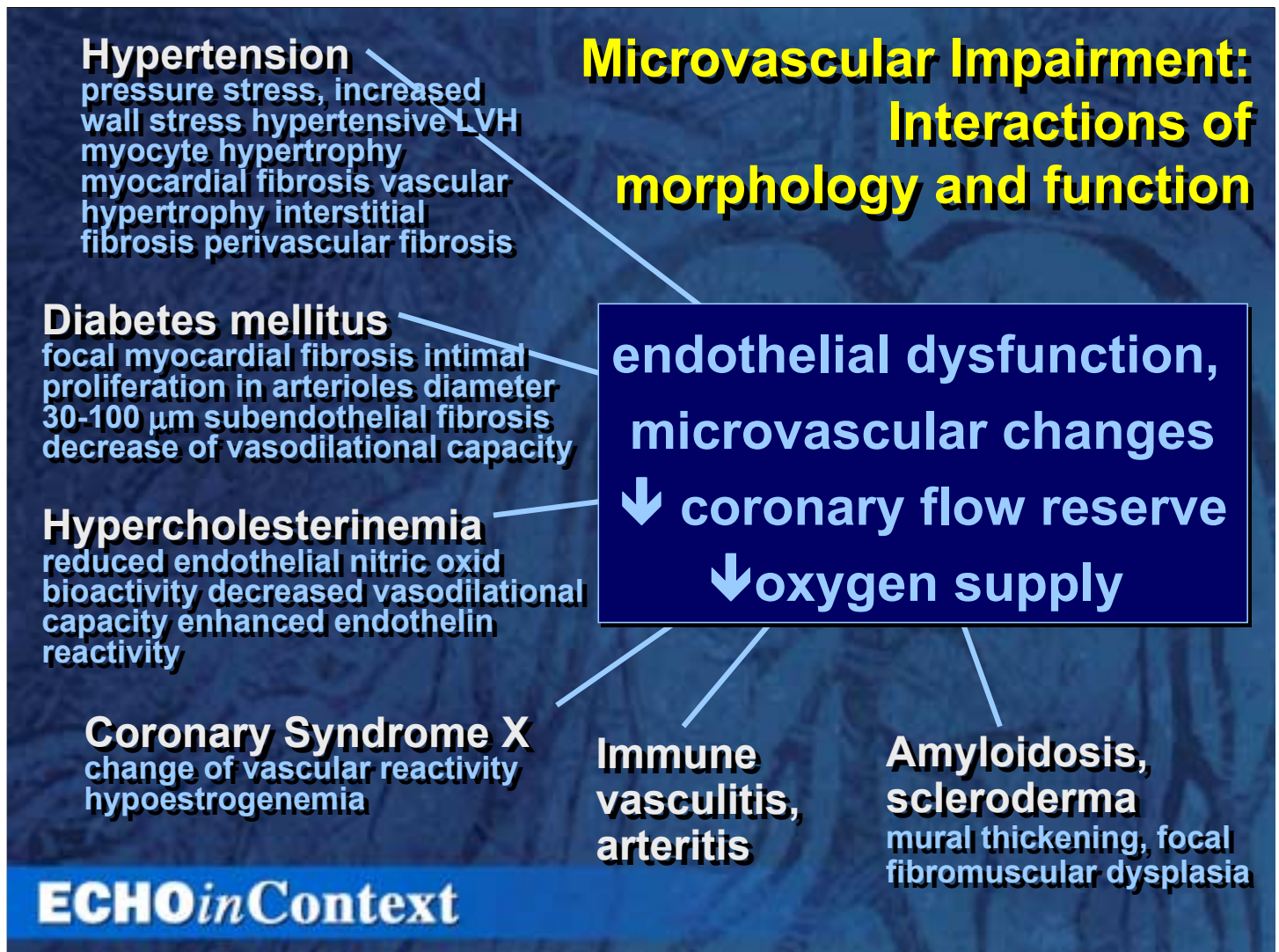
Functional causes of disturbances in coronary microcirculation

Vascular hypertensive microangiopathy
diabetic microangiopathy systemic
collagen diseases, immune vasculitis
posttransplantation vasculopathy

Metabolic diabetic endothel dysfunction
hyperlipoproteinemia disturbance of
oxygen diffusion and transport

Rheologic paraproteinemia polyglobulia, polycythemia
hyperlipoproteinemia

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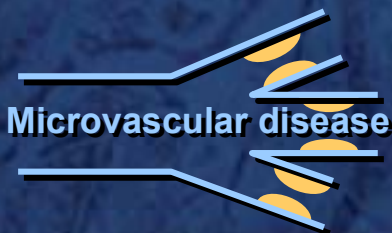
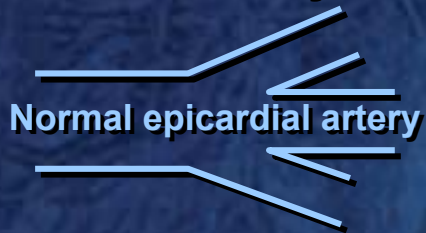


Microvascular impairment – interactions of morphological and functional changes

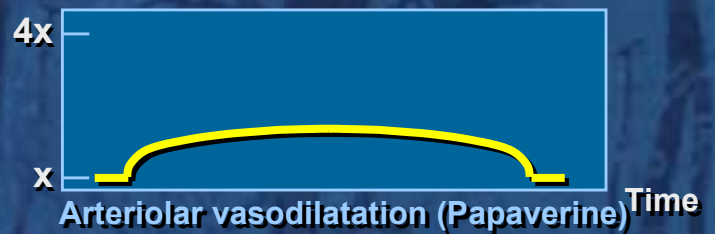
microvascular impairment is prominent in patients with angina pectoris, manifested in systemic hypertension, in diabetic patients, in cases of other metabolic and rheologic disorders as a result of endothelial dysfunction, vascular remodelling, changes in vascular reactivity and cardiac muscle hypertrophy.

Correlation of coronary anatomy and physiology: The concept of coronary flow reserve

Anatomy



Physiology



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In the presence of normal epicardial arteries and normal microvasculature, the CFR is normal. Severe flow limiting epicardial stenosis or microvascular pathologic state of the coronary arterioles result the diminution of the CFR.

Methods for determining CFR

Invasive

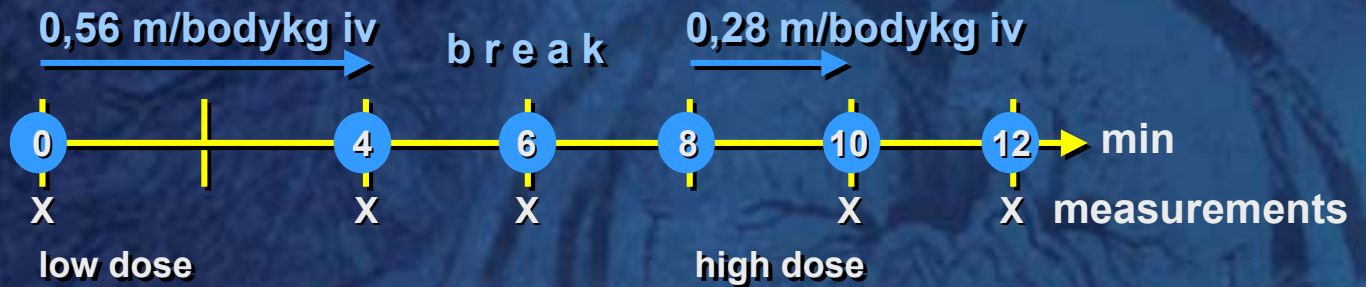
- *timed venous collections from great cardiac veins*
- *thermodilution catheters*
- *electromagnetic flowmeters*
- *intravascular Doppler flowmeters – measure coronary flow velocities, which are proportional to flow quantity*
CFR = *maximal flow velocity / basal flow velocity*
- *quantitative digital subtraction angiography DSA*
CFR = *hyperemic / initial density of contrast medium*

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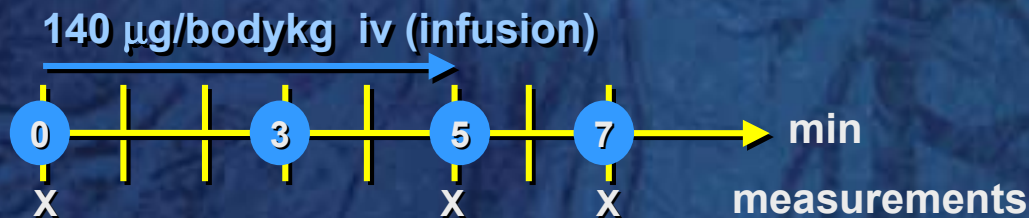
A normal coronary flow reserve is approximately four to five. With methods measuring not absolute coronary blood flow, but relative changes in perfusion or flow velocities the values are lower. The CFR is influenced by age, heart rate, preload, use of vasoactive pharmacological agents.

Methods to attain vasodilation

Dipyridamol



Adenosine

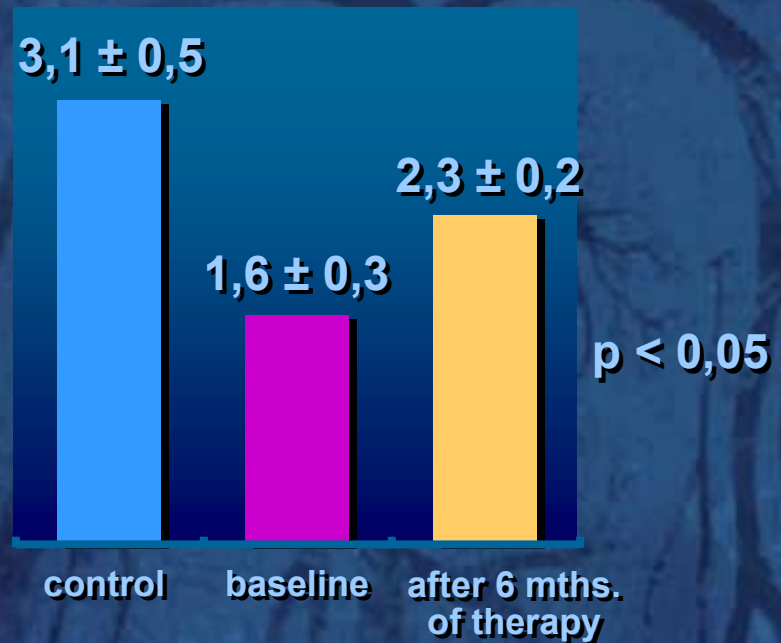


CFR in Hypertension and LVH

14 patients,
all male



mean age 43 yrs

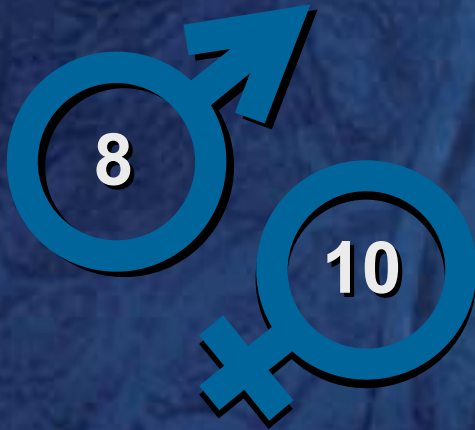


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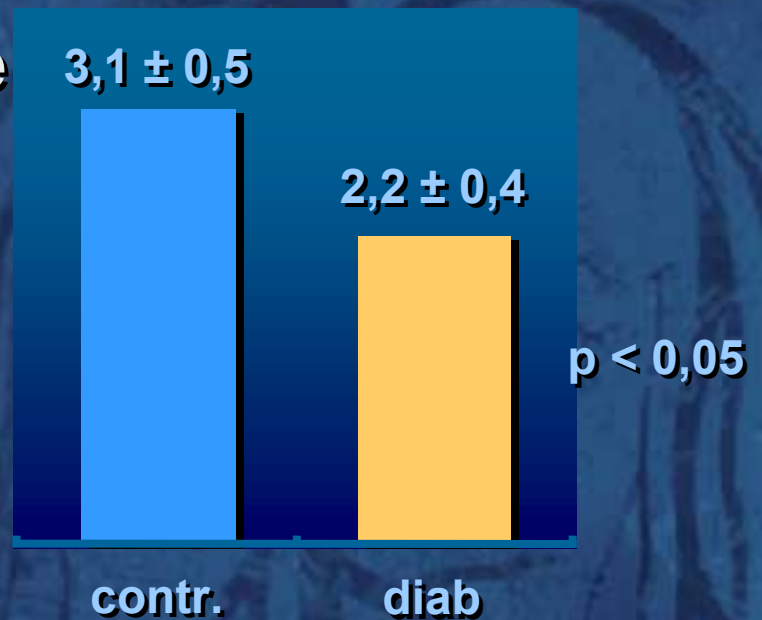
In a group of patients with systemic hypertension and left ventricular hypertrophy (pts with valvular disease or known CHD and diabetes mellitus were excluded) the CFR was reduced. 6 months after effective antihypertensive therapy with ACE-inhibitor or Ca-antagonist the repeated CFR increased significantly, although it did not reach the normal value.

CFR in Diabetes

18 patients,
8 male, 10 female



mean age 56 yrs

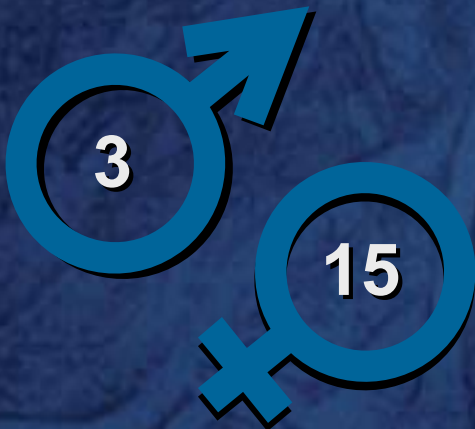


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A group consisting of 18 pts with diabetes mellitus was examined (exclusion criteria were hypertension, left ventricular hypertrophy, evidence of valvular or coronary heart diseases) with the method of TEE using Dipyridamole. The measurements confirmed significantly reduced CFR.

CFR and perfusion in angina and normal coronary angiograms

18 patients,
3 male, 15 female



mean age
51 yrs

Pathological – Decreased
CFR 2.25 ± 0.2 + perfusion
disorder

- inhomogeneous distribution
- reversible perfusion decrease
- reverse redistribution

Normal - Normal
CFR 3.20 + minor
inhomogeneity

Normal - Normal
CFR 3.25 +
normal perfusion

CFR 3.20

CFR 3.25

CFR 2.25

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In 18 pts coronary angiography showed no substantial alterations despite chest pains and positive ergometry test. The perfusion was also determined through stress myocardial scintigraphy SPECT examination. In 15 of 18 pts the CFR had reduced distinctly (2.25), perfusion disorders in all of these pts have been observed, especially inhomogeneity of perfusion and reverse redistribution.