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This presentation includes only the most important terms and facts. Its content by itself is not a sufficient source of information required to pass the Physiology exam.



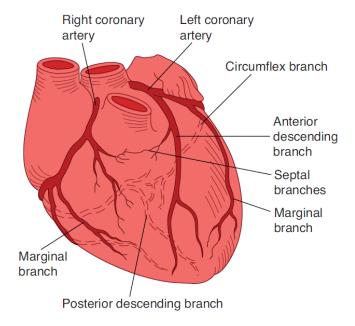
#### • a. cor. sinistra

85% of the blood flow (the frontal part of septum, the conductive system, majority of the left ventricle)

#### • a. cor. dextra

(the right ventricle, the posterior part of septum and usually also the posterior part of the left ventricle)

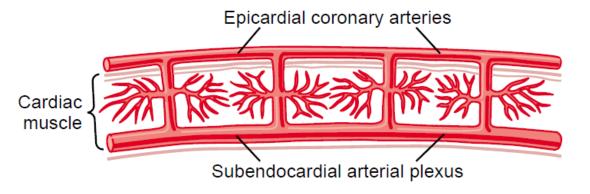
- O<sub>2</sub> diffusion directly from the blood situated in the cardiac cavities
- placing of coronary arteries and capillaries in the cardiac walls



Ganong's Review od Medical Physiology, 23<sup>rd</sup> edition



- epicardial coronary arteries supply most of the muscle
- intramuscular arteries (smaller) penetrate the muscle
- plexus of subendocardial arteries



Guyton and Hall. Textbook of Medical Physiology, 11<sup>th</sup> edition

 During systole, blood flow through the plexus of subendocardial arteries is reduced (compression of intramuscular arteries) – compensated through extra vessels in the plexus (sensitivity to coronary ischemia).

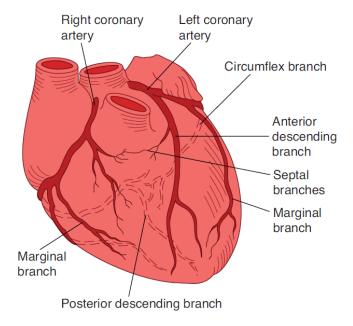
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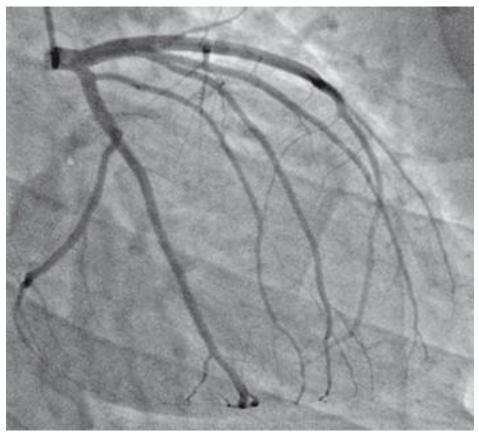
(the right ventricle, the posterior part of septum and usually also the posterior part of the left ventricle)

- O<sub>2</sub> diffusion directly from the blood situated in the cardiac cavities
- placing of coronary arteries and capillaries in the cardiac walls
- coronary angiography



Ganong's Review od Medical Physiology, 23<sup>rd</sup> edition





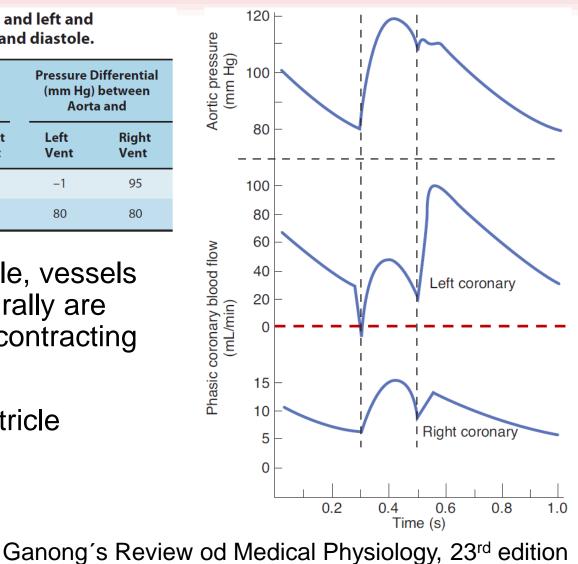
http://pochp.mp.pl/aktualnosci/show.html?id=55102



#### **TABLE 34–4**Pressure in aorta and left andright ventricles (vent) in systole and diastole.

|          | Pressure (mm Hg) in |              |               | Pressure Differential<br>(mm Hg) between<br>Aorta and |               |
|----------|---------------------|--------------|---------------|---|---------------|
|          | Aorta               | Left<br>Vent | Right<br>Vent | Left<br>Vent  | Right<br>Vent |
| Systole  | 120                 | 121          | 25            | -1  | 95            |
| Diastole | 80                  | 0            | 0             | 80  | 80            |

- during the systole, vessels situated intramurally are pressed by the contracting myocardium
- left vs. right ventricle
- high heart rate

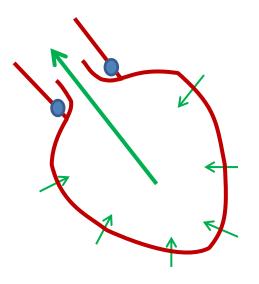


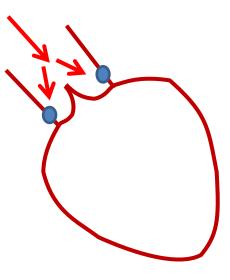


• orificia of the coronary arteries

ejection

isovolumic relaxation







- O<sub>2</sub> extraction is almost maximal already at rest, capillaries are open
- The only possibility how to increase O<sub>2</sub> supply (for example during exercise) is the coronary vasodilation!



Control of coronary blood flow

1) reduction/interruption of the blood flow or increased demands (exercise, increased blood pressure)

hyperaemia (reactive or active) based on the metabolic vasodilation mediators:  $\downarrow pO_2$ ,  $\uparrow pCO_2$ ,  $\downarrow pH$ ,  $\uparrow [K^+]_e$ , adenosine, bradykinin, prostaglandins, NO



Control of coronary blood flow

- 2) the neural regulation of the vessel diameter secondary impact
  - a) indirect effects
  - b) direct effects

(mostly opposite)



Control of coronary blood flow

- 2) the neural regulation of the vessel diameter secondary impact
  - a) indirect effects

sympathetic system (NE, E)

 $\uparrow$  HR + contractility  $\rightarrow$  rate of cardiac metabolism  $\rightarrow$  increased O<sub>2</sub> consumption  $\rightarrow$  activation of local vasodilating mechanisms

parasympathetic system (ACH) opposite changes  $\rightarrow$  vasoconstriction



Control of coronary blood flow

- 2) the neural regulation of the vessel diameter secondary impact
  - a) indirect effects
  - b) direct effects vasospastic sympathetic system (NE, E) vasocardial ischemia epicardial vessels – mostly  $\alpha$ -rec.  $\rightarrow$  vasoconstriction intramural vessels – mostly  $\beta$ -rec.  $\rightarrow$  vasodilation parasympathetic system (ACH)

vasodilation, but not significant (only few fibers)



Control of coronary blood flow

- 2) the neural regulation of the vessel diameter secondary impact
  - a) indirect effects
  - b) direct effects

Whenever the direct effects alter the coronary blood flow in the wrong direction, the metabolic control overrides them within seconds!



- the resting blood flow: 225 ml/min (4-5% of CO)
- at physical exertion:
  - cardiac output increased 4-7fold \_\_\_\_\_ cardiac work may higher afterload -
  - higher afterload
  - coronary blood flow increases only 3-4fold! -
  - efficiency of the cardiac utilization of energy has to increase to make up for the relative deficiency of coronary blood supply



#### **Cardiac Muscle Metabolism**

- at rest: 70% of energy fatty acids
- anaerobic/ischemic conditions: anaerobic glycolysis high glucose consumption + high quantities of formed lactic acid (one of causes of the ischemic pain + ↓pH)
- severe ischemia: degradation of ATP to ADP, AMP and, finally, to adenosine  $\rightarrow$  loss of adenosine into circulation through sarcolemma  $\rightarrow$  vasodilation

lost adenosine replaced by new synthesis of adenine, but very slowly (2% per hour)

Major cause of death of cardiomyocytes during ischemia is the adenosine deprival! (30 min of severe ischemia may cause irreversible changes and cell death)



#### **Coronary Reserve**

- ability of coronary vessels to adapt blood flow to the actual cardiac work (ergometry)
- the maximal blood flow / the resting blood flow
- reduction of the coronary reserve:
  - relative coronary insufficiency (too high resting demands, high resting blood flow cannot be sufficiently

increased)

 absolute coronary insufficiency (~ coronary heart disease) (the stenotic arteriosclerotic process)

Reduced coronary reserve is a limiting factor of the cardiac output, thus, also of the effort of organism!

