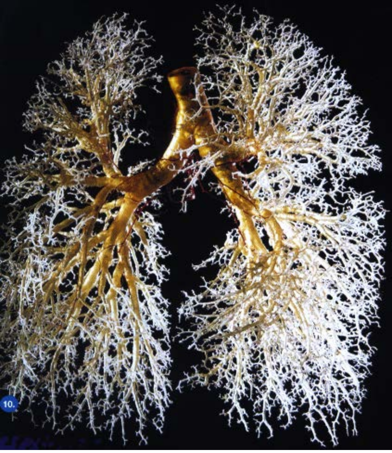


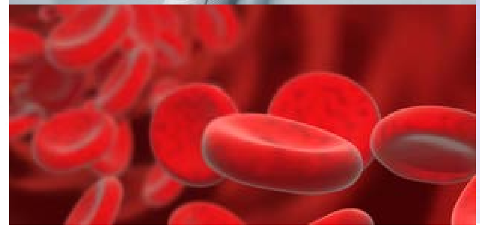

Biological and Medical Fluid Mechanics I

5. The cardiovascular system: the heart

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5. The cardiovascular system: the heart	Exercise
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5.1.1 Tasks and supply of the heart

- **task:** provision of the mechanical energy for the maintenance of the blood stream in the different circulation sections with:
  → convenient pressure level
  → sufficient volume flux
- **structure:**
  → two pump chambers (**ventricles, right** and **left**) that are **hydraulically connected in series**

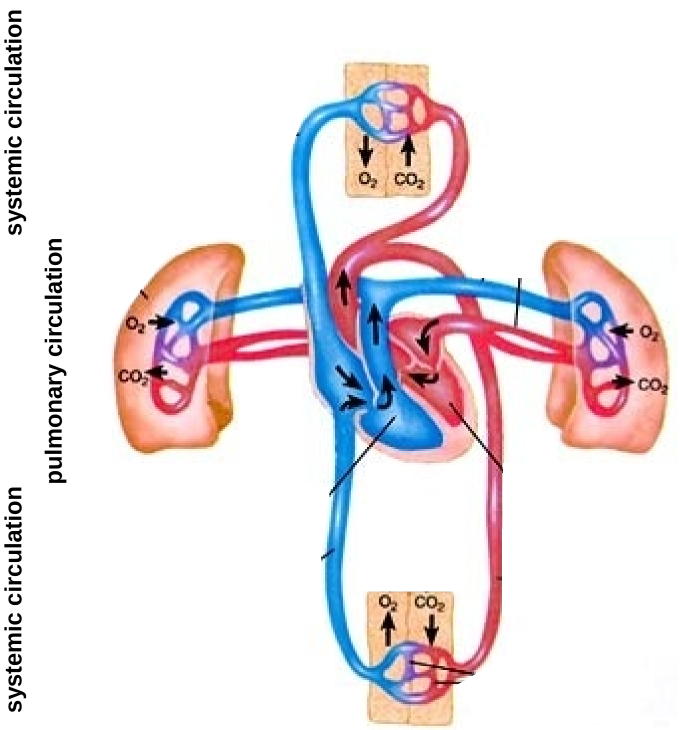

Fig. 5.1: The blood circulation system [1]

→ each chamber has an **inlet** and **outlet valve** that helps to **ensure one-way flow** of blood by opening (let blood through) and closing (prevent backflow)

5.1.1 Tasks and supply of the heart

→ each chamber has a pre-chamber (**atrium , right** and **left**)

→ the pump chambers differentiate in their form and muscle weight, which adapt to the consecutive loads (pressure):

  → **left ventricle:** stronger hollow muscle, ellipsoid form

  → **right ventricle:** thin wall hollow muscle, sickle form, which partly surrounds the left ventricle

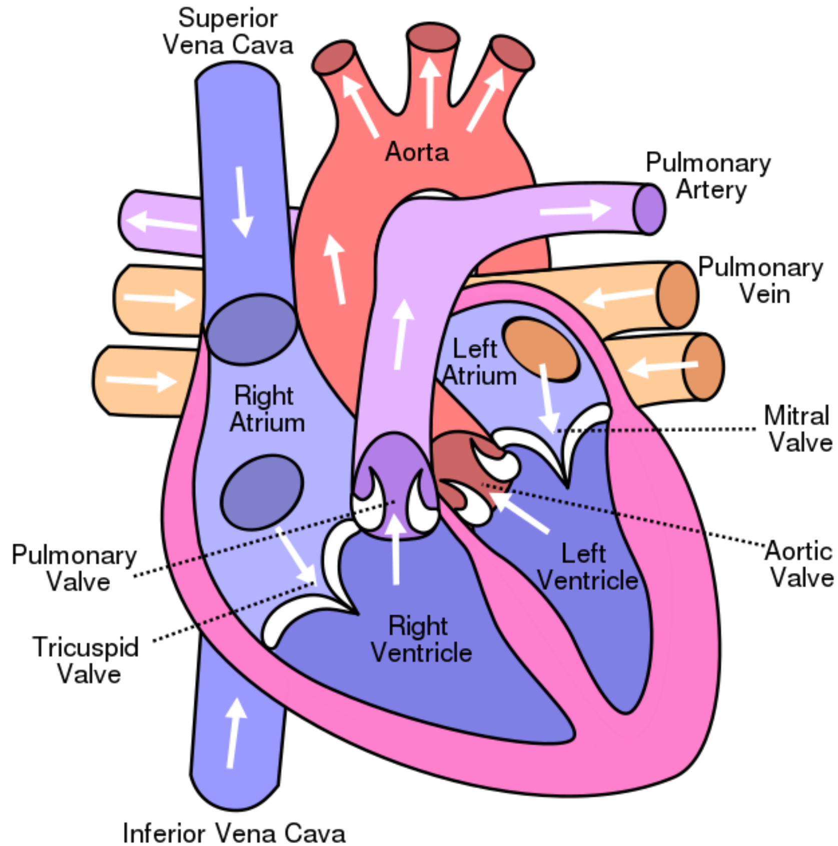

Fig. 5.2: Scheme of the human heart [2]

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5.1.1 Tasks and supply of the heart

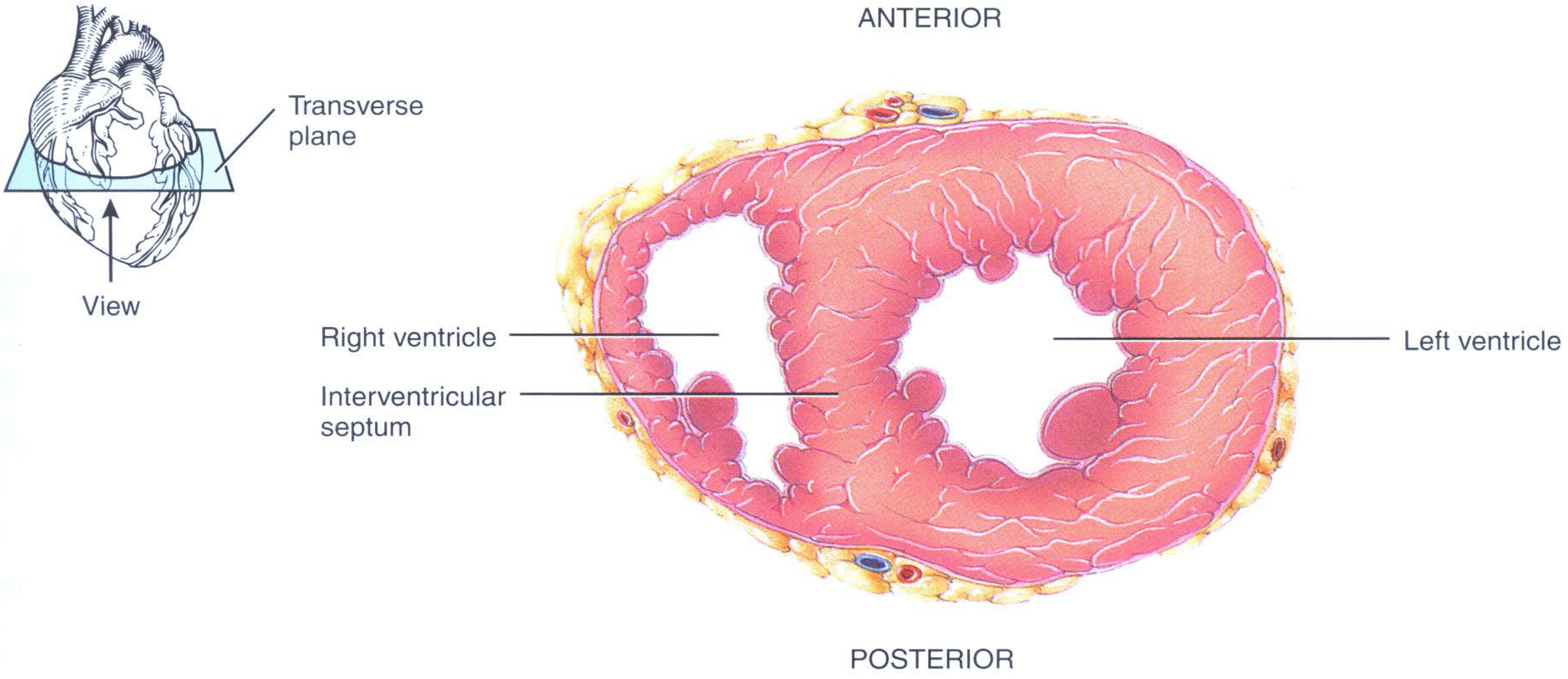

Fig. 5.3: Cut through the chamber plane [3]

5.1 The heart

5.1.1 Tasks and supply of the heart

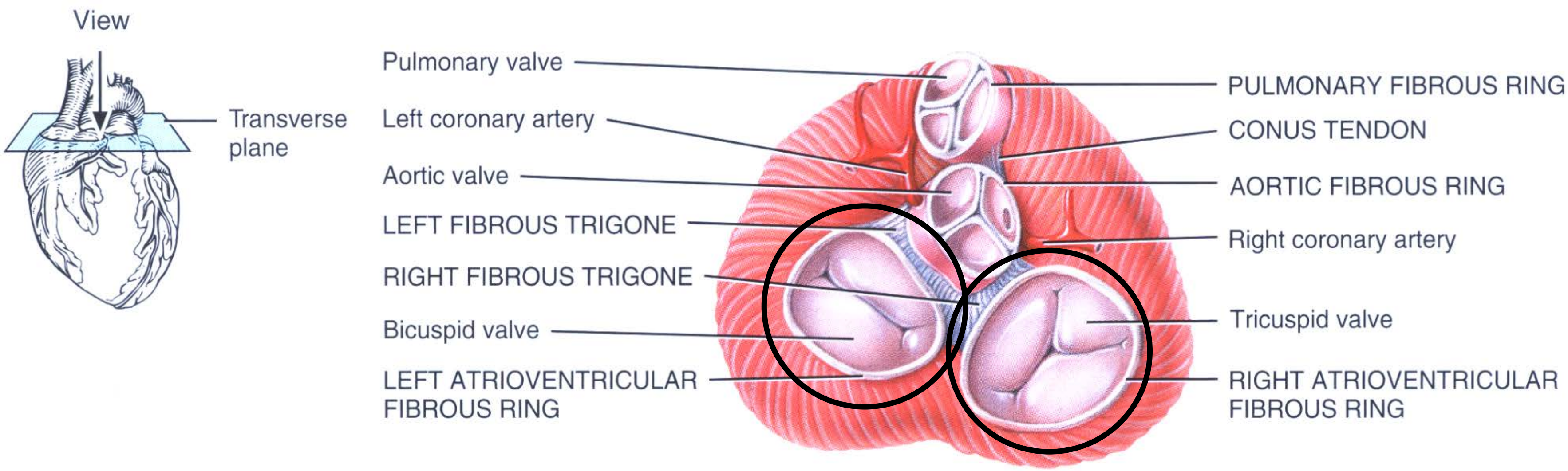

Fig. 5.4: Cut through the valve plane (inlet valves marked) [3]

- **inlet valves:** → Tricuspid valve ***(three cusps)*** **,** Bicuspid valve **(*two cusps)***

  → atrioventricular valves

5.1.1 Tasks and supply of the heart

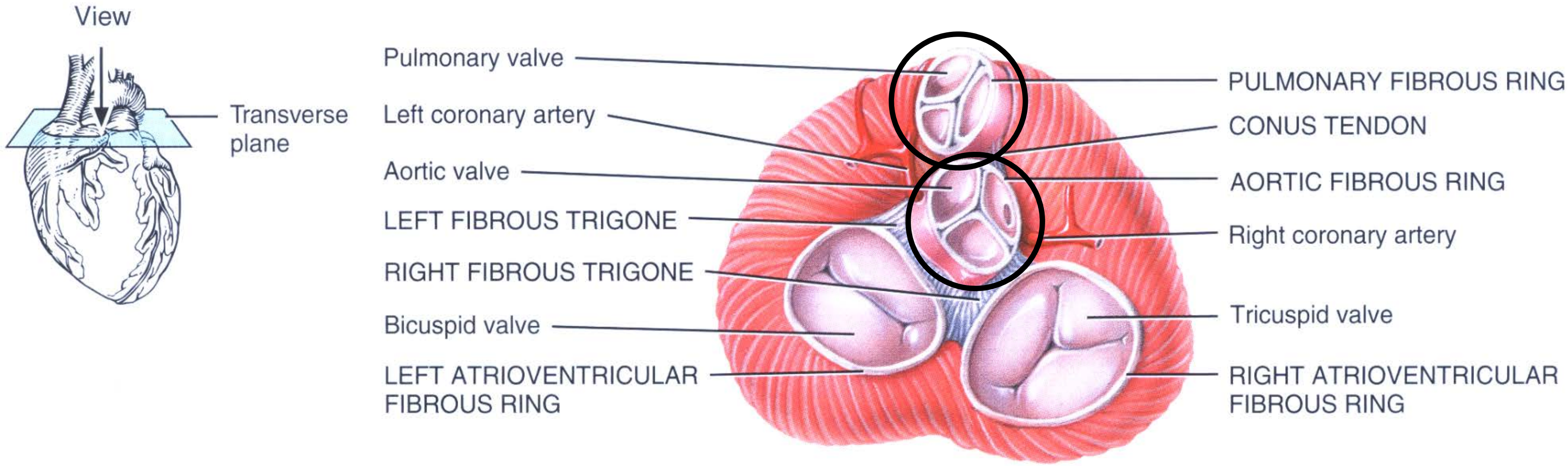

Fig. 5.5: Cut through the valve plane (outlet valves marked) [3]

- **outlet valves:** → aortic valve, pulmonary valve
  → semilunar valves
  → each valve has ***three cusps***

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5.1.2 Volume and pressure in the heart during heart contraction

- **contraction of the heart:**
  1. "initial action" **triggered** from the **sinusoidal node** (autonomic)
     → node lies in the opening of the vena cava superior in the right atrium
     → electrical impulse is **transmitted to the atrio-ventricular node** through the muscle of the atria
     → **contraction of the atria**

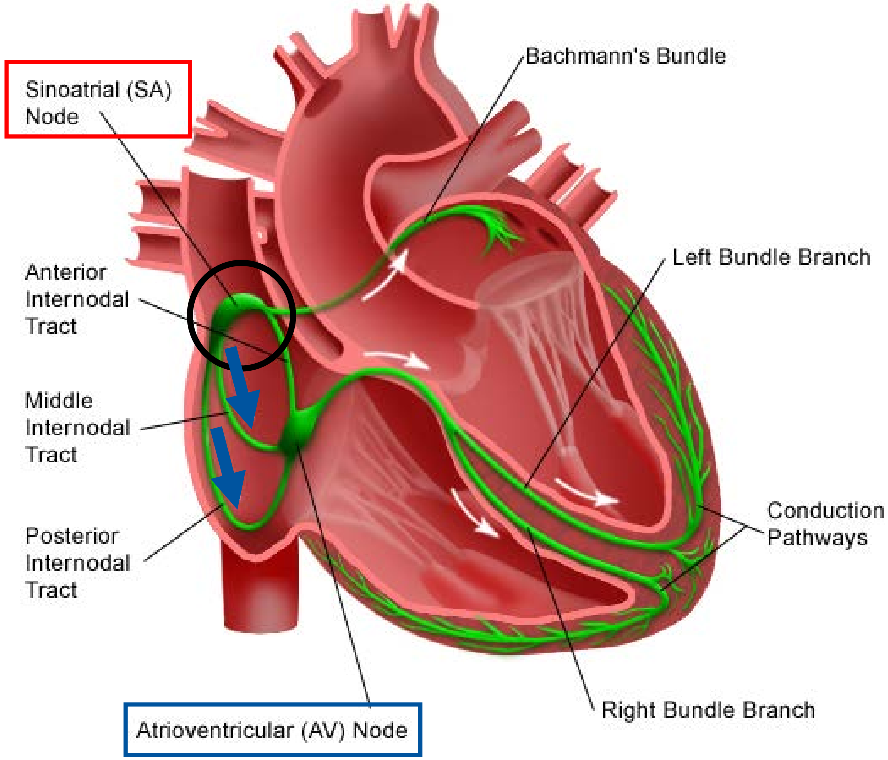

Fig. 5.6: Propagation of the electrical impulse through the heart [4]

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5.1.2 Volume and pressure in the heart during heart contraction

- **contraction of the heart:**

  2. electrical impulse is **transmitted to the ventricle musculature**

     → contraction wave of the ventricles starts with a small delay from the ventricle apex and travels up to get to the atria

  - impulse expands relatively slowly over the whole heart in **~ 20msec**
  - continuous fiber contraction (peristaltic) to the outflow pathway

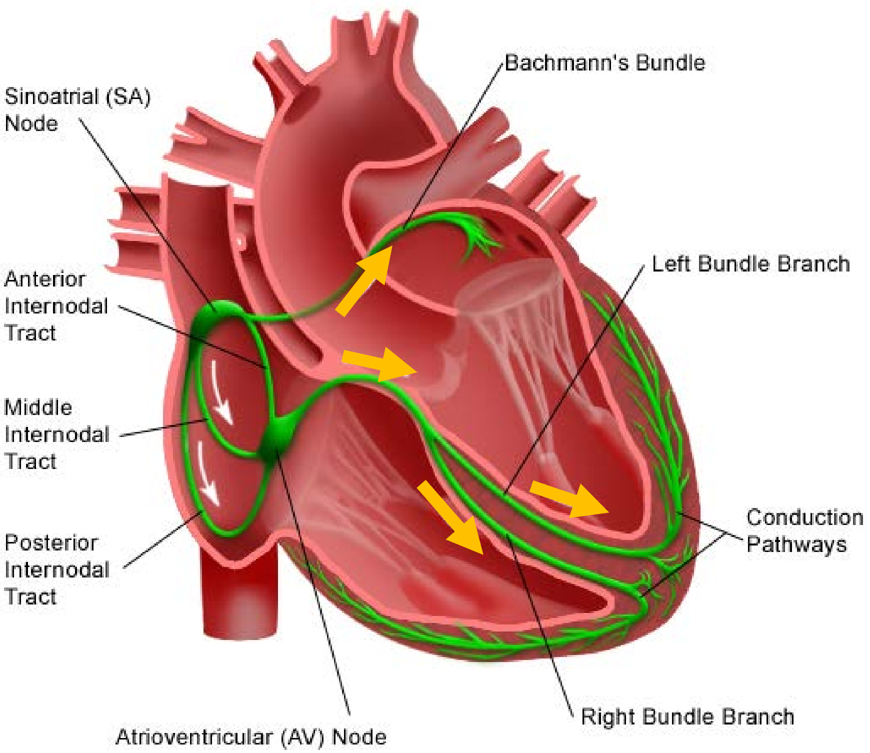

Fig. 5.6: Propagation of the electrical impulse through the heart [4]

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5.1.2 Volume and pressure in the heart during heart contraction

- **Volume and pressure course:**

  1. **Isovolumetric contraction:**

     → beginning of the ventricle contraction

     → musculature is tensed

     → **pressure in the ventricle rises**

     → **no volume change in ventricles**

     → when the ventricular pressure exceeds the aortic/pulmonary arterial pressure ($p_{ventricular} > p_{aortic/pulmonary}$) the **aortic/pulmonary valve open**

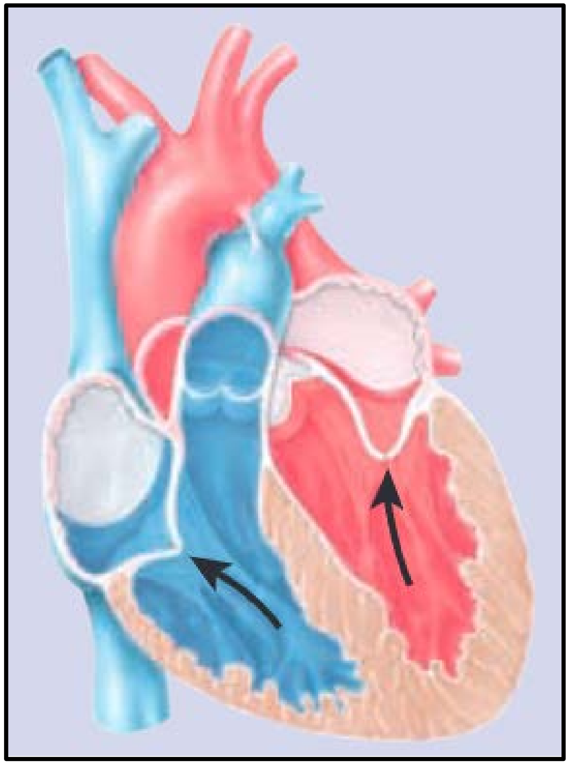

Fig. 5.7: Isovolumetric contraction [3]

5.1.2 Volume and pressure in the heart during heart contraction

- **Volume and pressure course:**

  2. **Ventricular ejection:**

     → ventricle **volume decreases fast**

     → flow is delayed

     (on both sides: about 70ml of blood is ejected)

     → remaining volume in each ventricle: about 60ml

     (end-systolic volume, see chapter 5.1.3)

     → pressure in left ventricle: up to 120mmHg

     → pressure in right ventricle: up to 25-30mmHg

     → when $p_{ventricular} < p_{aortic/pulmonary}$

     the **aortic/pulmonary valve closes**

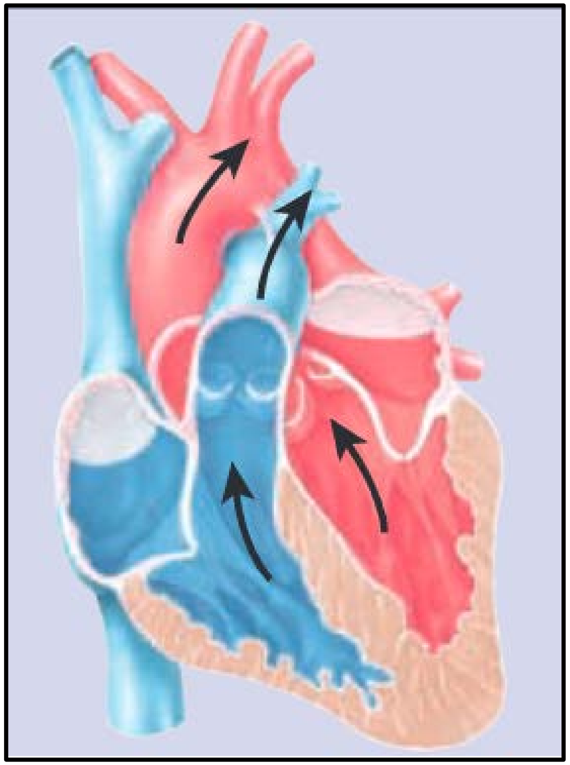

Fig. 5.8: Ventricular ejection [3]

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5.1.2 Volume and pressure in the heart during heart contraction

- **Volume and pressure course:**

  3. **Isovolumetric relaxation:**

     → all four valves are closed

     → **no volume change in ventricles**

     → **atria pressure** is **slightly increased**

     → **ventricle pressure** steeply **decreases** (protodiastole) under the atria pressure until the kinetic energy is exhausted and flow reversion starts

     → when $p_{ventricular} < p_{atrial}$ the **AV-valves open** (atrio-ventricular: bicuspid/tricuspid valves)

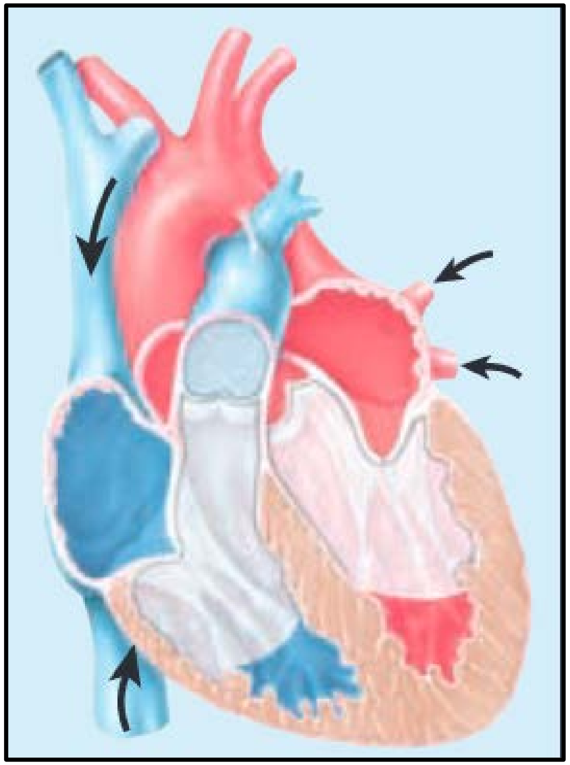

Fig. 5.9: Isovolumetric relaxation [3]

5.1.2 Volume and pressure in the heart during heart contraction

- **Volume and pressure course:**

  4. **Ventricular filling:**

     → ventricular **volume increases fast** (fast filling phase)

     → after 25% of the diastolic period, the chamber is normally filled to 80%

     → “Diastase” = slowly further filling of the ventricle

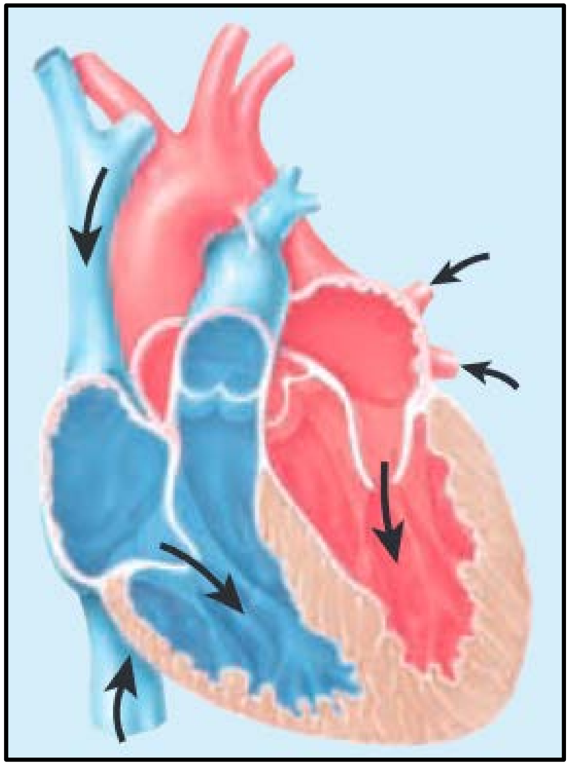

Fig. 5.10: Ventricular filling [3]

5.1.2 Volume and pressure in the heart during heart contraction

- **Volume and pressure course:**

  5. **Atrial contraction:**

     → atria contract and exert pressure on the blood within, which forces blood through the open AV valves into the ventricles

     → further filling of the ventricles is combined with minor pressure decrease in the ventricle

     → at higher heart frequencies: atria contraction follows immediately the fast filling phase

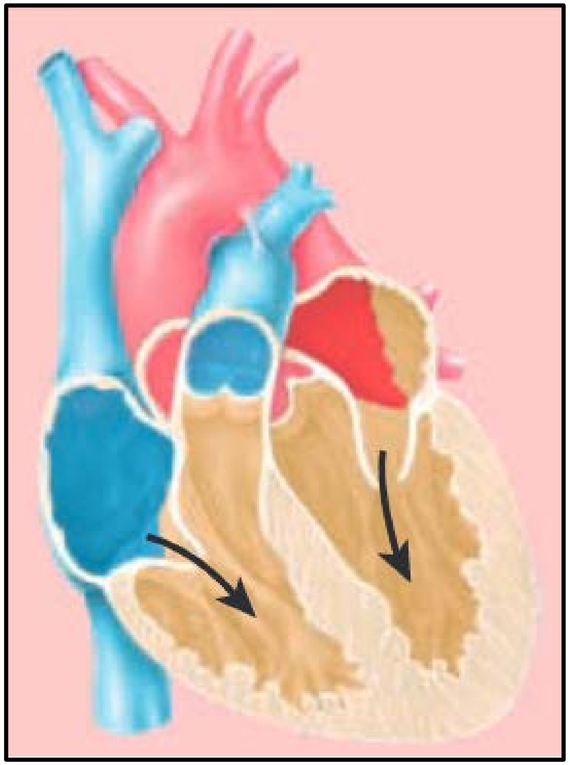

Fig. 5.11: Atrial contraction [3]

5.1.2 Volume and pressure in the heart during heart contraction

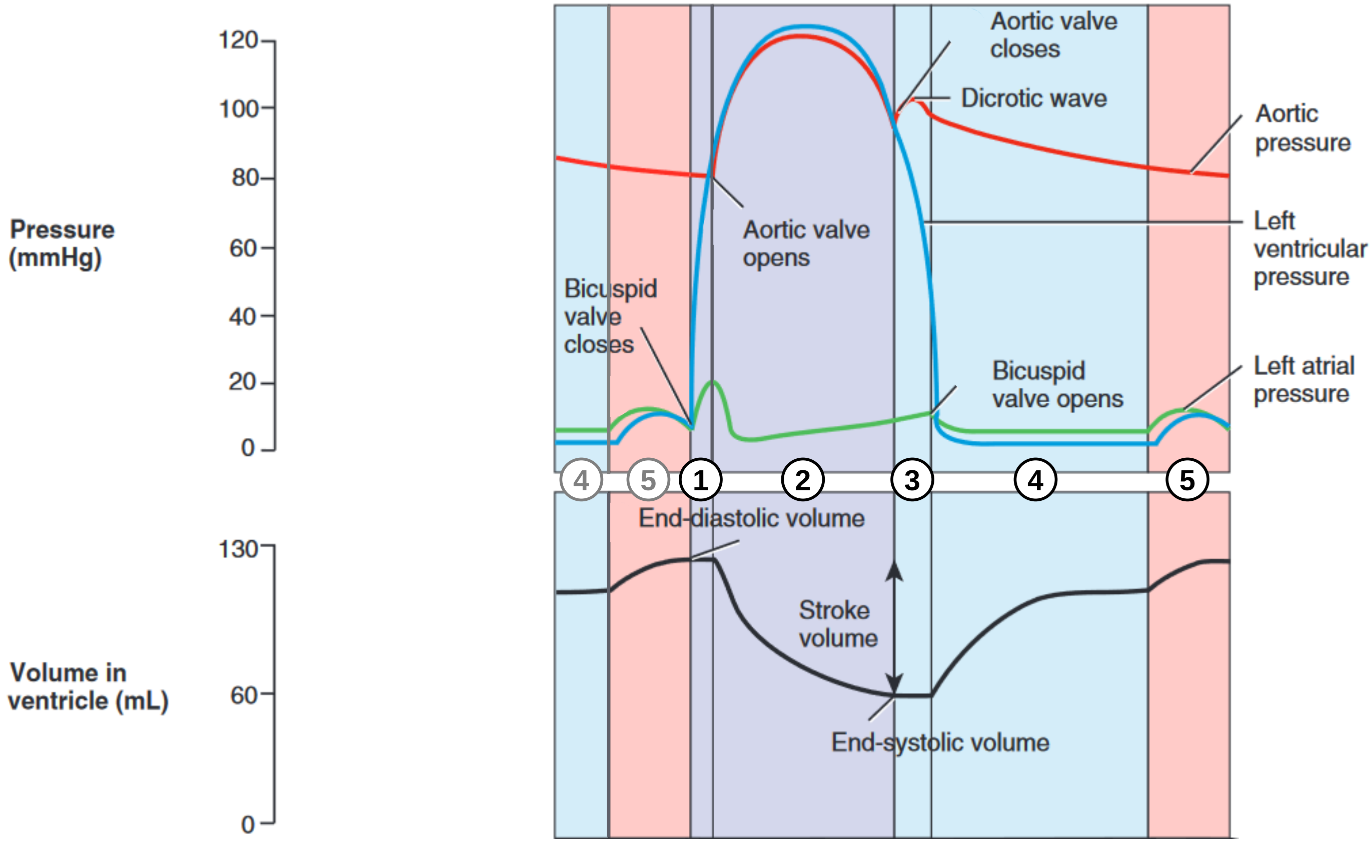

Fig. 5.12: Pressure and volume during the cardiac circle [3]

5.1.2 Volume and pressure in the heart during heart contraction

- **Heart sounds:**

→ result primarily from blood turbulence caused by the closing of the heart valves

→ smoothly flowing blood is silent

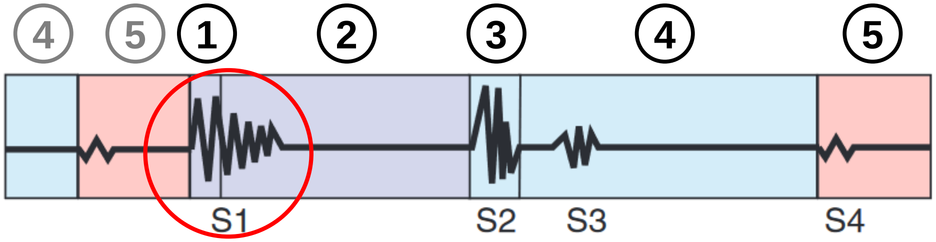

Fig. 5.13: Heart sound s during the cardiac circle [3]

→ **S1:** closing the cuspid valves, isovolumetric contraction of the ventricles

→ vibration of the cusps and the chamber wall

→ lubb sound

→ louder and a bit longer than the second sound (S2)

5.1.2 Volume and pressure in the heart during heart contraction

- **Heart sounds:**

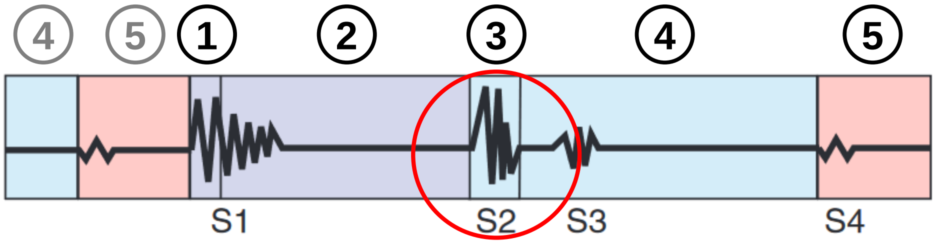

Fig. 5.13: Heart sound s during the cardiac circle [3]

→ **S2:** closing of the semilunar valves

→ caused by blood turbulence associated with closure of the SL valves at the beginning of ventricular diastole

→ shorter and not as loud as the first sound (S1)

→ dupp sound

5.1.2 Volume and pressure in the heart during heart contraction

- **Heart sounds:**

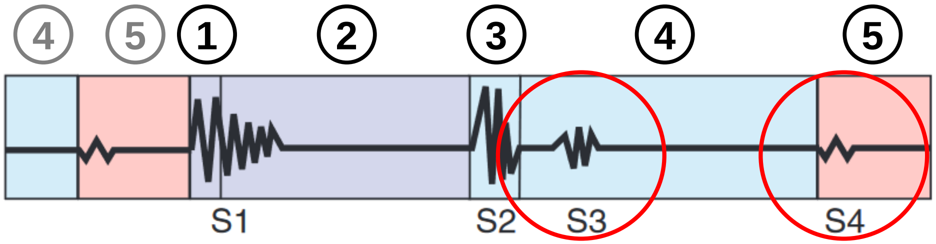

Fig. 5.13: Heart sound s during the cardiac circle [3]

→ **S3:** due to blood turbulence during rapid ventricular filling

→ **S4:** due to blood turbulence during atrial systole

→ normally **only the heart sounds S1 and S2 are loud enough to be heard**!

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5.1.3 Important heart measurements and their normal values

- stroke volume of ventricles: $V_s = 70\ cm^3$
- maximum stroke volume : $V_{s,max} \approx 140\ cm^3$
- end-diastolic ventricular volume: $V_{diast} = 120\ cm^3$
- end-systolic ventricular volume: $V_{syst} = 50\ cm^3$

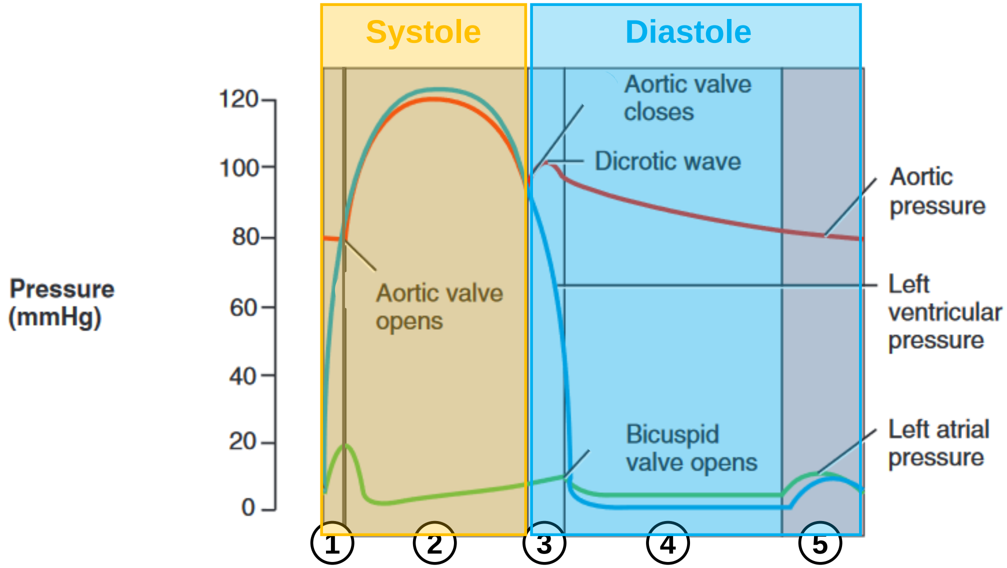

Fig. 5.14: Systole and Diastole [3]

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5.1.3 Important heart measurements and their normal values

• stroke frequency:	$f_s = 70\ min^{-1}$
• maximum frequency:	$f_{s,max} \approx 200\ min^{-1}$
• pulse-width Systole / Diastole:	$\lambda = 0{,}76 - 1{,}2$
• heart minute volume:	$V = 5 - 28\ l/min$
• end-diastolic ventricular pressure (left):	$p_{dia,l} = 2 - 8\ mmHg$
• end-systolic ventricular pressure (left):	$p_{sys,l} = 120\ mmHg$
• end-diastolic ventricular pressure (right):	$p_{dia,r} = 0 - 4\ mmHg$
• end-systolic ventricular pressure (right):	$p_{sys,r} = 25\ mmHg$
• pressure rise velocity (left):	$\dot{p}_{maxLV} = 1800\ mmHg/s$
• pressure rise velocity (right)	$\dot{p}_{maxRV} = 150\ mmHg/s$

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5.1.4 Regulation mechanisms of the heart

- the heart is composed of two pump units connected in series, that are bounded together by a throttle and capacity
- both pumps have to adapt to the same stroke volume with high accuracy
- otherwise: **overload or depletion of the lung circulation**!
- already a small difference from 5,0 l/min (left ventricle) to 5,1 l/min (right ventricle) leads in a short time to death
- adaptation of the heart on the circulation needs an autonomous („intrinsic“) mechanism because of the „extrinsic“ regulation by the central nervous system

→ **Frank-Starling mechanism**

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5.1.4 Regulation mechanisms of the heart

- **Frank-Starling mechanism:**
  - **based on special properties of the cardiac muscle fibers:**
    - → greater preload (stretch) prior to contraction increases force of contraction
    - → within limits, the more the heart fills with blood during diastole, the greater the force of contraction during systole
    - → preload is proportional to the end-diastolic volume (EDV)
  - equalizes the output of right and left ventricles
  - keeps the same volume of blood flowing to both the systemic and pulmonary circulations:
    - → if left side of the heart pumps a little more blood than the right side, the volume returning to the right ventricle (**venous return**) increases

5.1.4 Regulation mechanisms of the heart

- **Frank-Starling mechanism:**

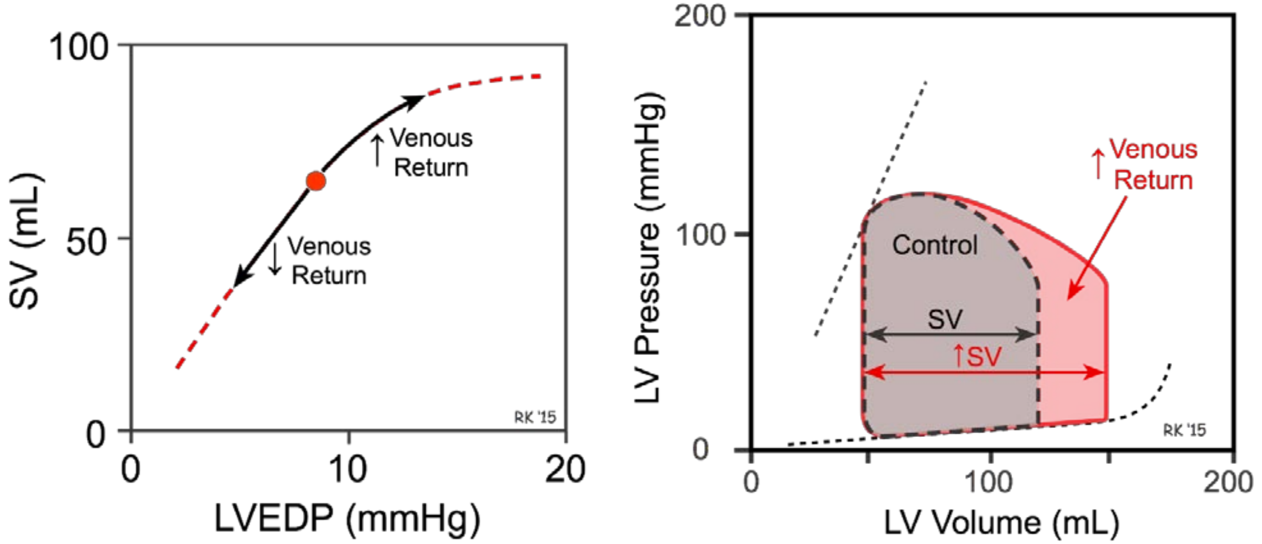

Fig. 5.15: Frank-Starling mechanism: venous return [5]

→ the **increased EDV** causes the **right ventricle to contract more forcefully** on the next beat, bringing both sides back to balance (homeostasis)

5.1.4 Regulation mechanisms of the heart

- **excursus: elementary contraction forms of the muscle:**

→ **isometric:** muscle force (“tension”) varies while the length of the muscle remains constant (in cardiac muscle, this represents an isovolumetric contraction, because the length of the muscle determines the atria or ventricular volumes)

→ **isotonic:** length of the muscle changes while the muscle force remains constant (in cardiac muscle, this also represents isobaric contraction, because the muscle force determines the atrial or the ventricular pressure)

→ **auxotonic:** muscle length and force both vary simultaneously

5.1.4 Regulation mechanisms of the heart

- **excursus: elementary contraction forms of the muscle:**

  → an isotonic or auxotonic contraction that builds on an isometric one is called an **after-loaded contraction**

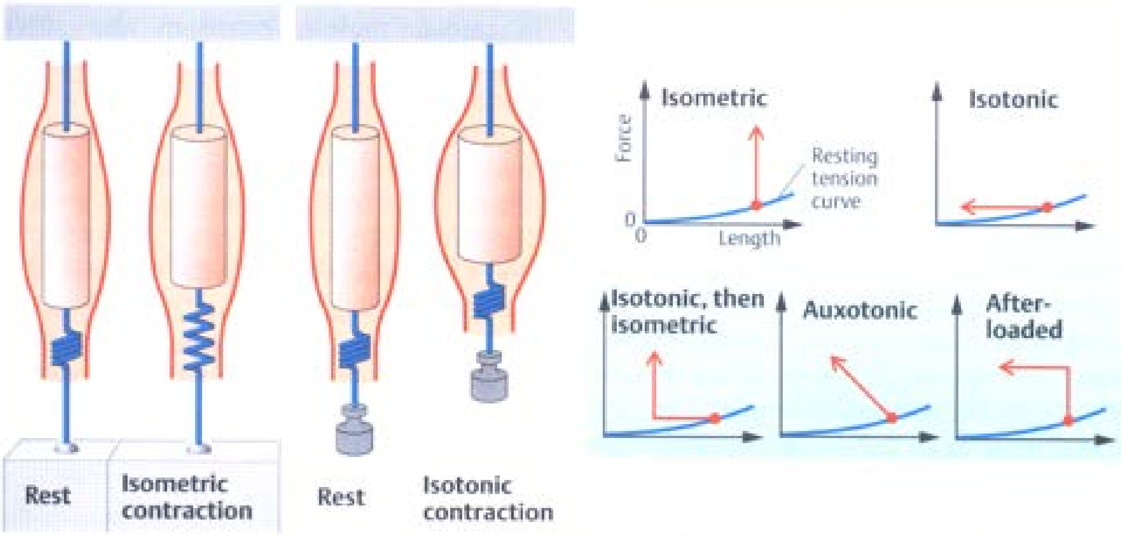

Fig. 5.16: Types of muscle contraction [6]

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Sources

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Thank you for your attention!