

WHICH IS THE ROLE OF MYOCARDIAL TRABECULAE?

M.L. Costantino

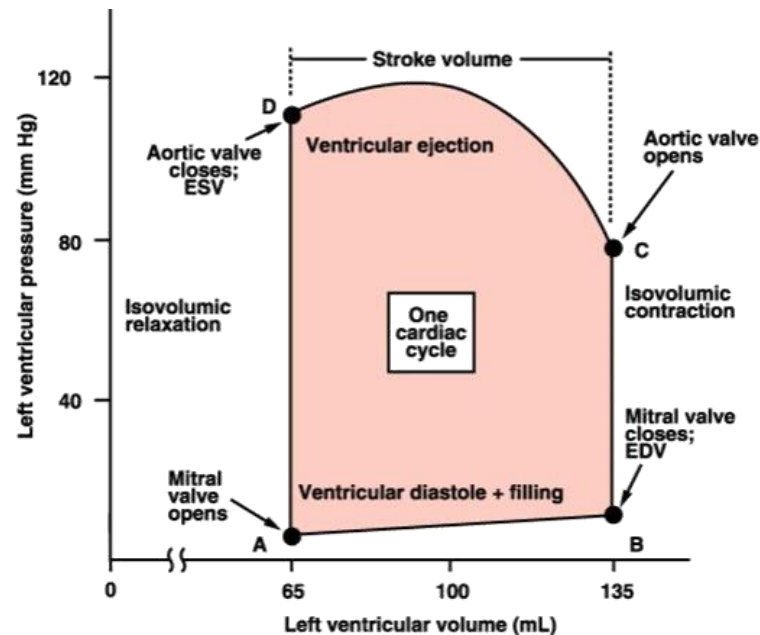
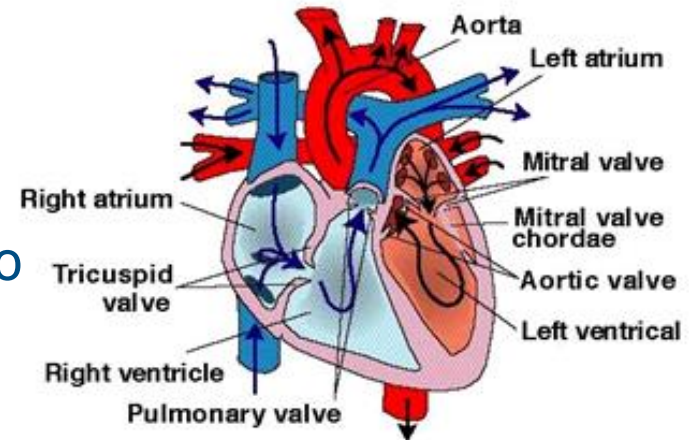
Department of Chemistry, Materials and Chemical Engineering "Giulio Natta", Politecnico di Milano

Scientific and Technological Advancements in Cardiac and Vascular Surgery

Erice, May 3rd 2015

The HEART pumps blood both into the systemic and pulmonary circulations

- Right and left atria: receive blood from veins
- Right and left ventricles: pump blood into arteries



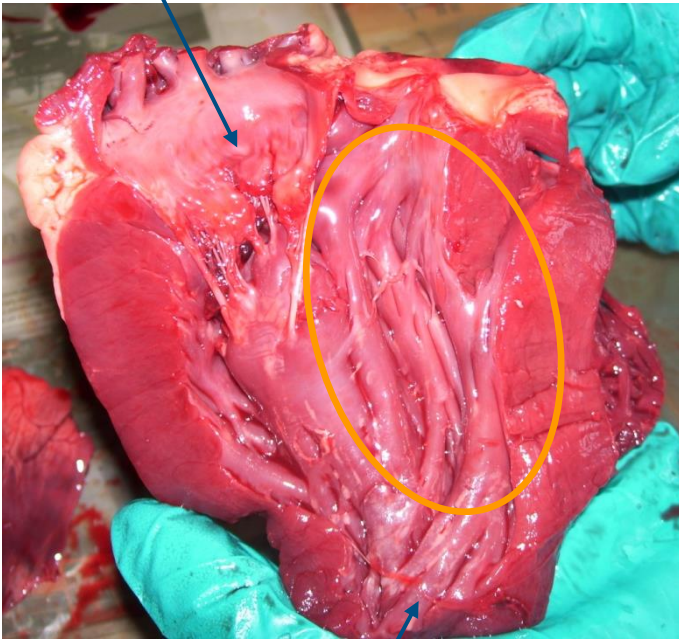
Cardiac cycle

- passive filling (diastolic phase)
- active contraction (systolic phase)

In literature studies some features of the cardiac structure are surprisingly disregarded

- **“strands” of axially arranged cardiac tissue**
- arise from the apex, and insert into the atrio-ventricular ring
- ventricle- and species-specific

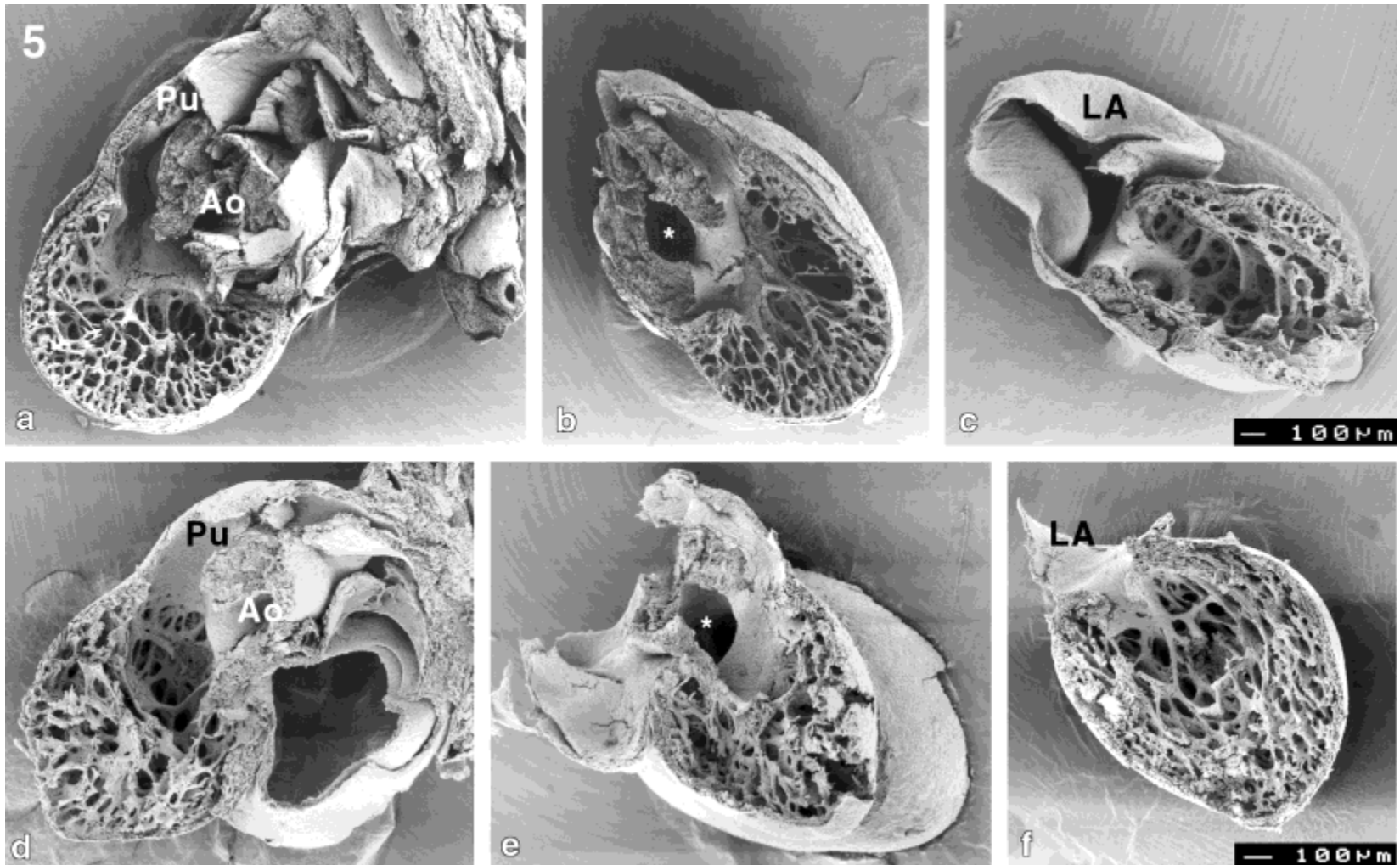
VENTRICULAR BASE



VENTRICULAR APEX

- Emergence of trabeculation during the embryonic development enables the myocardium to increase its mass also in the absence of coronary circulation.

[Jacquier A, Thuny F *et al*, European Heart Journal 2010].

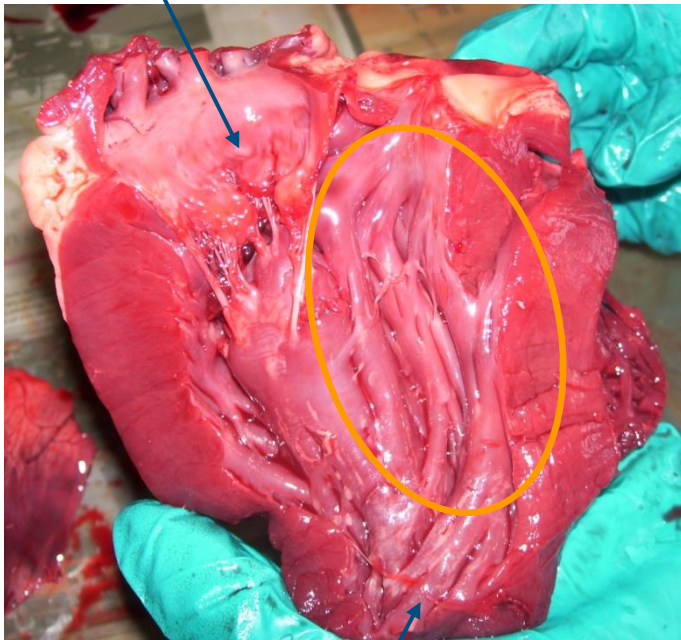


Sagittal dissection of trabeculated **chick** (a–c, 6th day/stage 29) and **human** (d–f, 41 days, Carnegie stage 18) hearts.

[Sedemera et al. The Anatomical Record 258:319–337 (2000)]

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VENTRICULAR BASE



VENTRICULAR APEX

- Emergence of trabeculation during the embryonic development enables the myocardium to increase its mass also in the absence of coronary circulation.
- In subsequent development, there is a compaction of these structures, **but an inner layer of trabecular tissue still remains.**

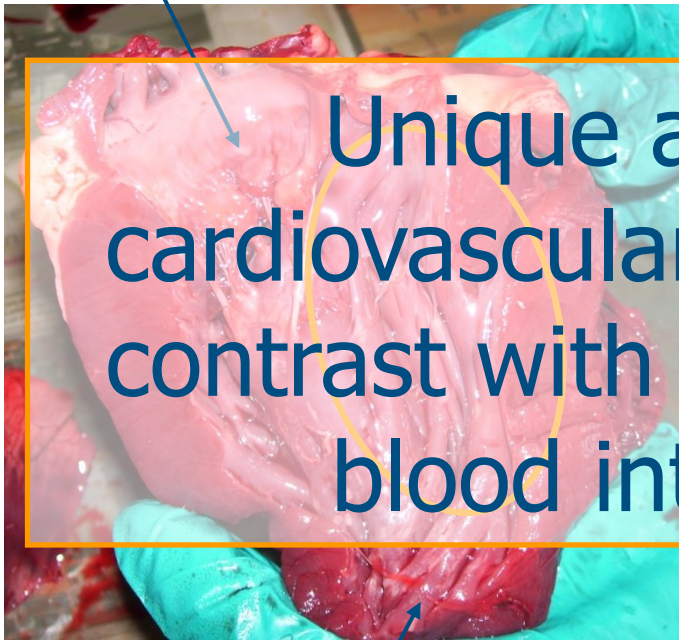


Trabecular mass is the ~ **12% - 17%** of ventricular mass in adult healthy subjects

[Jacquier A, Thuny F *et al*, European Heart Journal 2010].

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VENTRICULAR BASE



Unique architecture in the cardiovascular system, apparently in contrast with the typical structure of blood interacting surfaces

VENTRICULAR APEX

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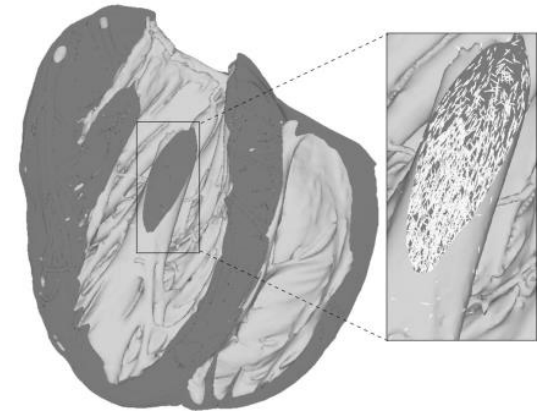
[Jacquier A, Thuny F *et al*, European Heart Journal 2010].

➤ QUALITATIVE HYPOTHESES ON TRABECULAE

- promote the loss of kinetic energy of blood during diastole,
- reduce the wrinkling of the sub-endocardial layer during systole, avoiding wall injuries,
- reduce the flow turbulence during the systolic phase,
- help the closure of the atrio-ventricular valves.

➤ QUANTITATIVE STUDIES

- fluid dynamic modelling: the blood “entrapped” into the inter-trabecular spaces is expelled by the contraction of the trabeculae, avoiding stagnation,
- electro-mechanical modelling: in simulation of cardiac arrhythmias, the excitation wavefront is not significantly affected by the trabeculated layer.



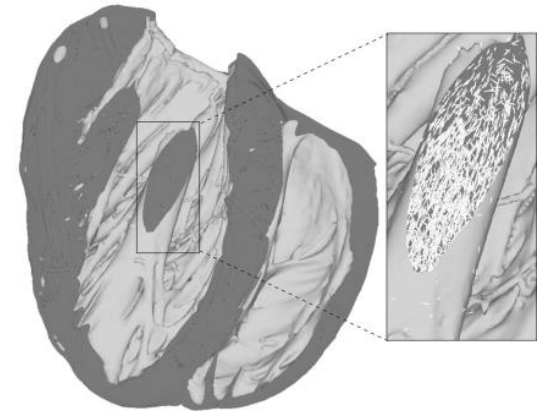
[Bishop JB, Planck G *et al*, The Journal of Physiology 2012]

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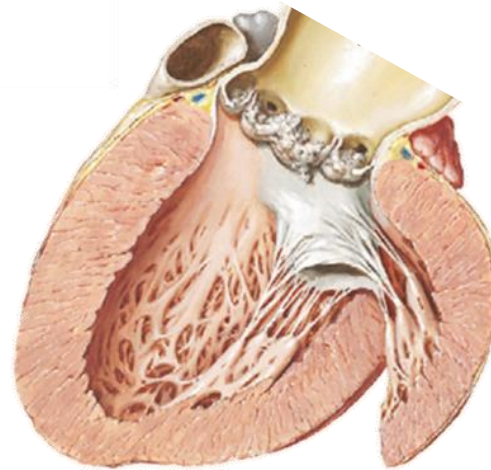
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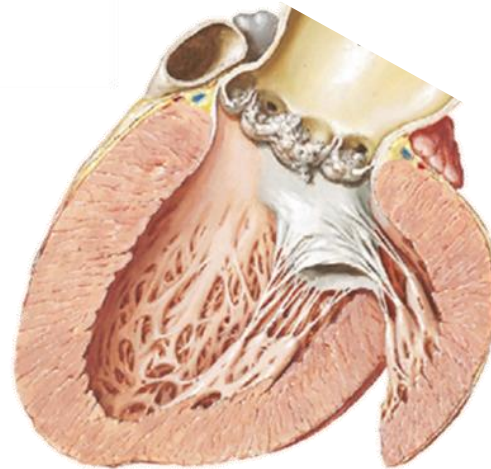
- **NO SPECIFIC MECHANICAL STUDIES**

[Bishop JB, Planck G *et al*, The Journal of Physiology 2012]

WHY DO TRABECULAE EXIST?



WHY DO TRABECULAE EXIST?



Study the influence of cardiac trabeculae on ventricular mechanics through the comparison of a trabeculated and a non-trabeculated ventricle (smooth ventricle)

✚ Finite element structural models (Abaqus®) of the left ventricle keeping constant

- **total ventricular muscular mass**
- **intra-ventricular volume**

both **in the presence (trabeculated model) and the absence of trabecular mass (smooth model)**

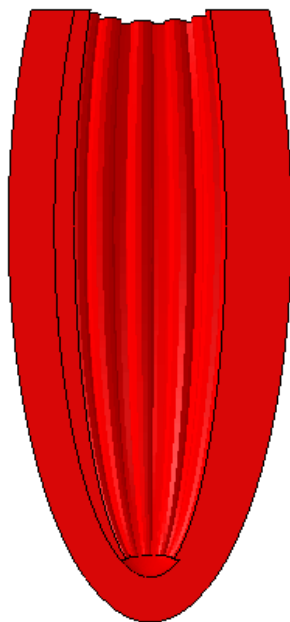
✚ Influence of some parameters characterising the trabecular layer

- **trabecular mass**
- **trabeculae diameter**
- **trabecular orientation**

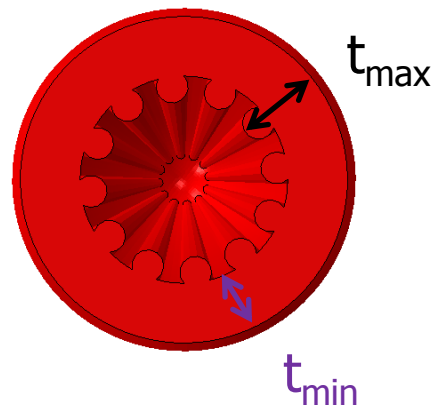
✦ Left ventricle simplified as a truncated ellipsoid in the reference, stress free configuration. **Intra-ventricular volume: 43 ml**

✦ **TRABECULATED MODEL:** 15% of the compact layer converted into trabeculae (physiological average value)

✦ **SMOOTH MODEL:** NO trabeculae

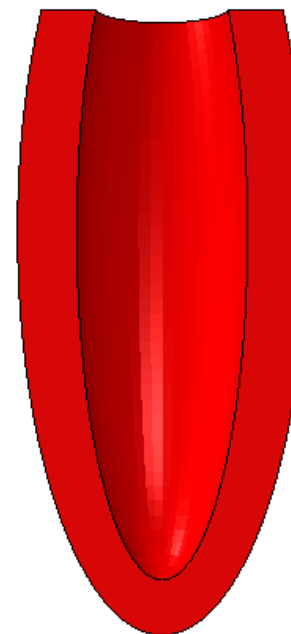


AXIAL SECTION

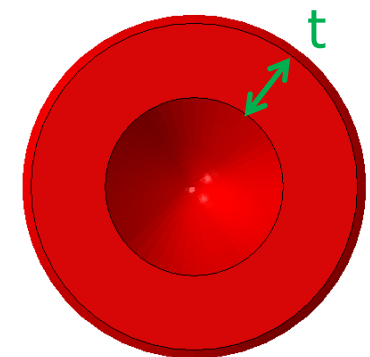


TOP VIEW

$$t_{min} = 7.2 \text{ mm}; t_{max} = 10.3 \text{ mm}; D_t = 4 \text{ mm}$$



AXIAL SECTION



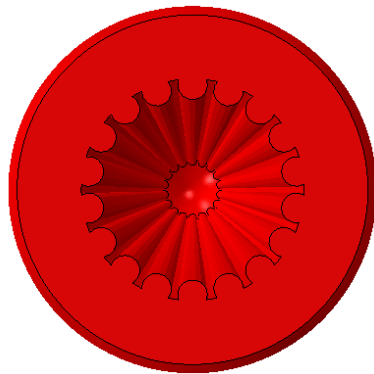
TOP VIEW

$$t = 9 \text{ mm}$$

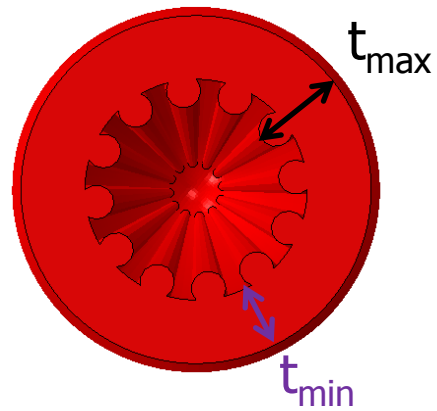
➤ Different **trabeculated layer diameter (D_t)**
(Constant thickness of the compact layer t_{min})

- Trabeculated layer mass = 15%

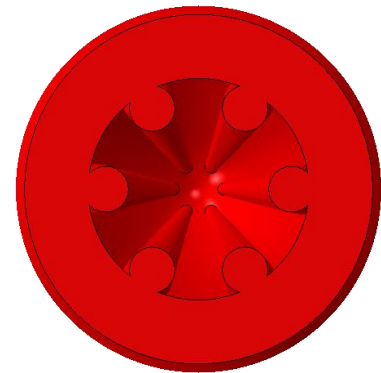
$D_t = 3.4$ mm



$D_t = 4$ mm



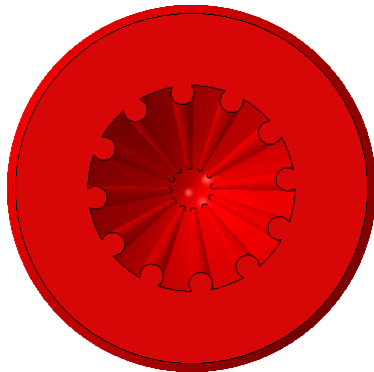
$D_t = 5.2$ mm



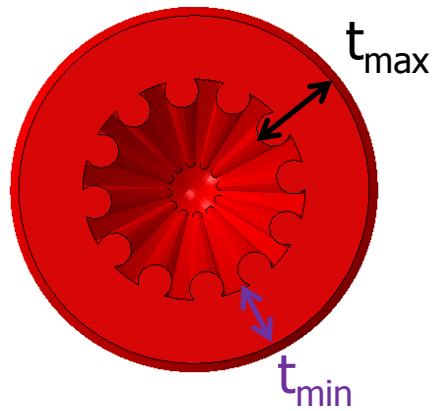
	$D_t = 5.2$ mm	$D_t = 4$ mm	$D_t = 3.4$ mm
t_{min} [mm]	7.25	7.25	7.25
t_{max} [mm]	9.6	10.45	11.85

↘ Different **trabeculated layer mass (M_t)**

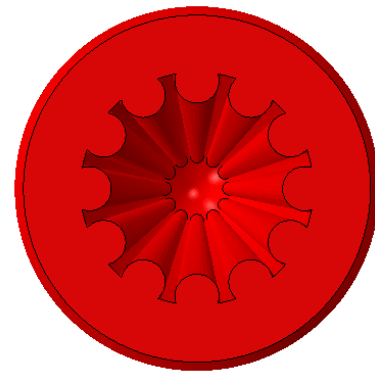
$M_t = 7\%$



$M_t = 15\%$



$M_t = 22\%$



	Smooth	$M_t = 7\%$	$M_t = 15\%$	$M_t = 22\%$
t_{\min} [mm]	9	8.2	7.2	6.5
t_{\max} [mm]	9	10.3	10.4	10.1

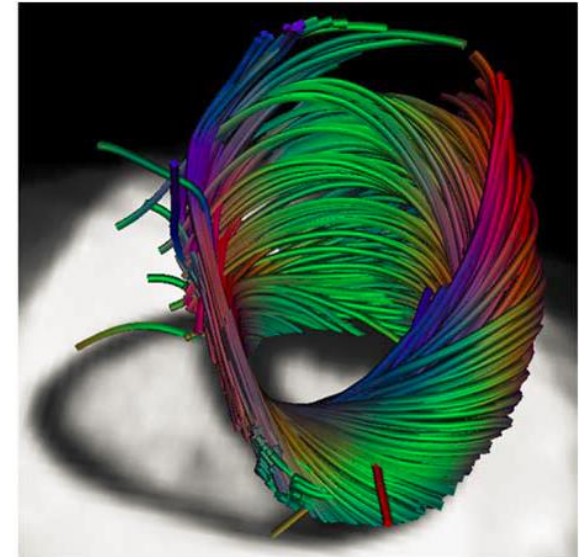
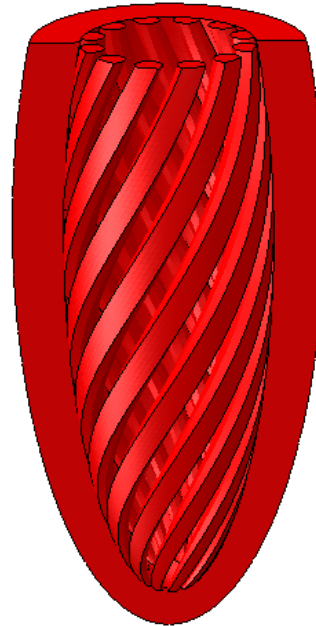
↘ Different **orientation** of the trabecular structures

→ Trabeculae oriented at $+60^\circ$ with respect to the circumferential direction, as suggested by experimental findings

- Trabeculated mass = 15%
- Trabeculae diameter = 4 mm



Ventricular cavity reconstruction obtained by laser technique (Mechanical Engineering Depart. and LaBS, Politecnico di Milano)



Spiral fibres architecture in the left human ventricle [Lombaert et al. IEEE Transactions on Medical Imaging 2012]

➤ MATERIAL CONSTITUTIVE LAW

➤ MUSCLE FIBER DISTRIBUTION

➤ MATERIAL CONSTITUTIVE LAW: passive behaviour

➤ MUSCLE FIBER DISTRIBUTION

Strain energy potential [Holzapfel et al.]:

$$\Psi = \underbrace{C_{10}(\bar{I}_1 - 3)}_{\text{MATRIX}} + \underbrace{\frac{k_1}{2k_2} (\exp(k_2(\bar{I}_4 - 1)^2) - 1)}_{\text{FIBRES}}$$

- Incompressible material
- One family of perfectly aligned fibers
- A_α is a unit vector which defines the fiber direction

$$\bar{I}_1 = \text{tr}(\bar{\mathbf{C}}) \quad \bar{I}_4 = A_\alpha \cdot \bar{\mathbf{C}} \cdot A_\alpha$$

➤ MATERIAL CONSTITUTIVE LAW: passive behaviour

➤ MUSCLE FIBER DISTRIBUTION

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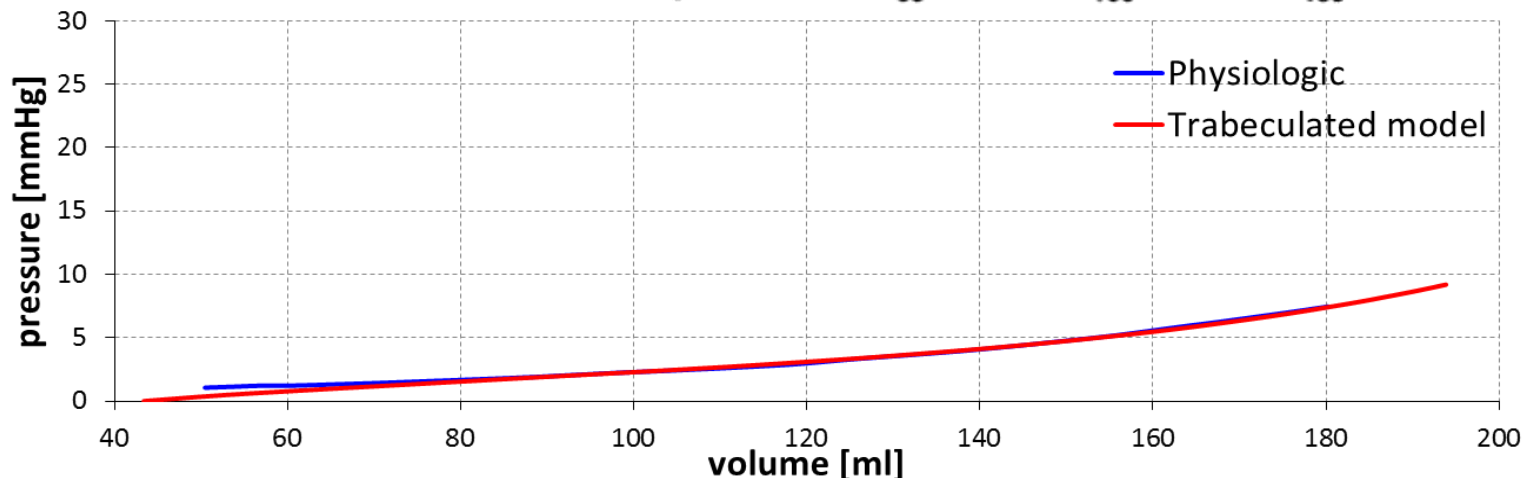
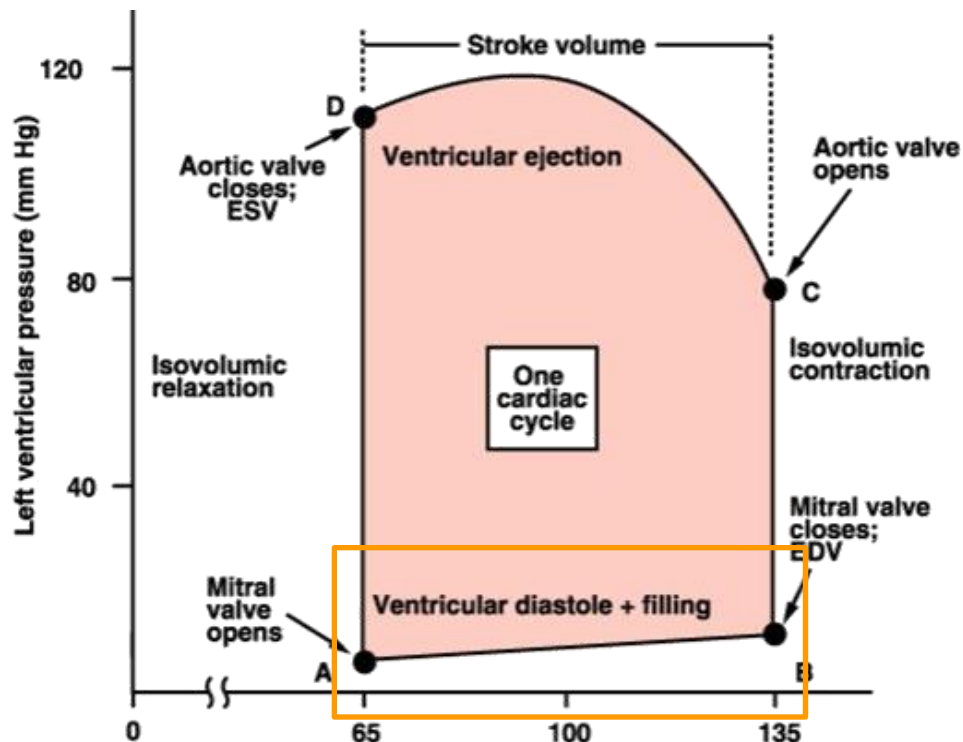
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➤ PARAMETERS ESTIMATION (C_{10} , k_1 , k_2) by fitting a physiologic pressure-volume relationship during ventricular filling of the trabeculated model

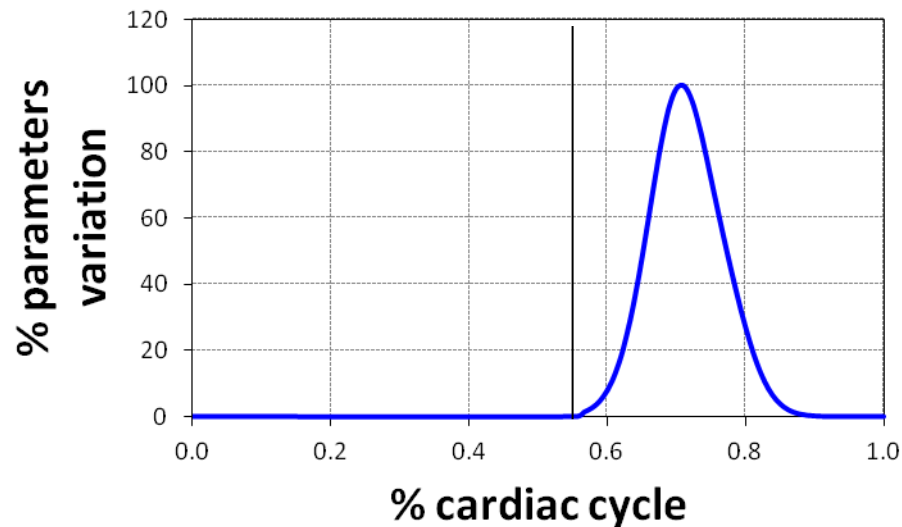
▶ **PARAMETERS ESTIMATION** (C_{10} , k_1 , k_2) by fitting a physiologic pressure-volume relationship during ventricular filling of the trabeculated model



➤ MATERIAL CONSTITUTIVE LAW: active behaviour

➤ MUSCLE FIBER DISTRIBUTION

➤ Myocytes contraction obtained by changing material parameters C_{10} , k_1 , k_2 during the cardiac systole



Parameter	Diastolic value	Systolic maximum value
C_{10}	0.2 kPa	6 kPa
k_1	1 kPa	150 kPa
k_2	2	20

➤ force generated by the muscle fiber secondary to an intracellular calcium variation

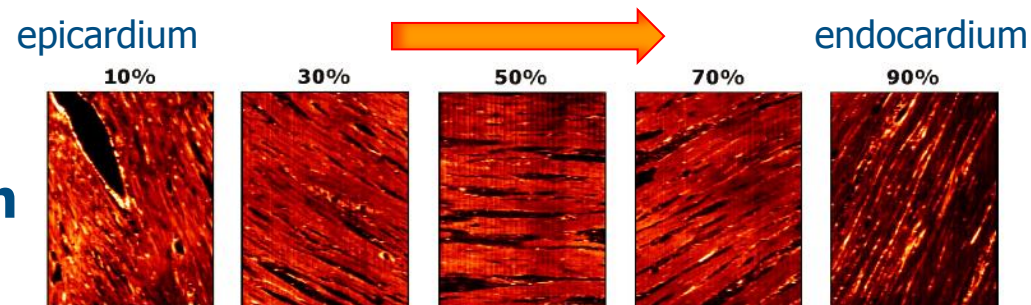
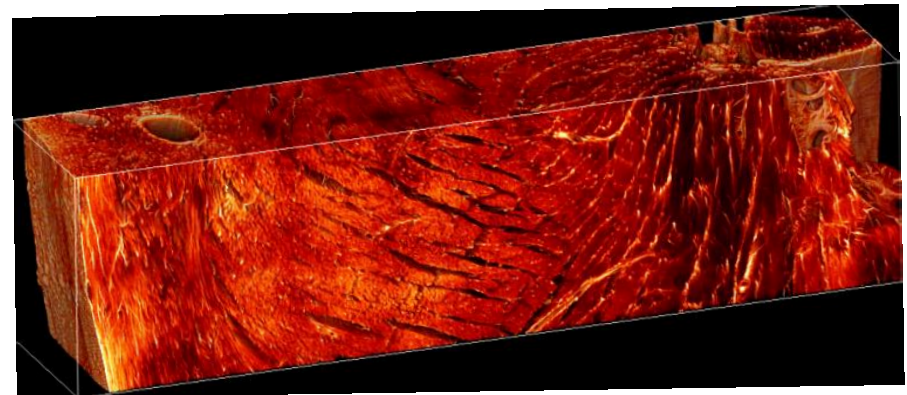
➤ MATERIAL CONSTITUTIVE LAW

➤ MUSCLE FIBER DISTRIBUTION

Ventricular wall = series of discrete layers of parallel myocytes



- The orientation of muscle fibers changes across the compact ventricular wall
(- 80° at the epicardium;
0° at the midwall;
+80° at the endocardium)
- Fibers are arranged **axially in the trabeculae**



[Sands GB, Dane A et al. *Microscopy research and technique* 2005]

➤ MATERIAL CONSTITUTIVE LAW

➤ MUSCLE FIBER DISTRIBUTION

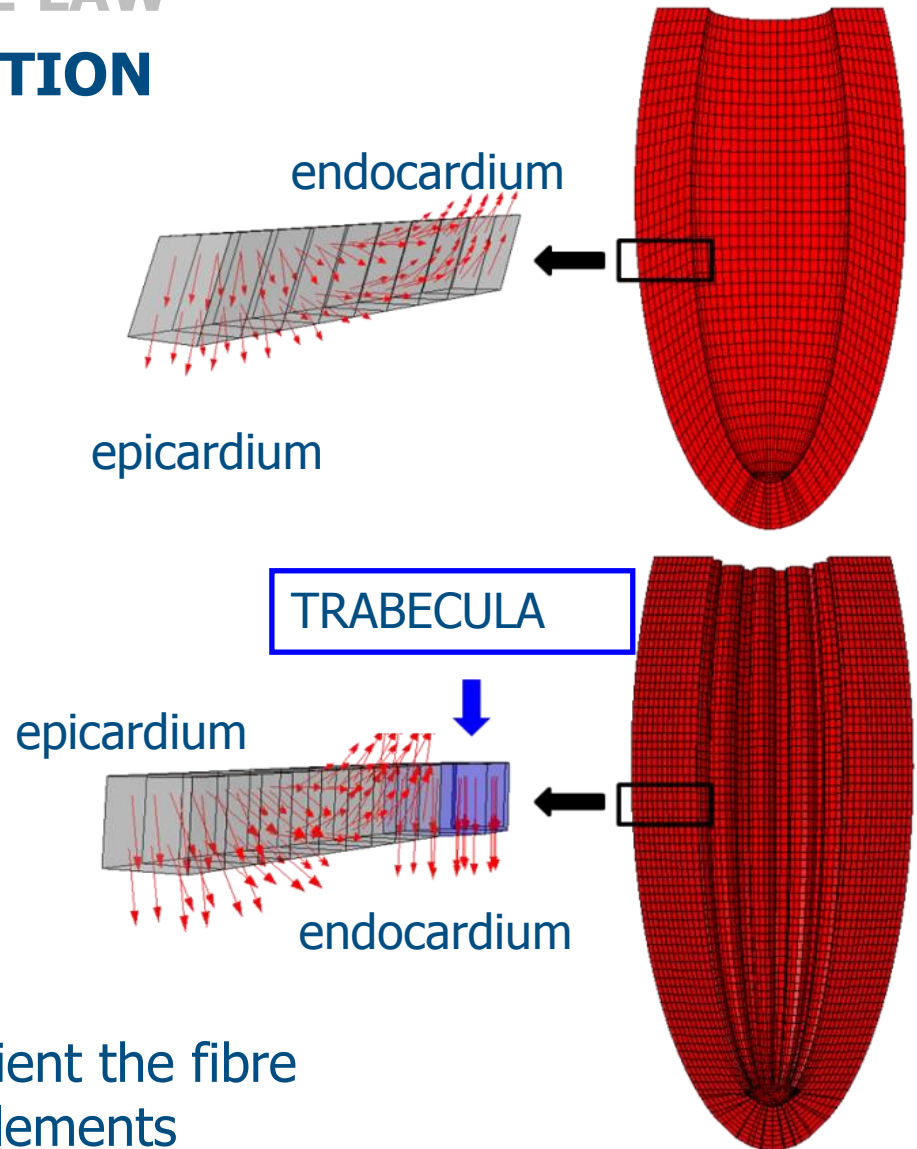
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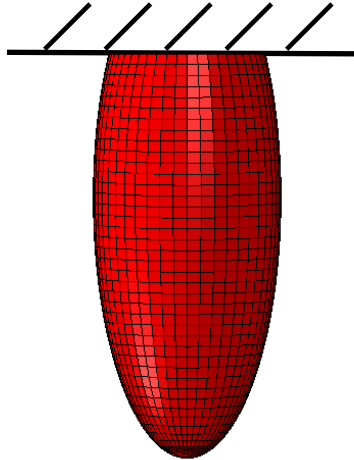
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Software routines to orient the fibre direction in the mesh elements

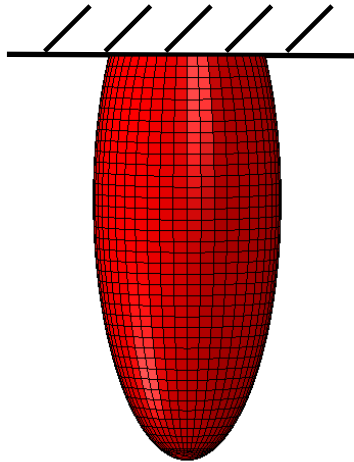


↘ KINEMATIC CONSTRAINTS



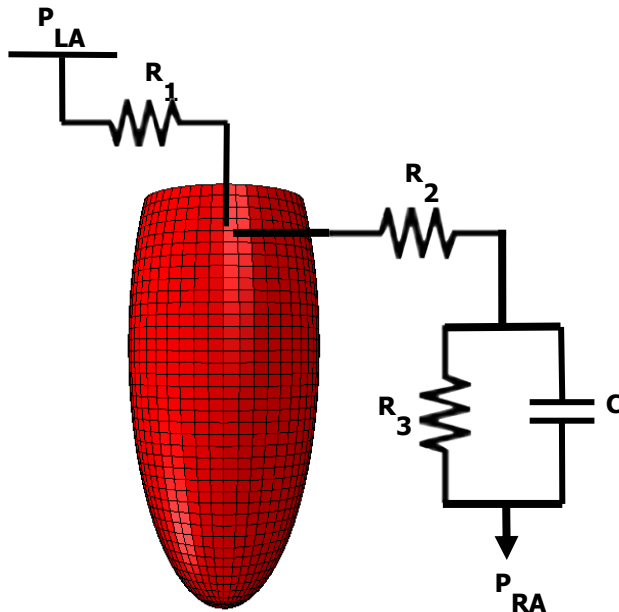
Displacements of the ventricular base were prevented.

✚ KINEMATIC CONSTRAINTS



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✚ PRELOAD AND AFTERLOAD CIRCUITS

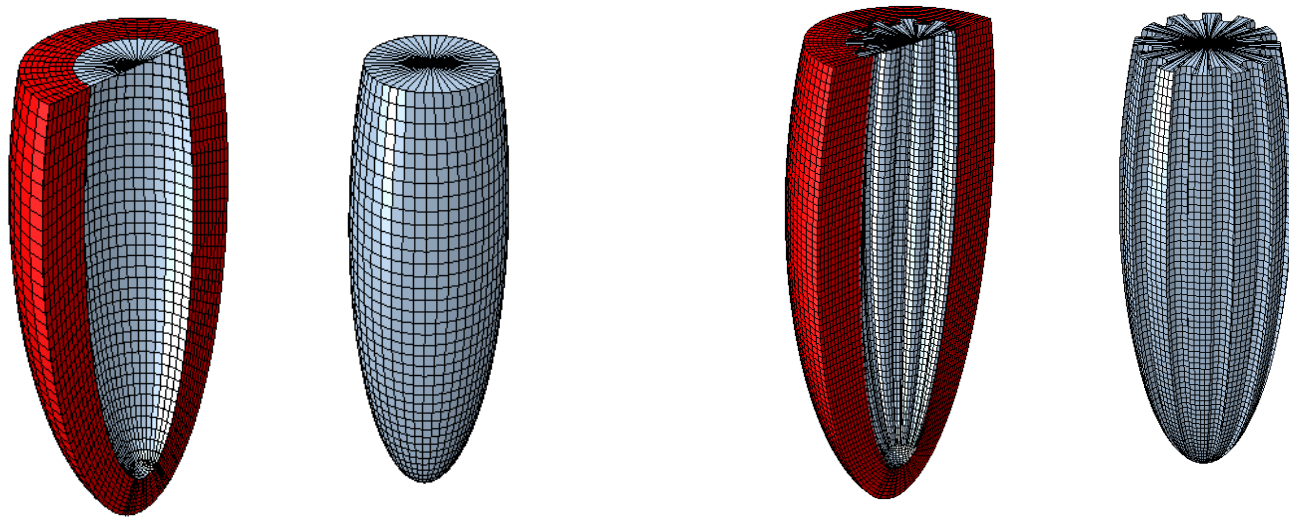


Physiological atrial pressure was set during the diastole to simulate the ventricular filling (preload).

A RCR model was coupled to the ventricle to simulate the systemic circulation (afterload).

PRELOAD AND AFTERLOAD CIRCUITS

- Hydrostatic fluid elements
- Surface elements which share the nodes with the structural elements, if present
- Closed cavities



- coupling between the deformation of the fluid-filled cavity and the pressure exerted by the fluid (uniform pressure, no gradient)
- simulation of blood inflow and outflow

- ↘ **Quasi-static** approach
- ↘ A minimum of **five cycles** were simulated for each model to reach the steady state of the system
- ↘ Standard heart rate = **75 bpm**



Other heart rates were simulated by changing the diastolic (T_D) and systolic duration (T_S)

$$T_S = \sqrt{kT}$$

$$k = 0.096 \text{ s}$$

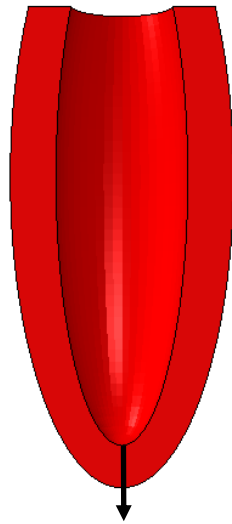
[Katz LN and Feil HS, 1932]

$$T_D = T - T_S$$

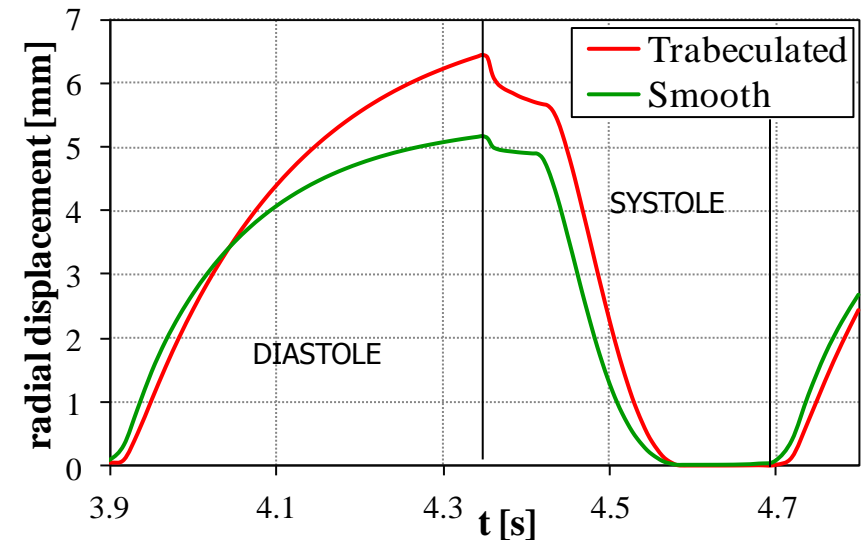
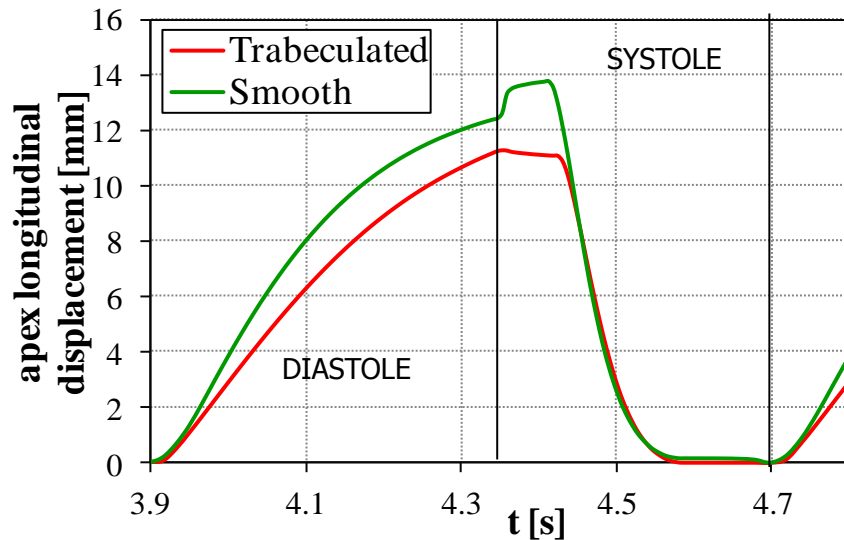
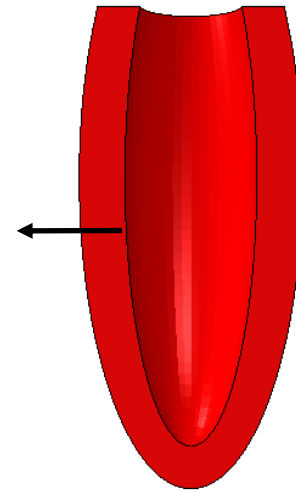
Simulated heart rates = 10, 30, 60, 75, 110, 180 bpm

Kinematic behaviour

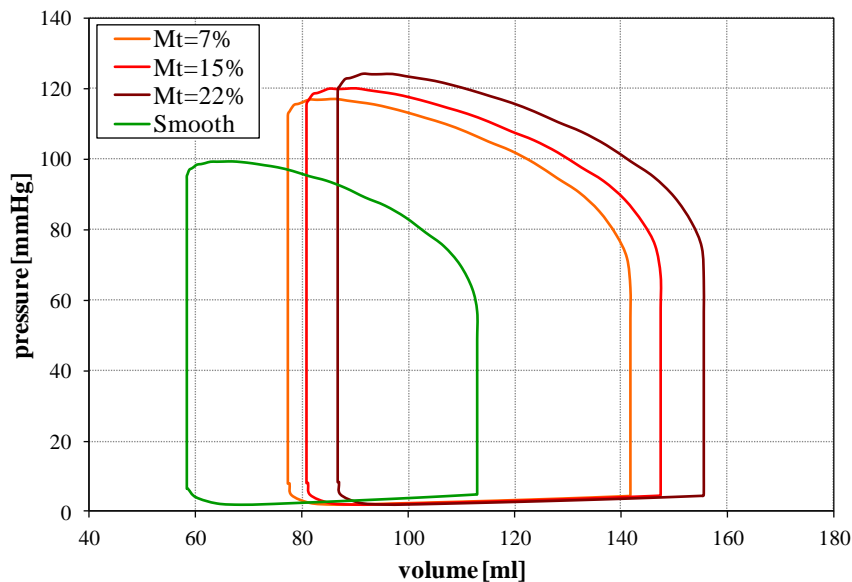
Apex longitudinal displacement



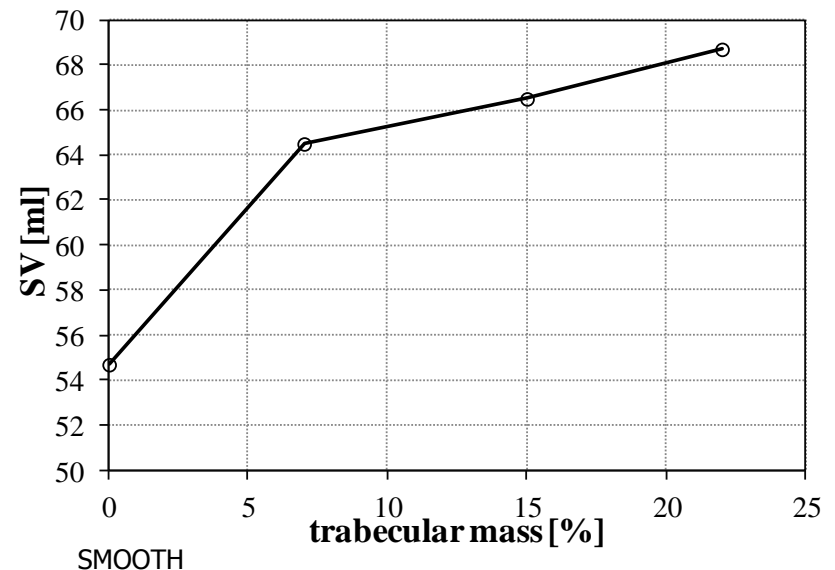
Mid-axis radial displacement



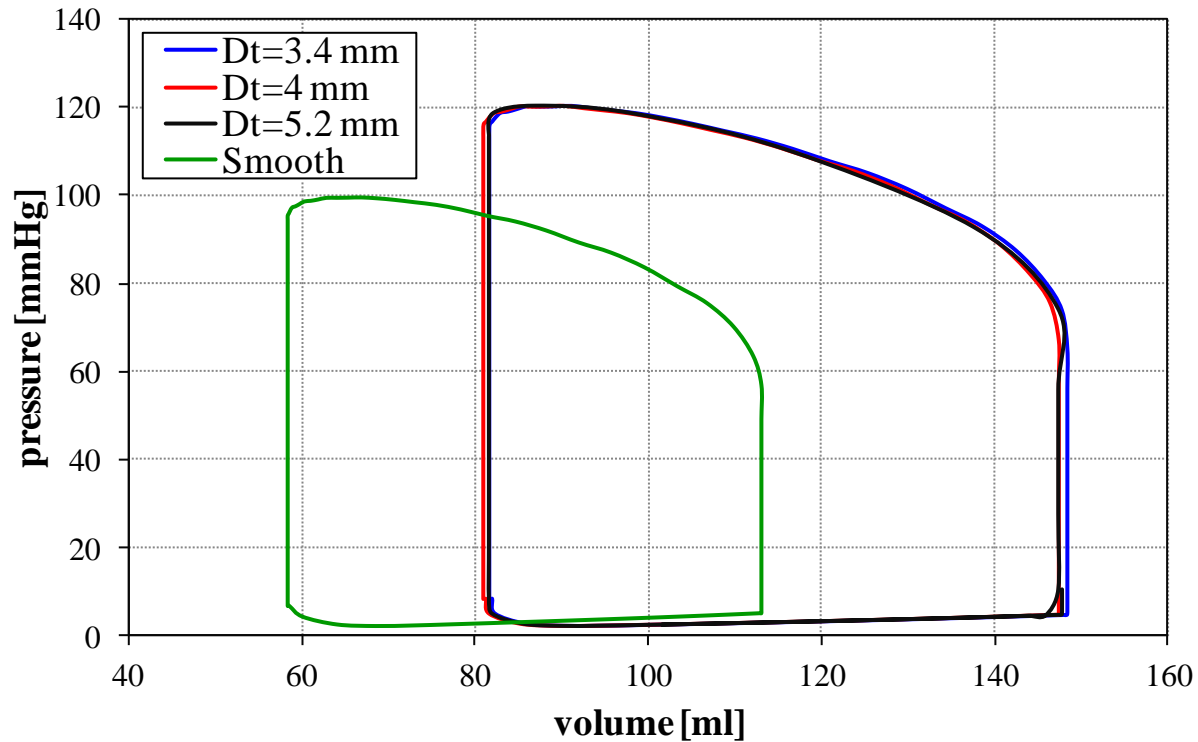
➤ PV loop



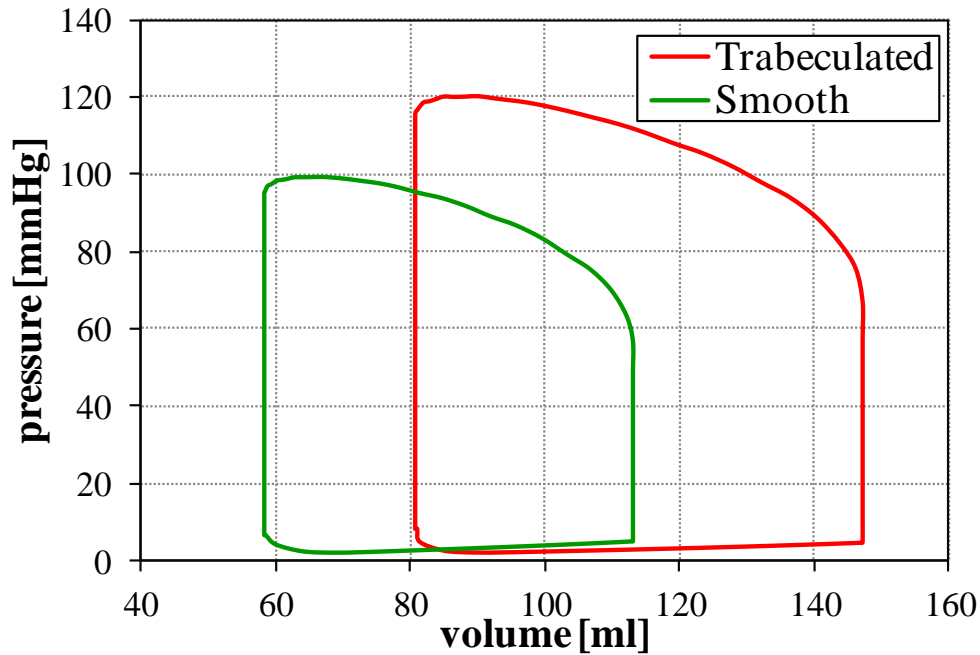
- A larger trabecular mass is responsible for a **non-linear increase of the EDV** and, consequently, of the SV



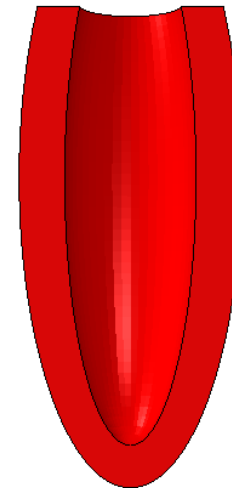
👉 PV loop



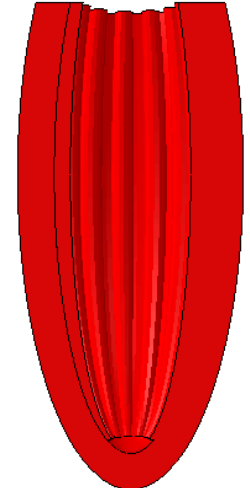
➤ PV loop



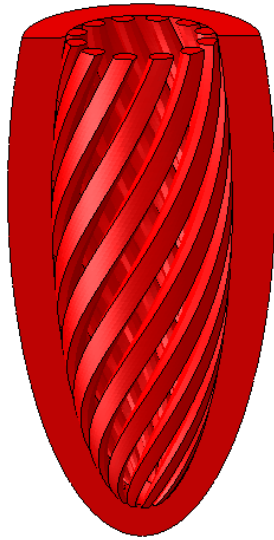
Smooth



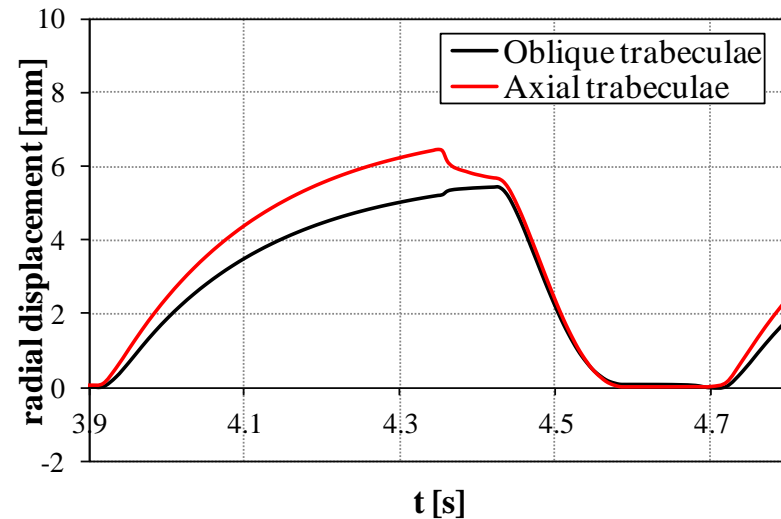
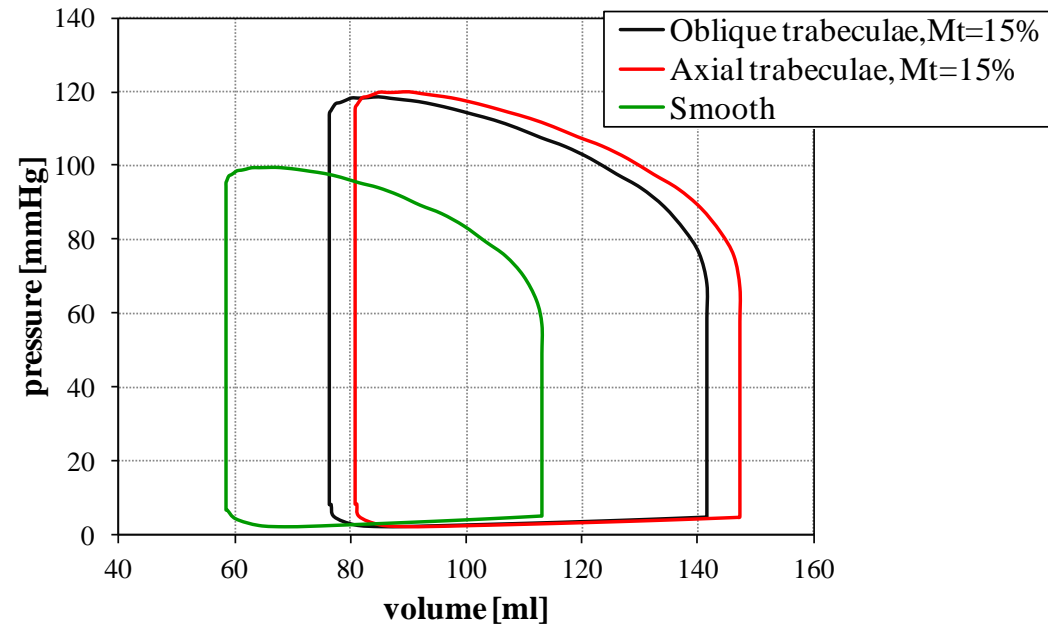
Trabeculated



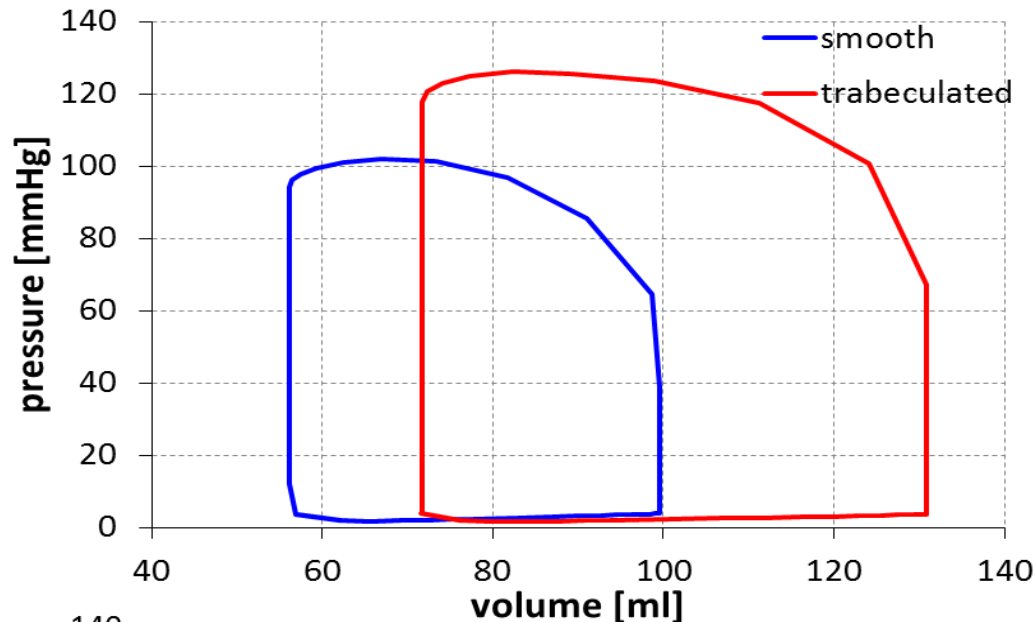
➤ PV loop



- The trabecular orientation contributes to a decrease of the EDV, due to a smaller radial displacement



RESULTS: Trabeculated vs Smooth Ventricle



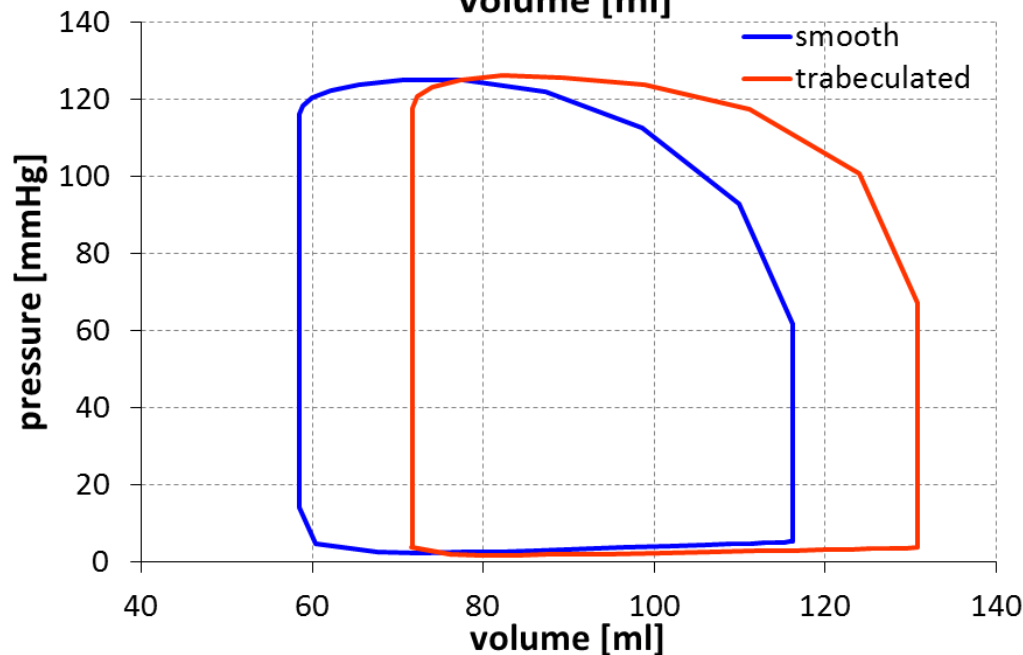
Atrial pressure = 4 mmHg



Different Stroke Volume

Smooth model = 43 ml

Trabeculated model = 60 ml



Stroke volume = 60 ml

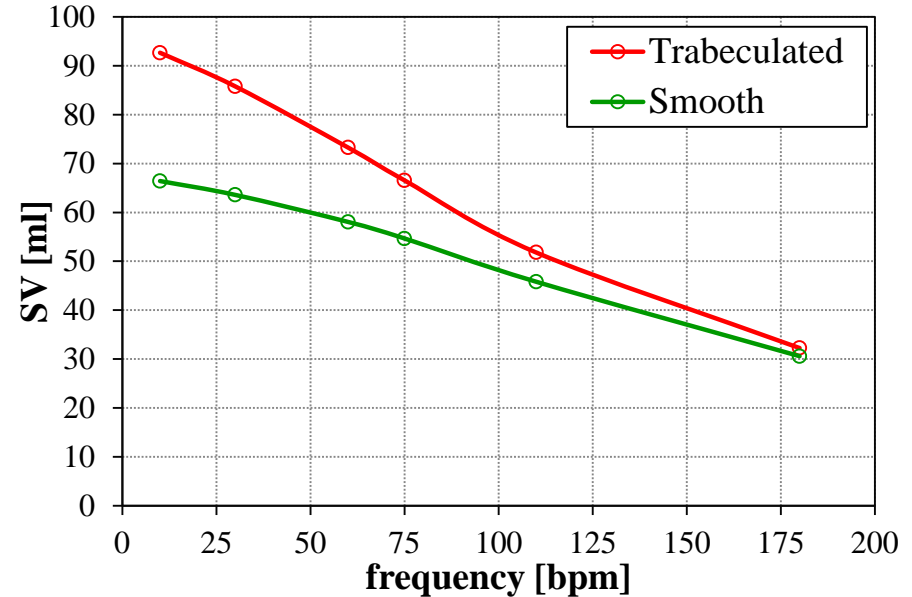
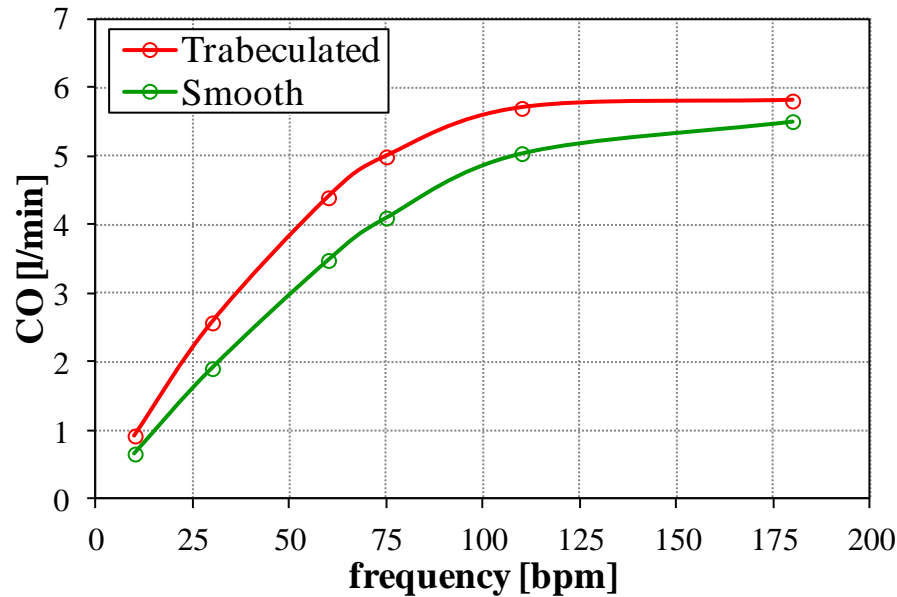


Different Atrial pressure

- Smooth model = 5.1 mmHg

- Trabeculated model = 4 mmHg

Effect of cardiac frequency



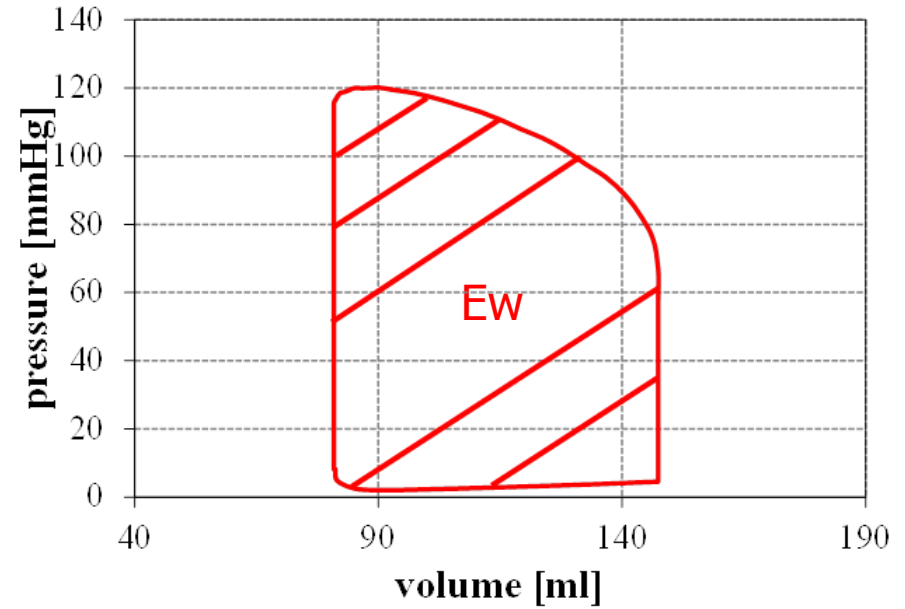
$$CO = \text{Heart rate} * SV$$

- The trabeculated model generates larger CO at each heart rate, than the smooth model
- The SV is higher too, especially at low heart rates.

↘ Ventricular efficiency

$$\eta = \frac{E_w}{E_{tot}} = \frac{\oint p dV}{E_{tot}}$$

Physiological value $\eta = 10\%-20\%$

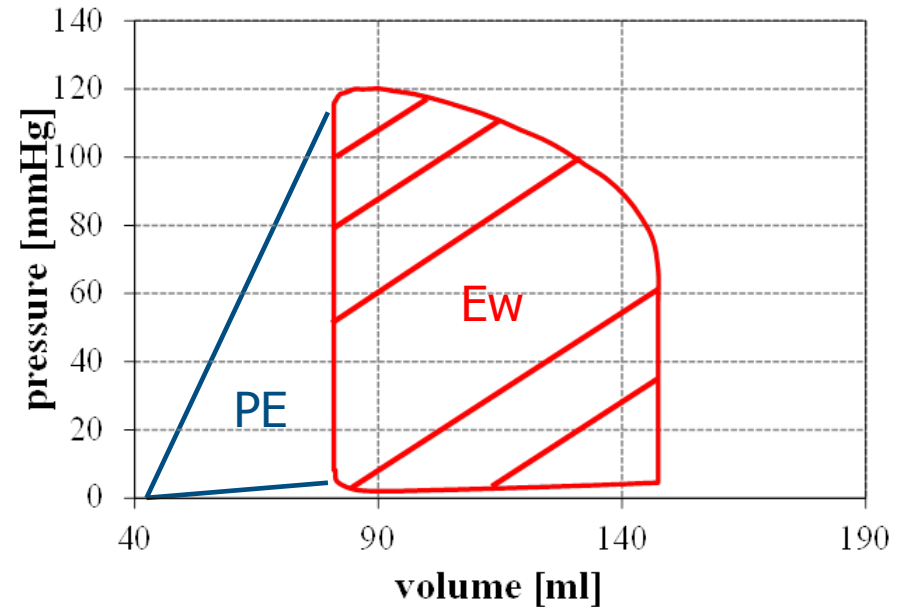


➤ Ventricular efficiency

$$\eta = \frac{E_w}{E_{tot}} = \frac{\oint pdV}{\boxed{MV_{O_2}}} = \frac{\oint pdV}{\oint pdV + TTI}$$



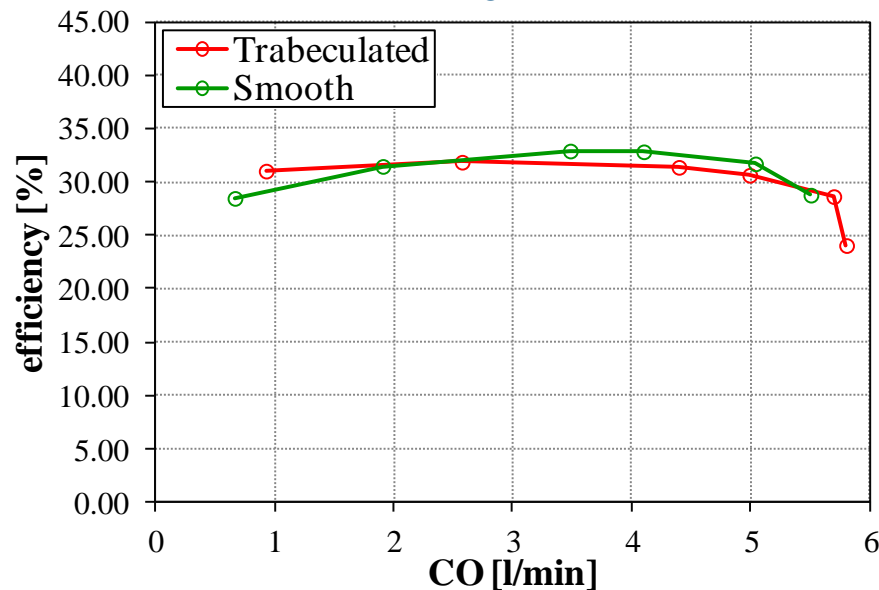
Different literature methods for the oxygen consumption estimation



1. **Experimental** linear relationship between MV_{O_2} and PVA (PVA=EW+PE) [Loiselle DS, Crampin EJ et al. *Progress in biophysics and molecular biology* 2008]
2. **Experimental** piecewise linear relation between MV_{O_2} and cardiac frequency [Nelson RR, Gobel FL et al., *Circulation*. 1974]
3. **Estimation** of the tension-time index (TTI) by the ventricular fibre stress [A.C. *Physiology and biophysics of the circulation* 1972]

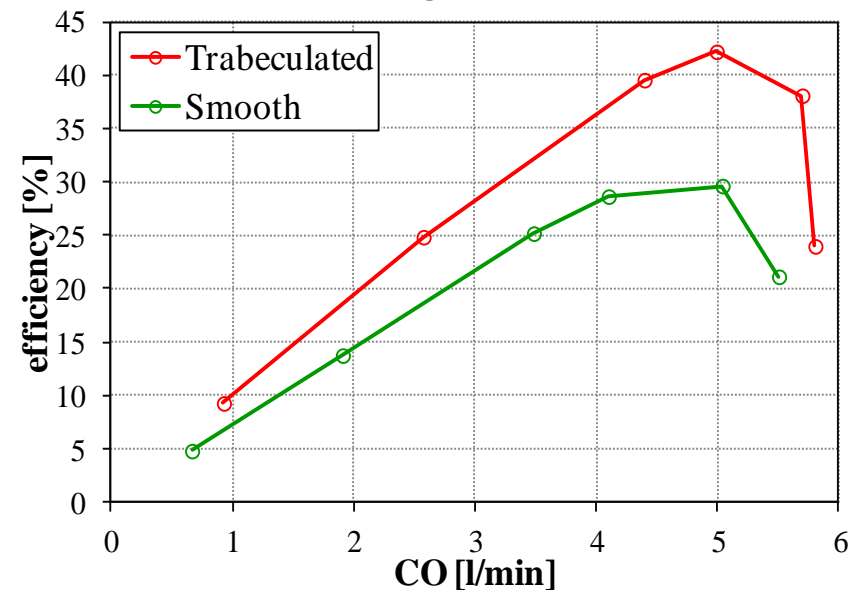
➤ Ventricular efficiency (Physiological range $\eta = 10\%$ - 20%)

1. MV_{O_2} (PVA)



- Predicted efficiency above the physiological range
- No efficiency variation with CO not in agreement with physiology
- No significant differences between models

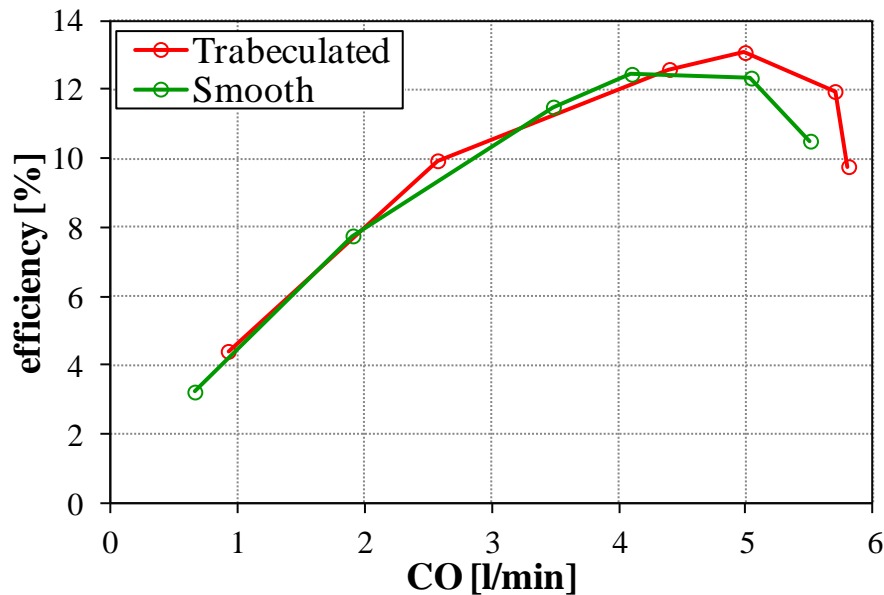
2. MV_{O_2} (heart rate)



- Predicted efficiency above the physiological range
- Efficiency-CO relationship in good agreement with the physiological one
- The trabeculated model shows a remarkable higher efficiency

➤ Ventricular efficiency (Physiological range $\eta = 10\%-20\%$)

3. MV_{O_2} (TTI)



$$TTI = a \int_{t_s} T dt$$

$$T = \int_V \sigma_f dV$$

$T = \text{fibre tension}$

$t_s = \text{systolic period}$

$$a = 9 [1/s]$$

- Realistic efficiency values
- Efficiency-CO relationship in good agreement with the physiologic one
- No significant differences between models

The presence of the cardiac trabeculae significantly influences the mechanical behaviour of the left ventricle

↘ **Higher ventricular compliance in the presence of trabeculae**



Enhanced ventricular filling
Higher stroke volume
Higher CO at the same heart rate

↘ **Trabeculae diameter**



Influences the wall stress distribution

↘ **Trabecular mass**



non-linear increase of the EDV and of the SV against the trabecular mass

↘ **Trabeculae orientation**



The ventricular compliance decreases if the trabeculae are not axially oriented

↘ **Efficiency?**

Thank you for your attention

This work was performed
with the help of
Marta Serrani PhD and
Massimiliano Mariani