Many cardiovascular system (CVS) models describe the heart contraction with phenomenological models (like the varying elastance model). In this work, a more realistic model of the CVS is presented, where the heart contraction is described instead at the cellular scale.

Membrane potential $V$ is described with the following equation:

$$\frac{dV}{dt} + \sum I_i + I_{ion} = 0$$

where an expression for each ionic current and the stimulus current is needed.

Ionic concentrations are also described by this model. Intracellular calcium allows for the connection between the electrophysiology and the mechanical contraction.

Passive chambers: pressure and volume are linked with the elastance of the chamber:

$$P = EV$$

Blood flow is related to the pressure at the entrance and at the exit of a chamber and to the resistance of the blood vessels:

$$Q = P_e - P_a$$

Volume of a chamber varies according to

$$\frac{dV}{dt} = Q_{in} - Q_{out}$$

The four cardiac valves act as diodes to allow for the unidirectionality of the flow.

The contraction of a half-sarcomere of length $L$ is described as follow:

$$L = X + h \frac{dX}{dt} - B(h - h_0)$$

The myosin head cycle is described by a 6-state model:

$$N \text{ half-sarcomeres of length } L_{in} \text{ are aligned along a circle of radius } R:$$

$$L_{in} = \frac{2\pi R}{N}$$

The equilibrium between the two half-spheres gives the relationship between the active pressure and the half-sarcomere normalized force:

$$P = F_m \frac{L_{in} \left( \frac{L_{out}}{L_{in}} \right)^2 - 1}{L_r}$$

Our multiscale model can account for a healthy behaviour and for basic hemodynamic experiments like preload variations. More importantly, it is able to reproduce pathological behaviours that originate at the cellular scale, like heart failure, and their consequences on the whole CVS.

P.D. acknowledges for FRS-FNRS travel support.

Contact: sarah.kosta@ulg.ac.be