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A simplified mechanical modeling for myocardial contractions and the ventricular pressure–volume relationships

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ABSTRACT

We present a simplified heart model with the aim of introducing a mechanical point of view in the interpretation of the pressure–volume loops. In particular, we propose a mechanical description of *muscle contraction*, and discuss its implications with reference to a specific pressure–volume loop representative of a normal human patient.

1. Introduction

The pressure–volume loops represent an assessment of the overall mechanical activity of the heart. Here, we present and discuss a mechanical point of view in the interpretation of the loops, based on a muscle modeling which takes into account for muscle contraction through the notion of active deformation. This point of view considers kinematics – used to describe contractions – as a primary notion in muscle modeling, and is opposite to the well known approach based on active force: we assume that an unconstrained muscle under stimulus experiences a contraction, that we model as a *stress-free* change of its length, called *active deformation. Thus, the active deformation describes how a muscular tissue shortens once stimulated and left free to contract; it follows that a muscle exerts a force only if its contraction is hampered by some constraints (as in isometric exercise, as example), while its actual length depends on both the amount of stress it sustains, and on the level of activation.*

We used this material model to study the mechanics of the left ventricle following a top-down modeling approach (DiCarlo et al., 2009; Evangelista et al., 2011). Here, we use the same material model to present our point of view regarding the PV loops. Our modeling defines a correspondence between each point of a PV loop and the underlying muscle contraction, and allows to view a PV loop as determined by three main state variables: pressure, volume, and contraction. To better illustrate and discuss our point of view, we use a specific sampling of pressure–volume pairs measured by Zhong et al. (2005) with reference to a normal human patient.

2. Methods

Here, we sum up the macroscopic model of muscle which embodies the notion of contraction presented in Nardinocchi and Teresi (2007) and DiCarlo et al. (2009).

We model an isolated fibre of muscle tissue as a one-dimensional continuum. It may be visualized as a bar of length l_s, meant to represent the muscle fibre in its *slack* state, that is, unloaded and with no activation. Moreover, we denote with l_c the contracted and unloaded length of the same fibre, and with l the length that the fibre reveals to an in vivo observation (the visible length). Here, we limit our analysis to homogeneous deformations and write:

$$l = \varepsilon l_s, \quad l_c = \varepsilon_c l_s \Rightarrow l = \varepsilon \varepsilon_c^{-1} l_c. \tag{2.1}$$

The elastic energy is then assumed to be a function of the ratio l/l_c between the visible and the rest state; thus, we define the *elastic deformation* φ as follows:

$$\varphi = \varepsilon \varepsilon_c^{-1}. \tag{2.2}$$

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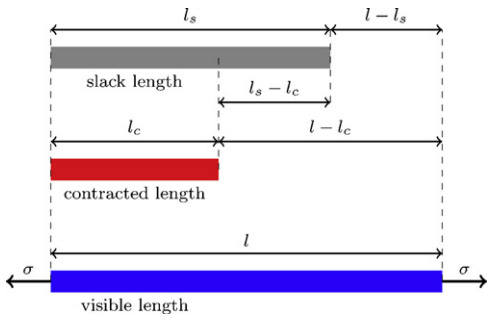

Fig. 1. Schematic of muscle contraction.

Fig. 1 shows the relationship represented by Eq. (2.1) through a cartoon. Eq. (2.2) allows a peculiar reading: the *deformation* $\varepsilon = l/l_s$ of the fibre is multiplicatively decomposable into an *active deformation* $\varepsilon_c = l_c/l_s$, measuring the contraction and denoted in the medical literature as unloaded shortening, and an elastic deformation $\varphi = l/l_c$, responsible for any variation in the elastic energy. We have: $\varepsilon_c < 1$ upon activation; $\varphi > 1$ under tension; $\varepsilon_c = \varphi = 1$ at the slack state. This approach is founded on a two-layer kinematic (Rodriguez et al., 1994; DiCarlo and Quiligotti, 2002): the total deformation ε lives at the visible layer, insofar it determines the visible length of the specimen. The active deformation (for us, the contraction) ε_c alters the length of the fibre but does not induce any tension, so that l_c corresponds to a ground state, that is, a state at zero tension. Thus, the active deformation characterizes a hidden layer as it takes up at a macroscopic level phenomena corresponding to microscopic cellular processes; as example, in Cherubini et al. (2008), it is assumed that the microscopic mechanism of excitation–contraction coupling governs the active deformation ε_c of the muscle fibre, and a relation is postulated between ε_c and the calcium concentration.

Finally, we assume as constitutive law $\hat{\sigma}$, relating the elastic deformation φ to the tension σ, the following relation:

$$\sigma = \hat{\sigma}(\varphi) = Y\lambda^3(\varphi), \qquad \lambda(\varphi) = \frac{1}{2}(\varphi^2 - 1), \qquad \varphi = \frac{\varepsilon}{\varepsilon_c} = \frac{l}{l_c}, \tag{2.3}$$

with Y the elastic stiffness and λ the Green–Saint Venant strain measure associated to the deformation φ. Eq. (2.3) comes from the assumption of a quadratic energy in the strain λ (here proposed in a 1D context), a generalization of the well-known quadratic energy describing the Green–Saint Venant materials.

The rather simple choice (2.3) for $\hat{\sigma}$ has been motivated by the following desiderata: (i) a very compliant response for $\lambda \simeq 0$; (ii) a quite stiffening response with increasing values of strain. These properties, assumed at the fibre scale, must persist at the LV scale, that is, the chamber must be very compliant at low pressure, and very stiff at high pressure. Thus, the proposed law captures, at least qualitatively, the passive response (when $\varepsilon_c = 1$), and the response upon activation (when $\varepsilon_c < 1$) of cardiac muscle.

3. Results

The macroscopic model of LV we present here is a zero-dimensional model that considers a spherical approximation of the chamber: it is simple enough to enlighten the key ideas at the basis of the modeling, yet capable to capture the important features of the pumping function of the heart which are collected in the PV loop. Moreover, it is rigorously extendible to the full fledged nonlinear 3D elasticity theory and the main mechanical relationships we aim to discuss are not altered by our simplified geometry.

Let us consider a spherical surface, whose hoop fibres behave as the one-dimensional fibre presented in the previous section; it follows that the length of the fibres is directly related to the radius r and the volume v of the sphere, and we may write

$$\varphi = \frac{l}{l_c} = \frac{r}{r_c} = \left(\frac{v}{v_c}\right)^{1/3}, \qquad \varepsilon_c = \frac{r_c}{r_s} = \left(\frac{v_c}{v_s}\right)^{1/3}, \tag{3.4}$$

with r_c, v_c, and r_s, v_s the pair radius, volume corresponding to the contracted and the slack state, respectively. Using (3.4), the constitutive relation (2.3) rewrites as a function relating the tension σ to the actual volume v of the chamber, its slack volume v_s, and the contraction state ε_c of the muscle

$$\begin{aligned}\sigma &= Y\lambda^3(\varphi) = Y\left(\frac{1}{2}\left(\left(\frac{r}{r_c}\right)^2 - 1\right)\right)^3 \\ &= Y\left(\frac{1}{2}\left(\left(\frac{v^{1/3}}{v_s^{1/3}\varepsilon_c}\right)^2 - 1\right)\right)^3.\end{aligned} \tag{3.5}$$

Finally, let us consider the balance equation: for h the constant wall thickness of the surface, a handy relation between the mean tension σ generated in the wall and the pressure p inside the spherical chamber is the well known Laplace formula:

$$p = 2\frac{\sigma h}{r}. \tag{3.6}$$

Granted for (3.5), it is easy to re-write the balance equation (3.6) in terms of pressure, volume and contraction state:

$$p = 2\frac{Yh}{\hat{r}(v)}\left(\frac{1}{2}\left(\left(\frac{v^{1/3}}{v_s^{1/3}\varepsilon_c}\right)^2 - 1\right)\right)^3, \quad \hat{r}(v) = \left(\frac{3}{4}\frac{v}{\pi}\right)^{1/3}. \tag{3.7}$$

Relation (3.7) represents the main result of our toy model of cardiac contraction: it can be viewed as a state function f, relating pressure, volume and contraction, with the stiffness Y acting as a parameter:

$$f(p, v, \varepsilon_c; Y) = 0. \tag{3.8}$$

The function f can be solved with respect to each one of the three variables (p, v, ε_c), and completely characterizes the pumping action of the heart. Of course, the plainness of Eq. (3.7) is due to the constitutive law (3.5), to the assumption on the hoop fibres which give rise to the relation (3.4) between the elastic deformation and the volume of the sphere, and to the easy form assumed by the balance Eq. (3.6). When the same material model is applied in more refined contexts, Eq. (3.8) is substituted by a set of nonlinear PDEs that cannot be solved explicitly (Evangelista et al., 2011). To frame the relation (3.8) within the cardio-mechanics literature, it is worth mentioning the notion of time-varying elastance $E(t)$, introduced by Suga in the late 60s to describe the pressure–volume relationship during the entire cycle: denoted with v_o a reference volume corresponding to zero pressure, we have

$$p(t) = E(t)(v(t) - v_o). \tag{3.9}$$

The idea underlying (3.9) is that of a state function linking three fundamental variables: for example, the knowledge of the time course of elastance $E(t)$ and volume $v(t)$ during the cycle allows for the prediction of the time course of pressure. The notion of elastance has been a key concept for cardiac physiologists and cardiologists since then; see Suga (1990) for review. It follows that the role of (3.8) is analogous to that of (3.9) and of the other empirical formulas relating pressure and volume as in Lankhaar et al. (2009), that is, to subsume through macroscopic quantities the main features of PV loops. Nevertheless, it replaces the notion of time-varying elastance $E(t)$ with that of time-varying contraction $\varepsilon_c(t)$, thus expressing through a physically based parameter the subtle notion of chamber contraction. In the end, let us note that the assumption that the