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On the Active Response of Soft Living Tissues

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Abstract Soft tissues exhibit a nonlinear, essentially incompressible (visco-) elastic response; a key issue is the active nature of muscle fibres, in other words their ability to contract and relax in response to biochemical signals. Here we present a continuum model able to describe an active elastic medium.

Keywords Active deformations · Soft tissues · Finite elasticity

Mathematics Subject Classifications (2000) 74B20 · 74G35 · 75L15

1 Introduction

Living tissues such as the myocardium and arterial walls exhibit a nonlinear, essentially incompressible (visco-) elastic response, as is typical of all soft tissues. Important features to be accounted for are the complex distribution of the predominant orientation of muscle fibres across the tissues and the active nature of these fibres [1–3].

Cardiac myocites are able to contract and relax at high frequency producing heartbeats; the contraction and relaxation of smooth muscle influence the distribution of stress and strain in the vascular wall. A constitutive model of the mechanical

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behaviour of such tissues must account for both their *passive response*, due to the extra-cellular matrix, and their *active response*, due to muscle fibres. The passive mechanical response has been extensively investigated through uniaxial and biaxial tests; however, very few data is available on the active response due to the presence of muscle fibres, even though the physiological mechanism regulating the contraction of muscle fibres is well understood.

The excitation-contraction coupling (ECC) in cardiac muscular fibres is a complex mechanism involving many variables such as membrane potential, ionic conductance, intracellular calcium concentration, membrane strain and stress, and changes in the rest length of muscle fibres due to the interaction of actin and myosin. The definition of a realistic model that is sufficiently complete to account for the principal mechanism involved in ECC, yet simple enough to be effective is an arduous task.

The issue has been addressed from the microscopic point of view in various papers aimed at establishing muscle models that relate sarcomere dynamics to calcium kinetics (see [4–7]). From the macroscopic point of view, finite elasticity theory with specific orthotropic constitutive prescriptions provides a reasonable framework for analysing ventricular mechanics. Moreover, the contractile excitable nature of the medium is usually accounted for through additive decomposition of the stress into a *passive* component, the standard one, plus an *active* component.

In [8], the additional active stress acts in the direction of the muscle fibres and depends on the concentration of free intracellular calcium (characterising the level of activation of a cardiac muscle cell) and on the time-varying muscle fibre extension ratio. In [9], the orthotropic muscle architecture of the dog ventricle is accounted for through the introduction of an additional active stress component with a diagonal structure with respect to the fibre-sheet axes. In [10] and [11], an electromechanical model of the excitable tissue capable of conducting non-linear waves of excitation is defined. The action potential enters a kinetic law driving the evolution of an isotropic active stress component; this latter, summed with a constitutively determined passive stress component, enters the balance equations of mechanics and determines the deformation of the medium. Furthermore, these kinematic deformations have a feedback effect on the excitation properties of the medium.

Typically, in arterial walls, smooth muscle cells contribute to the long-term adaptive response involved in growth and remodelling processes; they constitute the primary adaptive mechanism aimed at keeping the flow-induced shear stress and the wall stress distribution at their baseline values. The analysis of such remodeling processes has been addressed in many papers; in [12] and in [13], by assuming the substantial alignment of the smooth muscle cells to be in the circumferential direction, the contribution of muscle fibres in the remodeling processes is accounted for through the introduction of an active stress component acting in the circumferential direction and depending on the concentration of free intracellular calcium and on the actual stretch ratio in that direction; again, the total stress is given by the sum of the active and passive components.

Key points to be found in the literature on biomechanical cardiac and vascular models are the following: at the macroscopic level, the presence of muscle fibre enters the model through the tension generated by the fibre itself (the active stress); in addition, this tension has a preferred direction, which is defined by the orientation of the fibre. When activable soft tissues are considered as a whole, the overall tension state is described by adding up the passive and active stress.

Here, we propose a different point of view. We describe, at the macroscopic level, the excitable–contractile behaviour of the elastic medium by considering the change in the rest state of the tissue due to the contraction of active fibres. The *active* deformation, a (local) measure of the fibre's rest length, does not induce any stress. There follows a multiplicative decomposition of the *visible* deformation gradient into two components: the active and the *elastic* deformations; the latter is defined as the difference between the visible deformation gradient and the active component.

In living tissues, the active deformation field is related to the microscopic players in the ECC mechanism through specific laws based on the electrophysiology of the tissue, whose form is not the subject of this paper. In [14] an electromechanical model of excitable tissue based on the present setting is proposed.

As standard, we assume that the tissue is (elastically) incompressible and that a strain energy function exists, depending only on the elastic deformation gradient: the derivative of the energy with respect to the strain measure yields the energetic part of the stress; the overall stress measure is built by adding the indeterminate pressure field.

Thus, the energetic component of the stress is uniquely defined once the strain energy function has been chosen; moreover, given the basic kinematical decomposition, it may be represented as a function of the visible and the active deformation. Apart from alternative choices of the strain energy function, our assumptions deliver a stress measure that differs from those cited above ([8–10]) insofar as it does not allow reduction to a function that is additively decomposable into a passive part, depending only on the visible deformation, plus an active part, depending on the active deformation. It is shown that this additive decomposition is recovered under the hypothesis of *small* active stretches.

To start with, we sketch the main features of our modeling process using a prototype example involving a unidimensional cardiac muscle fibre. Then, a simple, albeit not trivial problem of three-dimensional elasticity, suitably revisited, allows us to present the consequences of our procedure and to discuss the results of our point of view.

2 A Preparatory Problem

Our approach is similar to that presented in [4], in which a macroscopic model for excitation-contraction coupling of a unidimensional cardiac muscular fibre is presented.

Given a reference configuration of the fibre, two strain measures are introduced: the visible strain ε, measuring the deformation of the actual (visible) configuration with respect to the reference configuration, and the rest strain ε_o, defining the stress-free state of the fibre with respect to the same reference. The elastic energy is then assumed to be a function of the elastic strain φ, a measure of the difference between the visible and the resting state, defined by the composition

$$\varphi = \varepsilon \, \varepsilon_o^{-1} . \tag{2.1}$$

Given a linear dependence of the tension σ on the elastic strain φ, we have

$$\sigma = Y\varphi , \tag{2.2}$$

where Y is the elastic modulus. From (2.2) and (2.1) we have the following representation formula

$$\sigma = \sigma_\varepsilon \, (\varepsilon_o)^{-1} \,, \qquad \sigma_\varepsilon = Y \, \varepsilon \,, \tag{2.3}$$

which can be viewed as a (multiplicative) decomposition of the response function for the tension into a passive part σ_ε, which depends on the elastic properties of the fibre, and an active part $(\varepsilon_o)^{-1}$, which depends on the change in the rest length of the fibre.

In [4], it is assumed that the microscopic ECC mechanism governs the rest strain ε_o of the muscle fibre. There, a simple reaction–diffusion system triggering the action potential is coupled to a linear stress-strain equation for the cardiac tissue (see (2.2)) via the intermediate of the calcium ions; the membrane potential and the effective membrane conductance satisfy a FitzHugh–Nagumo type system, and the membrane potential drives the evolution of the calcium concentration through a kinetic equation. The microscopic ECC at the basis of the model is completed by a relation, built from experimental data, between the rest length of the cardiac muscle fibre ε_o and the calcium concentration c: $\varepsilon_o = \hat{\varepsilon}_o(c)$.

From our point of view, the key points of this approach are two: (a) the tension in the muscle fibre is directly related to its elastic deformation, which in turn depends on both the visible deformation ε and the rest state ε_o; (b) calcium dynamics, as suggested by electrophysiology, (see [15]), governs the variation in rest length of the muscle; this last aspect prompts the use of the term *active strain* to describe the change in the rest length of excited muscle fibres. It is worth noting that the simple multiplicative decomposition (2.3) relies on both the unidimensional setting of the problem and the linear structure of the constitutive relation: if (2.2) were replaced by a more general dependence on the elastic strain, $\sigma = \hat{\sigma}(\varphi)$, the result shown by the representation formula $(2.3)_1$ would not be achieved.

In the following section, we present a model of an active three-dimensional tissue based on the key point (a), and show how the resulting Cauchy stress tensor is not decomposable into a passive and an active component except in certain special cases. Our main aim is to describe the mechanics of excitable tissues through a model that is well founded and energetically consistent; the underlying ECC mechanisms at the basis of activation in living tissues are not among the aims of our present effort.

3 A Model for Active Tissues

We introduce the notion of active deformation as distinct from both visible and elastic deformations, and develop a model for a hyperelastic body under the assumptions of incompressibility and isotropy. It should be recalled that the incompressibility constraint is a common hypothesis typically underlying the constitutive response of soft living tissues; isotropy, on the contrary, is less convincing but allows us to point out the essence of the model avoiding awkward expressions; in addition, it may easily be set aside and replaced by a more plausible anisotropic material response.

3.1 Kinematics

We regard a piece of tissue – which we shall here refer to as the body – as a smooth region $\mathcal{B}$ (with boundary $\partial\mathcal{B}$) of the three dimensional Euclidean space $\mathcal{E}$, and refer to any smooth embedding

$$\chi : \mathcal{B} \to \mathcal{E} \tag{3.1}$$

of the body into $\mathcal{E}$ as a placement. Tangent vectors at the body itself are referred to as line elements and the set of all line elements attached to a single body point $y \in \mathcal{B}$ is referred to as the body element at y, and denoted $T_y\mathcal{B}$, the tangent space to $\mathcal{B}$ at y. Given the identification of the body $\mathcal{B}$ with a region of $\mathcal{E}$, the tangent space $T_y\mathcal{B}$, for any $y \in \mathcal{B}$, is identified with the translation space $\mathcal{V}$ of $\mathcal{E}$; the term tensor will be used to denote the elements of $\mathbb{L}\mathtt{in}$, the space of linear maps from $\mathcal{V}$ onto $\mathcal{V}$.

We assume that, at the macroscopic scale, activation of the muscle fibres of the tissue is described by a variation in the rest length of the body elements, and that this variation is measured by a distortion field $\mathbf{F}_o : \mathcal{B} \to \mathbb{L}\mathtt{in}$, to be known as the *active deformation*. Then, we call *visible* deformation $\mathbf{F} := \nabla\chi$ the gradient of the placement χ, and we define the *elastic deformation* $\mathbf{F}_e$ of the body elements as the difference between the active and the visible deformations in the sense of multiplicative composition:

$$\mathbf{F}_e = \mathbf{F}\mathbf{F}_o^{-1}\,; \tag{3.2}$$

it is worth noting that $\mathbf{F}$ is given by a gradient, but $\mathbf{F}_o$ and $\mathbf{F}_e$ are not, in general. Then, given the visible strain measures $\mathbf{C} = \mathbf{F}^T\mathbf{F}$ and $\mathbf{B} = \mathbf{F}\,\mathbf{F}^T$, the corresponding elastic strain measures $\mathbf{C}_e$ and $\mathbf{B}_e$ read as

$$\mathbf{C}_e = \mathbf{F}_o^{-T}\,\mathbf{C}\,\mathbf{F}_o^{-1} \quad \text{and} \quad \mathbf{B}_e = \mathbf{F}\,\mathbf{C}_o^{-1}\,\mathbf{F}^T\,, \tag{3.3}$$

with $\mathbf{C}_o = \mathbf{F}_o^T\mathbf{F}_o$ being the strain induced by the active deformation field. Other elastic strain measures could easily be derived from (3.2).

We believe as relevant to account for possible anisotropic behaviour as an outcome of the active fibres architecture: the distribution of fibres may enter the model by assigning specific active deformation fields. As an example, an isotropic fibre distribution (as in [10]) is described by a spherical tensor: denoted by $\mathbf{I}$ the identity tensor, we have

$$\mathbf{F}_o = \gamma_o\,\mathbf{I}\,. \tag{3.4}$$

An architecture with a specific *line of activation*, the so-called mean myofibre axis in physiology (as in [8]), allows the corresponding active deformation field to be represented as

$$\mathbf{F}_o = \gamma_o\,\mathbf{e}\otimes\mathbf{e} + \check{\mathbf{I}}, \quad \check{\mathbf{I}} = \mathbf{I} - \mathbf{e}\otimes\mathbf{e}\,, \tag{3.5}$$

with $\mathbf{e}$ being the tangent field to the activation line.[1] In both cases, the electrophysiology of the tissue enters the model through the scalar field γ_o whose value may depend on the outer stimuli (see [14]).

[1] More complex is the orthotropic myofiber architecture discussed in [9]; it would enter our modeling through an active deformation field

$$\mathbf{F}_o = \alpha_o\mathbf{e}_1\otimes\mathbf{e}_1 + \beta_o\mathbf{e}_2\otimes\mathbf{e}_2 + \delta_o\mathbf{e}_3\otimes\mathbf{e}_3\,, \tag{3.6}$$

where $\mathbf{e}_i$ with $i = 1, 2, 3$ denote the fiber-sheet directions, and the fields α_o, β_o, and δ_o may be assigned in terms of the microscopic stimuli according to electrophysiological data.

3.2 Constitutive Prescriptions

We consider a nonlinear, essentially incompressible, elastic response, and we postulate the existence of a real-valued strain energy function, assuming that, at each body point, the present value of the strain energy per unit rest volume $\hat{\psi}$ depends only on the present value of the elastic deformation $\mathbf{F}_e$ at that point:

$$\hat{\psi} : \mathbf{F}_e \mapsto \hat{\psi}(\mathbf{F}_e)\,, \quad \hat{\psi}(\mathbf{I}) = 0\,. \tag{3.7}$$

The elastic deformation $\mathbf{F}_e$ also satisfies the incompressibility constraint:

$$\det \mathbf{F}_e = 1\,. \tag{3.8}$$

The assumed hyperelasticity delivers the energetic part $\mathbf{S}$ of the Piola–Kirchhoff stress measure as

$$\mathbf{S} = \hat{\mathbf{S}}(\mathbf{F}_e)\,, \quad \hat{\mathbf{S}}(\mathbf{F}_e) = \frac{\partial \hat{\psi}}{\partial \mathbf{F}_e}(\mathbf{F}_e)\,; \tag{3.9}$$

the (Cauchy) stress may easily be derived through standard formulas as

$$\mathbf{T} = \mathbf{S}\mathbf{F}_e^T - p\mathbf{I}\,, \tag{3.10}$$

where the field $p : \mathcal{R} \to \mathcal{R}$ is the indeterminate pressure field accounting for the incompressibility constraint. The stress measure $\mathbf{S}_R$ on the reference configuration, known as the referential stress measure, is given by

$$\mathbf{S}_R = \mathbf{T}\,\mathbf{F}^* = \mathbf{S}\,\mathbf{F}_o^* - p\mathbf{F}^*\,, \tag{3.11}$$

where the cofactor $\mathbf{A}^*$ of a tensor $\mathbf{A}$ is defined as $\mathbf{A}^* = (\det\mathbf{A})\,\mathbf{A}^{-T}$.

Several forms of strain energy function have been proposed to describe the passive material response of soft tissues such as the myocardium and arterial walls.[2] Here, we focus on the contribution of the active fibres to the material response of the tissue and set up the mechanical behaviour as simple as possible by describing the response of the incompressible hyperelastic body through the Mooney–Rivlin strain energy function, whose representation form is

$$\hat{\psi}(I_e, \mathbb{I}_e) = \frac{\alpha}{2}(I_e - 3) + \frac{\beta}{2}(\mathbb{I}_e - 3)\,. \tag{3.12}$$

Here, I_e and $\mathbb{I}_e$ are the first and the second invariant of $\mathbf{C}_e$ (or, equivalently, of $\mathbf{B}_e$)

$$I_e = \mathbf{C}_e \cdot \mathbf{I}\,, \quad \mathbb{I}_e = \frac{1}{2}((\mathbf{C}_e \cdot \mathbf{I})^2 - \mathbf{C}_e^2 \cdot \mathbf{I})\,, \tag{3.13}$$

and α, β are elastic moduli; the neo-Hookean strain energy function is recovered for $\beta = 0$. By using (3.9) and (3.10), the representation formula of the stress $\mathbf{T}$ corresponding to a Mooney–Rivlin material takes the form

$$\mathbf{T} = \hat{\mathbf{T}}(\mathbf{B}_e) - p\mathbf{I}\,, \quad \hat{\mathbf{T}}(\mathbf{B}_e) = (\alpha + \beta\,\mathbf{I}\cdot\mathbf{B}_e)\,\mathbf{B}_e - \beta\,\mathbf{B}_e^2\,. \tag{3.14}$$

[2] Extensive reviews may be found in [2] and in [16].

A representation formula for the stress in terms of the visible and the active deformation fields is now straightforward: using $(3.3)_2$ we may write

$$\mathbf{T} = \hat{\mathbf{T}}(\mathbf{F}\,\mathbf{C}_o^{-1}\,\mathbf{F}^T) - p\mathbf{I}\,; \tag{3.15}$$

the active deformation $\mathbf{F}_o$ is interwoven with the summands of $\hat{\mathbf{T}}(\mathbf{F}\,\mathbf{C}_o^{-1}\,\mathbf{F}^T)$, through both $\mathbf{C}_o$ and $\mathbf{F} = \mathbf{F}_e\,\mathbf{F}_o$, in a way that does not allow for an additive decomposition of the stress into a passive component, depending on $\mathbf{F}$ only, and an active component, depending on $\mathbf{F}_o$. We conclude this section with an important remark.

The present model of active tissues aims to describe at a macroscopic level the time-varying contractile behaviour of an elastic medium excited by an external source (a prototypical example is the cardiac myocardium excited by the activation transmembrane potential). Typically, in such applications, time scale is that of the cardiac cycle; thus, inertia forces may be neglected, but the stress power associated with the time varying field $\mathbf{F}_o$ should not be disregarded.

When such stress power component is accounted for, the present model needs to be generalized not to violate two relevant constitutive issues: the first concerns the requirement that the power expended on the motions compatible with the incompressibility constraint by the reactive stress be zero; the second is the requirement that, within the model, a dissipation inequality holds.

In [14], it is shown as the theoretical framework appropriate for setting the enlarged problem is that one defined in [19], where the energetic issues related to the generalized model are largely discussed.

4 Isotropic Active Fibres

At macroscopic level, the fibre architecture of a tissue determines the lines of activation in the body, that is, the directions along which active fibres contract: from our point of view, tension is an outcome of contraction.

Here, we discuss the architecture of isotropic active fibres (as in [10, 11]); in this case, the active deformation field $\mathbf{F}_o$ and the corresponding active strain $\mathbf{C}_o$ are given by

$$\mathbf{F}_o = \gamma_o\mathbf{I}\,, \quad \mathbf{C}_o = \gamma_o^2\,\mathbf{I}\,. \tag{4.1}$$

By accounting for the special representation form $(4.1)_2$, (3.15) may be written as follows

$$\mathbf{T} = \frac{1}{\gamma_o^4}\Big((\alpha\gamma_o^2 + \beta I_1)\,\mathbf{B} - \beta\,\mathbf{B}^2\Big) - p\mathbf{I}\,, \quad I_1 = \mathbf{I}\cdot\mathbf{B}\,. \tag{4.2}$$

Equation (4.2) shows that, even for the simple case of isotropic activation, the dependence of stress on the visible and the active deformations is not representable as the sum of a passive component $\mathbf{T}_p = \hat{\mathbf{T}}(\mathbf{B})$, plus an active component $\mathbf{T}_a = \mathbf{T}_a(\gamma_o, \mathbf{B})$.

In the special case of *small* active deformations, this additive decomposition is indeed possible. Let us assume

$$\gamma_o = 1 + \varepsilon\gamma\,, \tag{4.3}$$

with $\varepsilon \ll 1$, a smallness parameter, and γ the approximation of order 1 in ε of γ_o; a power series expansion in ε for (4.2), up to the same order, yields

$$\mathbf{T} = \hat{\mathbf{T}}(\mathbf{B}) - p\mathbf{I} + \mathbf{T}_a\,, \quad \mathbf{T}_a = \gamma\,\mathbb{F}(\mathbf{B})\,. \tag{4.4}$$

Nonetheless, even if an additive decomposition is granted, an isotropic $\mathbf{F}_o$ (i. e., an isotropic fibres architecture) does not imply a spherical active stress: here, the active component $\mathbf{T}_a$ depends linearly on the intensity of the active deformation γ through the map $\mathbb{F} : \mathbb{S}ym \to \mathbb{S}ym$, a nonlinear function of the visible strain measure $\mathbf{B}$ defined by

$$\mathbb{F}(\mathbf{B}) = -2\Big((\alpha + 2\beta I_1)\mathbf{B} - 2\beta\mathbf{B}^2\Big)\,. \tag{4.5}$$

In the next section, with reference to a well-known problem of nonlinear elasticity, the so-called Rivlin cube, we show some results relative to isotropic activation.

5 The Active Rivlin Cube

We consider a cube of neo-Hookean, active material, described by the energy function (3.12) with $\beta = 0$; the cube is uniformly loaded by a pressure field acting on its faces. The sketch is simple but it is enough to present the consequences of our modeling procedure and to discuss the main differences in comparison with models proposed in the literature. We first briefly summarize the solution to the classical problem (see [17, 18]); we then solve the same problem for the case of isotropic activation.

5.1 The Classical Solution

The undeformed body $\mathcal{B}$ is a homogeneous unit cube of neo-Hookean material centred on an orthogonal coordinate system whose unit vectors are perpendicular to the faces of the cube, and is loaded uniformly by three identical pairs of equal and oppositely directed forces acting normally to its faces [18]. The referential (Piola–Kirchhoff) traction on the face with normal $\mathbf{e}_i$ $(i = 1, 2, 3)$ is

$$\mathbf{S}_R\mathbf{e}_i = f\,\mathbf{e}_i\,, \quad (i = 1, 2, 3)\,. \tag{5.1}$$

Rivlin studied homogeneous solutions of the form

$$\mathbf{S}_R = f\,\mathbf{I}\,, \quad \mathbf{F} = \lambda_i\,\mathbf{e}_i \otimes \mathbf{e}_i\,, \tag{5.2}$$

with $\lambda_i > 0$, satisfying the incompressibility condition $\lambda_1\,\lambda_2\,\lambda_3 = 1$. These solutions satisfy balance with zero body forces

$$\mathrm{Div}\,\mathbf{S}_R = \mathbf{0}\,, \tag{5.3}$$

and the traction conditions (5.1). Rivlin found seven possible states, defined by the stretches λ_i $(i = 1, 2, 3)$ such that

$$(\lambda_i - \lambda_j)\Big(f_o - (\lambda_i + \lambda_j)\Big) = 0\,, \quad i, j = 1, 2, 3\,, \tag{5.4}$$

with $f_o = f/\alpha$. The trivial state

$$\lambda_1 = \lambda_2 = \lambda_3 = 1 \tag{5.5}$$

is always a solution. Moreover, there is a value $f_o^* = (27/4)^{1/3}$ of the parameter f_o such that for $f_o < f_o^*$ the trivial state is the unique solution; for $f_o > f_o^*$, there are six further solutions, given by

$$\lambda_i = \lambda_j \quad \text{and} \quad 0 < \lambda_k < \frac{1}{3} f_o \quad \text{or} \quad \frac{1}{3} f_o < \lambda_k < f_o \,, \tag{5.6}$$

with $i, j, k = 1, 2, 3$ and $i \neq j \neq k$. For $f_o = f_o^*$ each pair of the above solutions collapses to the one characterized by having two stretches equal to $(f_o/3)^{-\frac{1}{2}}$, and the remaining one uniquely defined by the incompressibility constraint. In [17], Rivlin showed that, for $f_o < 2$, four solutions are stable with respect to arbitrary superposed infinitesimal homogeneous deformations: the trivial one and

$$\lambda_i = \lambda_j \quad \text{and} \quad 0 < \lambda_k < \frac{1}{3} f_o \,, \quad i, j, k = 1, 2, 3 \,, \quad i \neq j \neq k \,. \tag{5.7}$$

For $f_o > 2$, the trivial solution is no longer stable, and there remain only three stable solutions.

5.2 The Active Cube

Let us consider the undeformed and homogeneous unit cube $\mathcal{B}$ as consisting of neo-Hookean active tissue. We account for an isotropic distribution of active fibres through the introduction of an active deformation field $\mathbf{F}_o = \gamma_o \mathbf{I}$; in this case, the role of control parameter is played by the scalar field γ_o. We look for homogeneous solutions of the form (5.2); the corresponding elastic deformation $\mathbf{F}_e$ is

$$\mathbf{F}_e = \varphi_i \, \mathbf{e}_i \otimes \mathbf{e}_i \,, \quad \varphi_i = \frac{\lambda_i}{\gamma_o} \,; \tag{5.8}$$

moreover, the incompressibility constraint on $\mathbf{F}_e$ requires that

$$\varphi_1 \, \varphi_2 \, \varphi_3 = 1 \,, \tag{5.9}$$

that is,

$$\lambda_1 \, \lambda_2 \, \lambda_3 = \gamma_o^3 \,. \tag{5.10}$$

Assuming (3.11) and (4.1), the referential stress measure $\mathbf{S}_R$ takes the form

$$\mathbf{S}_R = \lambda_1 \, \lambda_2 \, \lambda_3 \left(\frac{1}{\gamma_o^2} \alpha \, \mathbf{F} - p \, \mathbf{F}^{-T} \right) . \tag{5.11}$$

By incorporating the incompressibility condition (5.10), substituting $(5.2)_2$ in (5.11), and eliminating the indeterminate pressure field, one finds that the stretches λ_i must satisfy

$$(\lambda_i - \lambda_j) \left(f_o - \gamma_o \, (\lambda_i + \lambda_j) \right) = 0 \,, \quad i, j = 1, 2, 3 \,. \tag{5.12}$$

Let us consider the set $\mathcal{P} \subset \mathbb{R}^+ \times \mathbb{R}^+$ of all parameters $(\gamma_o,\ f_o)$, and the curve $\mathcal{C} \subset \mathcal{P}$ defined by (see Fig. 1)

$$f_o = c\,(\gamma_o)^2\,, \quad c = \left(\frac{27}{4}\right)^{\frac{1}{3}}\,. \tag{5.13}$$

The curve $\mathcal{C}$ divides $\mathcal{P}$ into an upper part $\mathcal{U}$, and a lower part $\mathcal{L}$. For any pair $(\gamma_o,\ f_o) \in \mathcal{P}$, there is the trivial and symmetric solution

$$\lambda_1 = \lambda_2 = \lambda_3 = \gamma_o\,; \tag{5.14}$$

moreover, for $(\gamma_o,\ f_o) \in \mathcal{L}$, the trivial solution is unique; for $(\gamma_o,\ f_o) \in \mathcal{U}$, there are six further solutions, given by

$$\lambda_i = \lambda_j \quad \text{and} \quad 0 < \lambda_k < \frac{1}{3}\frac{f_o}{\gamma_o} \quad \text{or} \quad \frac{1}{3}\frac{f_o}{\gamma_o} < \lambda_k < \frac{f_o}{\gamma_o}\,, \tag{5.15}$$

with $i,\ j,\ k = 1, 2, 3$ and $i \neq j \neq k$. For $(\gamma_o,\ f_o) \in \mathcal{C}$, each pair of the above solutions collapses to the one characterized by having two stretches equal to $(f_o/(3\,\gamma_o))^{-\frac{1}{2}}$, and the third uniquely determined by the incompressibility constraint (5.10). As in the classical solution, here (5.14) is the only symmetric solution. Figure 2 shows the stretches versus the load f_o, for a given value of γ_o. Solid lines represent the solutions for which $0 < \lambda_k < \frac{1}{3}\,f_o/\gamma_o$ (stable solutions), and dashed lines those corresponding to $\frac{1}{3}\,f_o/\gamma_o < \lambda_k < f_o/\gamma_o$ (unstable solutions). We have plotted λ_k on the left, and $\lambda_i = \lambda_j$ on the right; the classical Rivlin solution is recovered for $\gamma_o = 1$.

Thus, it turns out that isotropic distortions $\mathbf{F}_o = \gamma_o\,\mathbf{I}$ modify the critical value of the load f_o below which the trivial state (5.14) is the unique solution: precisely, a contraction $\gamma_o < 1$ reduces the critical load and an expansion $\gamma_o > 1$ increases it. In Fig. 1 the locus of critical pairs $(\gamma_o,\ f_o)$ is represented by the curve $\mathcal{C}$; the value $\gamma_o = 1$ identifies the classical critical parameter f_o^* discussed in [17].

It is worth noting that modeling the isotropic active structure of the incompressible cube simply by adding a spherical active stress to any given constitutive relation

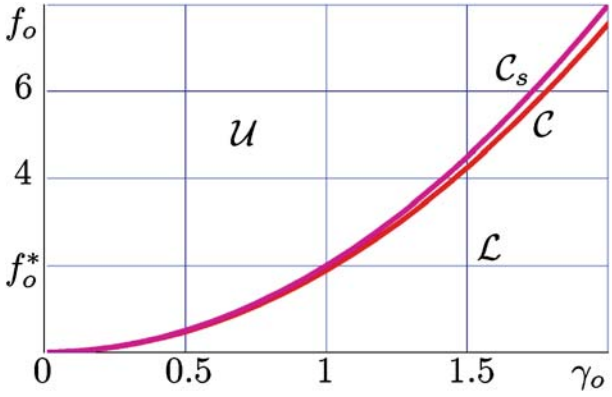

Fig. 1 Curve $\mathcal{C}$ is the locus defining the critical values of the parameters f_o and γ_o: above it, there are six solutions, plus the trivial one; this curve shows how the critical value f_o for which the trivial solution is the only one increases with the distortion γ_o. Curve $\mathcal{C}_s$ represents the limit above which the trivial solution becomes unstable

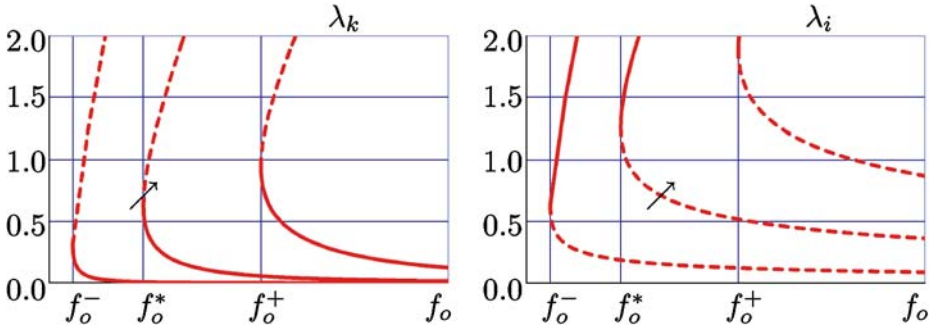

Fig. 2 Stretch λ versus load f_o, for given distortion γ_o. We plot λ_k on the left, and $\lambda_i = \lambda_j$ on the right, for $i, j, k = 1, 2, 3$, and $i \neq j \neq k$. Stable solutions are plotted with solid line; arrows denote increasing values of γ_o, valued 0.5, 1, 1.5. To each distortion γ_o, there correspond a critical value of the loading parameter f_o, denoted by: f_o^- for $\gamma_o = 0.5$, f_o^* for $\gamma_o = 1$, and f_o^+ for $\gamma_o = 1.5$

for incompressible materials turns out to be ineffective. To be more precise, in a constitutive relation that reads as

$$\mathbf{T} = \hat{\mathbf{T}}(\mathbf{B}) - p\mathbf{I} + \tau_o \mathbf{I}\,, \tag{5.16}$$

with τ_o having the role of control parameter (see [10]), any variation of τ_o is compensated by the pressure field p such that the difference $(\tau_o - p)$ does not change: incompressibility makes it impossible to trigger any activation of the tissue through a spherical active stress.

5.3 Stability Issues

Let us introduce a relaxed energy function $\hat{\psi}^r$, derived from (3.7) by adding an indeterminate pressure field p playing the role of Lagrange multiplier

$$\hat{\psi}^r(\mathbf{F}_e) = \hat{\psi}(\mathbf{F}_e) - p(\det\mathbf{F}_e - 1)\,; \tag{5.17}$$

then, the relaxed energy density per unit reference volume ψ^r is related to $\hat{\psi}^r$ by

$$\psi^r = (\det\mathbf{F}_o)\,\hat{\psi}^r\,. \tag{5.18}$$

The stability of each of the previous states may be discussed with respect to arbitrary superposed infinitesimal deformations in terms of the energy functional

$$\Phi\{\mathbf{F}\} = \int_{\mathcal{B}} (\psi^r - \mathbf{S}_R \cdot \mathbf{F})\,. \tag{5.19}$$

The functional Φ allows for the following representation:

$$\Phi\{\mathbf{F}\} = \int_{\mathcal{B}} \Big(\big(\hat{\psi}(\mathbf{F}_e) - p\,(\det\mathbf{F}_e - 1)\big)\det\mathbf{F}_o - \mathbf{S}_R \cdot \mathbf{F} \Big)\,,\ \ \mathbf{F} = \mathbf{F}_e\mathbf{F}_o\,; \tag{5.20}$$

the variations of $\Phi\{\mathbf{F}\}$ must be evaluated with respect to any incremental visible deformation $\mathbf{F}(\varepsilon) = \mathbf{F} + \varepsilon\,\mathbf{H}$. Corresponding to any $\mathbf{F}(\varepsilon)$, there is an incremental elastic deformation $\mathbf{F}_e(\varepsilon) = \mathbf{F}_e + \varepsilon\,\tilde{\mathbf{F}}_e$ with $\tilde{\mathbf{F}}_e = \mathbf{H}\,\mathbf{F}_o^{-1}$; moreover, because

$$\det \mathbf{F}_e(\varepsilon) = 1\,, \tag{5.21}$$

we note that

$$\det \mathbf{F}_e(\varepsilon) - 1 = \varepsilon\,(\mathbf{F}_e^{*} \cdot \mathbf{H}\,\mathbf{F}_o^{-1}) + O(\varepsilon^2) \tag{5.22}$$

requires $\mathbf{H}$ to satisfy the constraint

$$\mathbf{H} \cdot \mathbf{F}^{-T} = 0\,. \tag{5.23}$$

Finally, an equilibrium state is said to be stable if the corresponding stationary value of Φ is a relative minimum, that is, if

$$\delta^2\Phi = \frac{d^2\Phi}{d\varepsilon^2}\,(\mathbf{F} + \varepsilon\mathbf{H})|_{\varepsilon=0} > 0\,, \tag{5.24}$$

for all admissible $\mathbf{H}$ which satisfy the incompressibility constraint (5.23). Accounting for the representation form of $\mathbf{F}_o$ in terms of the scalar field γ_o, the condition (5.24) may be written as

$$\delta^2\Phi = \int_{\mathcal{B}} \Big(\frac{\alpha}{\gamma_o^2}\,\mathbf{H} \cdot \mathbf{H} - p\,((\mathbf{F}^{-T} \cdot \mathbf{H})^2 - \mathbf{H}^T\mathbf{F}^{-T} \cdot \mathbf{F}^{-1}\mathbf{H}) \Big) \gamma_o^3 > 0 \tag{5.25}$$

for any $\mathbf{H}$ such that

$$\frac{H_{11}}{\lambda_1} + \frac{H_{22}}{\lambda_2} + \frac{H_{33}}{\lambda_3} = 0\,. \tag{5.26}$$

This analysis shows that the states

$$\lambda_i = \lambda_j \quad \text{and} \quad 0 < \lambda_k < \frac{1}{3}\,\frac{f_o}{\gamma_o}\,, \quad i,\, j,\, k = 1,\, 2,\, 3\,, \quad i \neq j \neq k\,, \tag{5.27}$$

are stable. Moreover, it is found that the trivial solution is stable below the curve $\mathcal{C}_s \subset \mathcal{P}$ defined by $f_o = 2\,\gamma_o^2$, and unstable above it; thus, in the small region bounded between $\mathcal{C}$ and $\mathcal{C}_s$ four stable solutions are possible, see Fig. 1.

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