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THE CONSTITUTIVE BEHAVIOUR OF PASSIVE HEART MUSCLE TISSUE: A QUASI-LINEAR VISCOELASTIC FORMULATION

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Abstract—A quasi-linear viscoelastic law with a continuous relaxation spectrum describing triaxial constitutive behaviour of heart muscle tissue is presented. The elastic response of the viscoelastic law is anisotropic, while the relaxation behaviour is assumed isotropic. The law is designed for a biphasic description (fluid-solid) of the myocardial tissue. Biaxial and uniaxial stress-strain curves from the literature are used to evaluate the parameters of the model. The non-linear elastic response, the difference between fibre and cross-fibre stiffness, the phenomenon of stress relaxation, the stiffening of the stress-strain relationship with increasing strain rate and the weak frequency dependency of the dissipated energy during cyclic loading are fairly well described by the proposed law. However, it is found that the model produces realistic values for the dissipated energy during cyclic loading only when relaxation parameter values are chosen which result in an overestimation of the stress relaxation data by more than 100%. This finding may indicate non-quasi-linearity of viscoelasticity of passive heart muscle tissue.

NOMENCLATURE

- scalar
- vector
- second order tensor
- conjugate of second order tensor
- inverse of second order tensor
- conjugate of the inverse of the second order tensor
- unit second order tensor
- dot product of the second order tensor and the vector

INTRODUCTION

At present there are no reliable means to measure wall stresses directly in the intact heart muscle (Huisman et al., 1980). Therefore, we must rely on mathematical models to predict those stresses. Knowledge of the constitutive relation of heart muscle tissue is a prerequisite to the reliability of any ventricular model. Full quantification of a three-dimensional constitutive relation can only be done on the basis of triaxial test data. At present, direct measurements from excised tissue specimens have been limited to uniaxial tests conducted on papillary muscles or strips of trabeculae (Sonnenblick, 1964; Pinto and Fung, 1973; Loeffler and Sagawa, 1975; Mirsky and Pasipoularides, 1980; Van Heuningen et al., 1982), and biaxial tests on strips of the left ventricular wall (Demer and Yin, 1983; Yin et al., 1987).

Demer and Yin (1983) characterize the myocardial tissue as a highly non-linear viscoelastic material (Fig. 1). A viscoelastic material has no well-defined
stress-free state, i.e. the stress-free state of the specimen may occur at different states of deformation. Demer and Yin did not specify the deformation of their specimen in the zero-load condition. All deformation following the application of the load was characterized with respect to this poorly defined reference state. In some uniaxial tests (e.g. Van Heuningen et al., 1982), however, the state of deformation of the specimen is specified by a measured sarcomere length. Thus, to characterize the constitutive behaviour, we use the uniaxial data of Van Heuningen et al. (1982) in addition to the relaxation data of Pinto and Fung (1973) and the biaxial data of Demer and Yin (1983).

The aim of this paper is to formulate a constitutive law of heart muscle tissue which (1) approximates the known viscoelastic and anisotropic properties of the tissue as well as possible and (2) can easily be implemented into a biphasic finite element code of the myocardial wall. In order to further evaluate the capability of the law to predict known properties of the diastolic left ventricle as a whole, the constitutive law is implemented into a two-phase finite element model of the left ventricle (Huyghe et al., 1991).

THE CONSTITUTIVE LAW

The myocardial tissue is represented as a mixture of a fluid component and a solid component, both of which are incompressible. This two-phase tissue model on the whole, however, can change its internal volume by expelling blood from its coronary vessels. The total Cauchy stress $\sigma$ in the mixture is the difference between the effective Cauchy stress $\sigma^e$ due to deformation of the solid and the intramyocardial pressure $p$. The constitutive law of the solid component is expressed as a relationship between the effective second Piola-Kirchhoff stress $\mathbf{S}^e$ and Green strain tensor $\mathbf{E}$ and time $t$, and describes the rheological behaviour of the empty solid matrix devoid of coronary blood. The stress $\mathbf{S}$ is split up into two components, one resulting from elastic volume change of the myocardial tissue $\mathbf{S}^e$, the other from viscoelastic shape change of the myocardial tissue $\mathbf{S}^v$:

$$\mathbf{S} = \mathbf{S}^e + \mathbf{S}^v(\mathbf{E}, t).$$

The elastic stress $\mathbf{S}^e$ is derived from the isotropic strain energy function $C$ by:

$$\mathbf{S}^e = \frac{\varepsilon C}{\varepsilon}$$

with

$$C = \frac{\varepsilon}{2} (J - 1)^2$$

where $\varepsilon$ is the volumetric modulus of the empty solid matrix devoid of coronary blood and $J$ is the Jacobian.

The viscoelastic component $\mathbf{S}^v$ is described in a spectral form of quasi-linear viscoelasticity as proposed by Fung (1981, pp. 226–238):

$$\mathbf{S}^v(\mathbf{E}, t) = \int_{-\infty}^{t} G(t - \tau) \frac{d}{dt} \mathbf{S}^v(\mathbf{E}(\tau))d\tau. \quad (4)$$

$\mathbf{S}^v$ is the anisotropic elastic response of the material. The reduced relaxation function $G(t)$ is a scalar function derived from a continuous relaxation spectrum $\Sigma(t)$ (Fung, 1973, p. 232):

$$G(t) =\left[1 + \int_{0}^{\infty} \Sigma(t)e^{-\gamma t}dt\right]\left[1 + \int_{0}^{\infty} \Sigma(t)dt\right]^{-1} \quad (5)$$

where $\Sigma(t)$ represents the continuous relaxation spectrum of passive heart muscle. The reduced relaxation function of a papillary muscle has been determined experimentally by Pinto and Fung (1973). A suitable spectrum fitting the experimental data of Pinto is:

$$\Sigma(t) = d + \Sigma_0 \quad \text{for} \quad \tau^1 \leq t \leq \tau^2$$

$$\Sigma(t) = 0 \quad \text{for} \quad \tau^1 < t < \tau^2 \quad (6)$$

where $d$ and $\Sigma_0$ are damping constants (Maessen, 1983). The spectrum is discretized in a number of parallel Maxwell elements. Two Maxwell elements per decade are sufficient to yield an accuracy of $G(t)$ of less than 4% for values of $t$ between $\tau^1$ and $\tau^2$. The elastic response $\mathbf{S}^e$ is taken to be orthotropic. The three axes of orthotropy are specified in Fig. 2 by the three orthonormal vectors $\mathbf{B}_1$, $\mathbf{B}_2$, $\mathbf{B}_3$. The vector $\mathbf{B}_3$ is parallel to the initial local fibre orientation. In this local coordinate system the stress $\mathbf{S}^e$ and the strain $\mathbf{E}$ are written as:

$$S^e_{ij} = B_i \cdot S^e \cdot B_j$$

$$E_{ij} = B_i \cdot \mathbf{E} \cdot B_j$$

Fig. 2. The elastic response of the tissue is described in terms of a local orthonormal basis ($\mathbf{B}_1$, $\mathbf{B}_2$, $\mathbf{B}_3$). The vector $\mathbf{B}_3$ is parallel to the initial local fibre orientation, $\mathbf{B}_1$ is perpendicular to the wall, and $\mathbf{B}_2$ is perpendicular to $\mathbf{B}_1$ and $\mathbf{B}_3$. 
and are mutually related by a strain energy function $W$:

$$S'_{ij} = \frac{1}{2} \left( \frac{\partial W}{\partial E_{ij}} + \frac{\partial W}{\partial E_{ji}} \right)$$  \hspace{1cm} (9)$$

with

$$W = c'^e (\exp(a^e F_{11}) - a^e F_{11}) + \sum_{i=1}^{2} \left[ \exp(a^i E_{ii}) - a^i E_{ii} \right]$$

$$- \sum_{i=1}^{2} \sum_{j=1}^{2} \left[ \exp(a^i E_{ij}) (\exp(a^j E_{jj}) - a^j E_{jj}) - \frac{E_{ij}^2}{2} \right]$$

$$+ c' \left\{ \exp(a^e (E_{12} E_{12} + E_{13} E_{13} + E_{23} E_{23})) - 1 \right\}$$  \hspace{1cm} (10)$$

where

- $c'$: initial normal stiffness
- $c^e$: initial shear stiffness
- $a^e$: exponential factor in cross-fibre stiffness
- $a^i$: exponential factor in fibre stiffness
- $a^f$: exponential factor in biaxial stiffness
- $a^s$: exponential factor in shear stiffness.

The functions $W$ and $C$ (equation (3)) and their derivatives with respect to the Green strain are zero for the reference state $E_{ij} = 0$, or else they are positive. The terms of expression (10) containing $u^e$ and $u^f$ ensure an exponential increase of the stress under uniaxial loading. The terms containing $a^e$ cause biaxial stiffness to be higher than uniaxial stiffness.

**EVALUATION OF PARAMETER VALUES**

We evaluate the volumetric modulus ($c'$), the initial normal stiffness ($c^e$), the fibre stiffness ($a^f$), the relaxation constants ($d$, $\Sigma_0$, $\tau^1$, $\tau^2$), the cross-fibre stiffness ($a^e$) and the biaxial stiffness ($a^s$). Whenever possible, preference will be given to data on isolated myocardial specimens, because, to our knowledge, no experimental data on this topic are available.

For the volumetric modulus ($c'$) we circumvent the problem of lack of data on isolated myocardial specimens as follows. Morgenstern et al. (1973) measured tissue volume changes of heart muscle following changes in coronary perfusion pressure. We assume that these changes of coronary pressure induce variations of the intramyocardial pressure of only a few mmHg. Morgenstern's data apply to a perfusion pressure range of 8-24 kPa. According to his data, an increase in perfusion pressure of 1 kPa yields an increase in myocardial volume of about 0.5%. A change in perfusion pressure from 8 to 24 kPa corresponds therefore to an increase in myocardial volume of about 8%. Let us now impose a volume increase to the model of 8% ($\Delta J = 0.08$). According to equations (2)-(3), the resulting stress change $\Delta S'$ equals approximately:

$$\Delta S' = c' \Delta J \frac{dJ}{dE}$$

$$= c' \Delta J \frac{dJ}{dE}$$

Assuming small deformations and neglecting the stress $S'$ (because Morgenstern's experiment involves primarily volume change), one finds the passive stress change:

$$\Delta S = \Delta S' = c' \Delta J$$  \hspace{1cm} (12)$$

Considering that the tensile stress (12) in the fibre network is balanced by the intramyocardial pressure, we find that:

$$p = c' \Delta J$$  \hspace{1cm} (13)$$

Equation (13) shows that for a relative volume change of 8%, the intramyocardial pressure increases about 0.8 kPa, if we choose:

$$c' = 10 \text{ kPa}$$  \hspace{1cm} (14)$$

Using now the experimental data on uniaxial loading shown in Fig. 3, we can evaluate $c^e$ and $a^f$. Neglecting viscous relaxation and transverse stiffness of the specimen, the model yields the relationship of the Cauchy fibre stress $s_{33}$ to the ratio of current to initial sarcomere length $l'/l^*$:

$$s_{33} = \left( \frac{l'}{l^*} \right)^2 S_{33} = \left( \frac{l'}{l^*} \right)^2 c^e a^f \exp \left( \frac{a^f (l^*)^2 - (l')^2}{2 (l')^2} \right).$$  \hspace{1cm} (15)$$

Equation (15) fits the experimental data fairly well (Fig. 3), when using:

$$c^e = 0.01 \text{ kPa}$$  \hspace{1cm} (16)$$

$$a^f = 18.$$  \hspace{1cm} (17)$$

Notice that for these values, it holds that:

$$c^e a^f < c'.$$  \hspace{1cm} (18)$$

Equation (18) mathematically translates the common experience that it is much easier to change the shape of
a muscle than to change its volume. That the stress $S'$ is neglected in equation (17) also is justified by equation (18). The relaxation parameters are evaluated from the experimental data of Pinto and Fung (1973). The resulting values are (Fig. 4):

$$
\begin{align*}
    d &= 0.01 \\
    \Sigma^0 &= 0.000185 \text{ s}^{-1} \\
    \tau_1 &= 0.01 \text{ s} \\
    \tau_2 &= 1000 \text{ s}.
\end{align*}
$$

(19)

The value of $a'$ is chosen such that the ratio of anisotropy in the model is of the same order of magnitude as in the experimental data of Demer and Yin (1983). Another finding of Demer and Yin is that their specimens were on the average stiffer under biaxial loading than under uniaxial loading. The model is able to reproduce their data on biaxial stiffness quantitatively, if we choose:

$$
\begin{align*}
    \alpha' &= 15 \\
    c' &= 12.
\end{align*}
$$

(20)

Using the above values for $a'$, $d'$, $c'$, $\alpha'$, $\tau$ and $\Sigma^0$, it is found that the model fits the average loading curves of Demer and Yin fairly well (Fig. 5) provided that the average of the reference states of the specimens used in their biaxial experiments correspond to the strain:

$$
E_{ij} = \begin{pmatrix} -0.13 & 0 & 0 \\ 0 & 0.08 & 0 \\ 0 & 0 & 0.08 \end{pmatrix}
$$

(21)

The question of whether or not fluid pressure and/or flow within the specimen are likely to affect the stress-strain relationships observed by Demer and Yin (1983) is addressed by computing the results of Fig. 6 successively by means of a single phase incompressible approach (i.e. fluid flow is negligible, permeability = 0), single phase compressible approach (i.e. fluid pressure gradient is negligible) and a two-phase approach. The finite element code described in Huyghe et al. (1991), is used for all three computations. The stresses in the loading directions are virtually the same for all three computations indicating that fluid flow and/or pressure does not affect these stresses significantly. Figure 7 shows the results of the same simulation as Fig. 6, except that the value of $d$ is multiplied by 1000:

$$
\gamma = 10.
$$

(22)

Figure 8 shows the dynamic stiffness of a papillary muscle when subjected to small sinusoidal length variations (4% of total length peak-to-peak) as a function of frequency. The frequency response of the
Fig. 5. Comparison of loading curves of model (broken lines) and experiment [solid lines, Demer and Yin (1983)] for isometric uniaxial loading. Unloading curves are not drawn. The left panel shows the fibre stress–strain relation at constant cross-fibre strain, the right panel shows cross-fibre stress–strain relations at constant fibre strain. Both the numerical and the experimental curves pertain to the situation after preconditioning. The rates of loading and unloading are 0.05 s⁻¹ in the model, and are unknown in the experiment. In the model, 10 Maxwell elements are used.

Fig. 6. Loading and unloading curves of the model for equibiaxial ($E_{33} = E_{22}$) loading of myocardial strips. $S_{33}$ is the passive second Piola–Kirchhoff fibre stress and $E_{33}$ is the Green–Lagrange fibre strain. The relaxation parameter $d$ is evaluated from the relaxation experiments of Pinto and Fung (1973). Ten Maxwell elements are used.

Fig. 7. Same computation as in Fig. 6 but after a thousand-fold increase of the relaxation factor $d$.

The constitutive law

The proposed law splits myocardial stress into two components: an elastic stress resulting from volume change of the myocardial tissue and a viscoelastic stress resulting from shape change. The experimental results of Pinto and Fung (1973), Noble (1977), Demer and Yin (1983, Fig. 1) and Yin et al. (1987), show inelastic behaviour. Strain components related to volume change are much smaller than the strain components associated with shape change in these experiments. Shape change, therefore, is assumed to generate viscous effects in the model. To the authors' knowledge, however, there is no experimental evidence yet that significant viscous effects occur during volume change. As viscous stresses are associated with shear rate, and little shear occurs during volume change, the stress $S_{33}$, governing the volume change of the empty matrix, is taken to be elastic. Since, to our knowledge, no anisotropic relaxation has been re-
Fig. 8. Simulated and experimental dynamic stiffness as a function of the frequency of the sinusoidal strain oscillations of 0.04 peak-to-peak about an initial Green strain of 0.24. Dynamic stiffness is normalized with respect to its value at 0.01 Hz. The experimental data pertain to rabbit papillary muscle and are borrowed from Pinto and Fung (1973). The model simulations are computed for $S^0=0.000185$ s$^{-1}$ (relaxation constant), $t^1=0.01$ s (lower bound of the relaxation spectrum), $t^2=1000$ s (upper bound of the relaxation spectrum) and for different values of the relaxation constant $d$.

ported in the literature, we assume for the sake of simplicity that the relaxation is isotropic. Hence $G(t)$ is a scalar valued function in equation (3).

The function $W$ reflects the anisotropic properties of myocardial tissue measured by Demer and Yin (1983). A closer look at the expression (10) reveals that the arguments of the exponential functions contain, either linear expressions in the normal strains, or quadratic expressions in the shear strains. A consequence of this choice is that, whereas the shear stresses resulting from shear strain of opposite sign are equal and of opposite sign, the normal stresses resulting from normal strain of opposite sign are unequal and of opposite sign (Fig. 9). Under tensile load, we expect the tissue fibres to generate exponentially increasing stresses in the loading direction (Fig. 10(A)). Under compressive load, however, the fibres in the loading direction tend to buckle (Fig. 10(B)). This behaviour is mathematically expressed by a low compressive stress at negative normal strains (Fig. 9). However, the model as a whole exhibits high compressive stiffness, because during compression transversally oriented fibres are subjected to tensile load via the stresses $S^c$ and the fluid pressure $p$ (Fig. 10(B)). The expression (10) contains an important assumption saying that the properties of the myocardial tissue are the same in the cross-fibre directions $B_{ij}$ and $B_{ji}$. Due to lack of experimental data on the stiffness in the transmural direction ($B_z$), this hypothesis cannot be checked.

Evaluation of parameter values

In experiments, the size of the hysteresis loop in the stress–strain relationship of heart muscle is fairly constant within a large range of loading rates (Pinto and Fung, 1973; Demer and Yin, 1983, Fig. 1). Also, it is seen that even for a large increase in loading rate, stiffening of the tissue is small (Pinto and Fung, 1973; Demer and Yin, 1983). The elastic response of the tissue is definitely non-linear: with increasing strain, the stiffness of the tissue increases. Each of these features of myocardial viscoelasticity are well described by the model. Comparison of this model
simulation pictured in Fig. 6 with Fig. 1 reveals that
the model simulation is qualitatively concordant with
the experiment: a faster loading rate results in a
slightly stiffer behaviour, and the stress–strain rela-
tionship at each loading rate is a closed hysteresis loop
followed in clockwise direction. However, the hyste-
resis loop of the model is much narrower than the
hysteresis loop of the experiment.

The hypothesis that flow of fluid relative to the solid
is a key to this discrepancy is debilitated by two
arguments. (1) Unlike the influence of viscoelasticity,
the influence of fluid flow within the specimen on the
measured stress–strain–time relationships depends on
the size of the specimen. The characteristic time
constant of creep flow depends quadratically on the
thickness of the specimen. On the basis of equa-
tion (17) in Huyghe et al. (1991), the time constant
associated with fluid flow in a specimen of 2 mm
thickness would be of the order of 0.1 s. In fact, the
classic manner of eliminating biphasic effects in an
experiment is to use specimens which are sufficiently
thin so as to obtain a time scale of the biphasic
phenomenon much smaller than that of the loading
scheme. In our judgement, this condition is met in the
experiments of Demer and Yin (1983). (2) Comparison
of incompressible single phase, compressible single-
phase and two-phase simulations of their experiments
indicate that changes in volume of the myocardial
tissue specimen have little effect on the computed
stress–strain curves in the loading direction, but rather
affect the strain in the direction perpendicular to the
loading direction. Further experimental verification of
the hypothesis of fluid flow might include experiments
with specimens of varying thickness.

Multiplication of the value \( d \) by 1000 yields a better
size of the hysteresis loop (Fig. 7). However, the value
\( d = 10 \) results in a relaxation function which disagrees
with the experimental relaxation data of Pinto and Fung (Fig. 4). This discrepancy occurs irrespective of the
mathematical tool used to fit the relaxation data,
and is therefore inherent to the comparison of a quasi-
linear law and the experimental data. Various ex-
planations can be found for this discrepancy. For
example, the large hysteresis loop measured by Demer
and Yin could be an experimental artefact: they point
out in their paper that the hysteresis loop could be
enlarged by the presence of needles used to hold the
specimen. However, hysteresis loops of a similar order
of magnitude are measured on rabbit papillary muscle
(Pinto and Fung, 1973; Fung, 1973). The stress relaxa-
tion data of Pinto and Fung (1973) used in Fig. 4 are
clearly consistent with data of other investigators. Pinto
and Patitucci (1980) measured a reduction of stress of
the order of 40–50% of the initial stress value after
holding the specimen at a constant strain for 15 min.
This result was found for rabbit papillary muscle, cat
trabecula and papillary muscle and circumferential and
longitudinal strips of a cat myocardium. Little and Wead (1971) measured stress relaxation of rabbit
papillary muscles: after keeping the muscle at a con-
stant strain for 5 min, the stress was found to decrease
by 15–30%. Similar values are mentioned by Lundin
(1944) and Hoffman et al. (1968). It is therefore very
unlikely that a papillary muscle subjected to a con-
stant strain of 30% should lose most of its tension
within one quarter of an hour, as predicted by the
model for the value \( d = 10 \) (Fig. 4). Significant negative
stresses are computed at positive strains. This phe-
nomenon can easily be explained physically on the
basis of a shift of the zero stress state. However, the
size of this shift needed to justify the result seems
unrealistic.

The model is also evaluated with respect to experi-
mental data on the frequency response of papillary
muscle [Fig. 8, after Pinto and Fung (1973)]. The
dynamic stiffness depends weakly upon the frequency,
indicating that lower values of \( d \) are more realistic as
far as frequency response is concerned. The experi-
ments of Noble (1977) point in the same direction as
they indicate that the dynamic stiffness of a papillary
muscle is poorly dependent on stretch rate.

It is beyond the scope of this study to decide
whether the discrepancies concerning the parameter
choice are due to specimen-to-specimen variability or
to non-quasi-linearity of the viscous properties of
heart muscle. However, considering the large gap
between the values of \( d \) computed from the different
types of experiments, we tend to believe that passive
myocardial viscoelasticity is not quasi-linear. Quasi-
linear viscoelasticity has been chosen for numerical
reasons. An infinite range of non-linear viscoelastic
laws which are not quasi-linear, and which are there-
fore numerically less advantageous, are not considered
in this research. It is possible that some of these non-
linear laws fit the experiments much better than quasi-
linear viscoelasticity does.

This model versus other models

The constitutive behaviour of myocardial tissue has
been the subject of many investigations in previous
years. Reviews of the work in this area is found
elsewhere (Mirskey, 1976; Yin, 1981; Humphrey and
Yin, 1987). We restrict ourselves to more recent work.

Since the biaxial loading experiments of Demer and
Yin (1983) there have been several attempts to develop
a mathematical formulation which is able to describe
their results. Yin et al. (1987) proposed a two-dimen-
sional strain energy function in terms of non-integral
powers of strain. They do not consider shear strain
and assume incompressibility and hyperelastic behavi-
our. It is shown that unlike quadratic strain energy
functions, the proposed strain energy function is able
to fit the results on biaxial stress loadings for different
ratios of fibre to cross-fibre strain. However, they also
point out that the proposed law is undefined for
compressive strains. Humphrey and Yin (1987) pro-
posed another law which does not have this dis-
advantage. The proposed strain energy function is
defined for both tensile and compressive strains and is
suitable to describe biaxial data for different ratios of
fibre to cross-fibre strain. An extended version of this law including shear strains and volume change would be adequate to be introduced into a two-phase finite element model. Considering however that stresses computed on the basis of their strain energy function do not vanish for zero strains, the finite element mesh would have a non-equilibrium reference state. A strain energy formulation which includes shear strain and volume change is the formulation proposed by Chuong and Fung (1984) for arterial walls. However, this form of strain energy is unable to fit the biaxial and uniaxial data of Yin and co-workers with a single set of parameter values.

An interesting approach to the constitutive behaviour of heart muscle tissue is the one proposed by Horowitz et al. (1988). They are able to introduce into their model quantitative histological data on the fibre structure of the myocardial tissue. While shear strain is considered in their research, they do assume incompressibility and hyperelasticity. It is obvious that their microstructural approach has more capabilities to it than the phenomenological approach chosen by ourselves and other researchers. As long as quantitative data on the histology of myocardial tissue are not available, however, we prefer the simplicity of a phenomenological approach particularly when considering the additional complication involved with introducing compressibility and viscoelasticity into a microstructural model. As pointed out by Humphrey and Yin (1987), implementation of a phenomenological model into a finite element formulation is more straightforward than implementation of Horowitz's model.

CONCLUSION

The experimental data on the mechanical behaviour of isolated myocardial tissue specimens are insufficient to quantify the stress-strain-time relationship governing three-dimensional myocardial deformation. Lack of triaxial data, of shear data, of data on compressibility and compressive behaviour and incomplete data on viscous dissipation are the main obstacles to this goal.

In this study we have reconciled the experimental data into one single mathematical formulation, which can be used as a basis for further research. The formulation presented here is defined for all values of the strains. The passive stress in the heart muscle model is the sum of an elastic stress resulting from volume-change of the tissue, and a viscoelastic stress resulting from shape change of the tissue. Four parameters describe the relaxation behaviour ($s^1, s^2, d, \Sigma^0$), while seven parameters describe the elastic response ($c', c', c', d, a', a, a$). Some aspects of the constitutive behaviour of myocardial tissue—such as the non-linear elastic response, the ratio of fibre vs cross-fibre stiffness, stress relaxation, weak stiffening of the stress-strain relationship with increasing strain rate and weak frequency-dependency of the dissipated energy during cyclic loading—are fairly well described by the proposed law. However, it appears that viscous dissipation during dynamic loading is underestimated relative to the viscous relaxation during constant deformation. This discrepancy may be due to the invalidity of the assumption of quasi-linearity of viscoelasticity of heart muscle. However, considering that quasi-linearity saves a large amount of memory in a finite element computation because of the applicability of the superposition principle, and that uncertainties remain as to whether the experimental data of excised myocardial strips actually reflect the properties of in vivo diastolic myocardium, we feel it is appropriate to implement the quasi-linear law into a finite element code and evaluate its validity on the level of the properties of the ventricular wall as a whole.

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