Muscle, the motor of movement: properties in function, experiment and modelling

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Abstract

The purpose of this paper is to review exemplary aspects of different views of skeletal muscle characteristics. A classical view of muscle characteristics plays a very important role in modelling of muscles and movement. However, it often also pervades concepts on which our understanding of muscle function is based. In this view length effects, velocity effects and effects of degrees of activation and recruitment are distinguished and, often implicitly, assumed to be independent effects. It will be illustrated that using the classical approach many valuable things may be learned about muscle function and adaptation. At the same time we should realize that such a classical approach is too limited for use in generating knowledge about properties of muscles during daily use. The use of scaling of force to estimate muscular properties during submaximal activity on the basis of properties during maximal activation is shown to be very inadequate. An alternative view is described and particular examples are provided of changes in length–force characteristics as a consequence of submaximal activation, previous length change, as well as the effect of short-term histories of these variables. In addition, effects of inhomogeneities of muscle in morphology as well as physiological properties are considered. It is concluded that length–velocity–force characteristics are not unique properties of a muscle, and that these characteristics are not only strongly influenced by actual effects of recruitment, firing frequency, shortening performed and actual velocity of shortening but also by the short time history of these factors. Therefore, length, velocity and activation cannot be considered as independent determinants of muscle functioning. It is also shown that we are confronted with many indications of physiological individuality regarding these phenomena. © 1998 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Muscular properties can be classified into two categories: intrinsic properties and contextual properties. The most general statement of function of muscle is that it has to contract. In more physical terms this means that the muscle has to deliver work to its outside world by exerting force while changing length. Work delivered will be determined by the properties that are related to the muscle material and architectural properties. Contextual properties, which are determined not by specific properties of the muscle itself but by the way it is built, are very important because they determine the specific function of a particular muscle. For instance, a particular muscle is a flexor of a joint because of a positive moment arm with respect to the flexion–extension axis of that joint. Another example of a very important contextual property of muscle is the number of joints it spans. By being biarticular a muscle may play the very important role of transporting power or energy from joint to joint (e.g. [9]), which plays a major role by (inter)muscular coordination (of movement) [41,42,74].

Intrinsic properties of muscle determine the functional capabilities of muscle to deliver external work. These properties are usually defined exclusively at the molecular, sub-sarcomeric or sarcomere level of intracellular
mechanisms of the muscle fiber. However, if understanding of movement of humans or other animals is the aim it is essential that intrinsic properties should also describe contributions at the level of the muscle and muscle tendon complex.

The aim of the present work is to review a selection of intrinsic muscular properties at these different levels of organization and discuss consequences for our insight into muscle functioning as well as for modelling of muscles and movement.

First, a classical way of looking at intrinsic muscular properties will be discussed and confronted with experimental results to assess its advantages and limitations. Subsequently, an alternative approach will be presented.

2. Intrinsic muscular properties: a classical approach

In addition to experimentation, modelling of muscle allows manipulation of parameters to assess their importance for muscular properties. It also contributes to forming simplified images of reality that is so important for human understanding. Much scientific effort has been devoted towards modelling of skeletal muscle and of movement. This effort started in the seventeenth century and is continued in the present at an enormous volume due to the development of easily accessible computational facilities.

2.1. Geometric models

In seventeenth century myology a controversy raged [43] between proponents of the idea that muscle could contract because of an increase in volume that flowed from the brain to the muscles [10,50] and those that were convinced that the volume of muscle is constant during contraction. Niels Stensen [62] made geometric models of muscle, which indicated that the fiber angle of pennate muscle has to change in order to accommodate increased fiber cross-sectional area. He was aware of the constancy of muscle volume because he came into contact with Jan Swammerdam who had performed experiments showing that fact (Fig. 1). This happened during his travels throughout Western Europe as Stensen visited Leiden University. Swammerdam’s results were not published until 1737 [68]. Due to strong opposition from the scientific community, the insights of Stensen and Swammerdam disappeared from the current body of knowledge of myology until the twentieth century. This paragraph may serve as a warning to all of us that particularly in myology there is a tendency for this to happen regularly (e.g. [40]), with sometimes disastrous consequences [30]. Predominantly in the second half of this century the basic ideas on muscle geometry have been rediscovered (e.g. [5,18,35,36,55,60,61,81–83]). Fig. 2 shows the basics of these principles. These angular changes are functionally important, particularly in very pennate muscle because they contribute to muscular length change at lower muscle lengths. This occurs at the expense of delivering force at these lengths [55], as is apparent from Eq. (1):

\[ F_{ma} = F_{fa} \frac{\cos(\alpha + \beta)}{\cos(\beta)} \]  

where \( F_{ma} \) and \( F_{fa} \) indicate muscle and total fiber force respectively. The angle \( \alpha \) represents the fiber angle and \( \beta \) the aponeurosis angle with respect to the muscle’s line of pull respectively.

It should be realized that these geometric models are not mechanical models but simply link fiber length to force exerted by the fibers by the definition of fiber length force characteristics. In other words mechanical
2.3. Combination of geometric and elastic effects

Recently it has been shown that elastic properties of aponeuroses (to which muscle fibers attach) can be incorporated in geometric models according to Eq. (2) derived by Zuurbier and Huijing [84]:

$$\Delta L_{ma} = \Delta L_{fa} \frac{\cos(\beta)}{\cos(\alpha + \beta)} + \Delta L_{aa} \frac{\cos(\alpha)}{\cos(\alpha + \beta)}$$

where $\Delta L$ indicates instantaneous length change, $\alpha$ and $\beta$ fiber angle and aponeurosis angle, respectively, and $L_{ma}$, $L_{fa}$ and $L_{aa}$ represent muscle, fiber and aponeurosis length of the active muscle, respectively.

2.4. Other effects of muscle geometry

Muscle geometry also affects adaptation of pennate muscle to altered conditions. These altered conditions may be growth, immobilization, etc. These conditions may cause the muscle to hypertrophy or atrophy (i.e. change its physiological cross-sectional area) or adapt the number of sarcomeres in series.

It is a well-known experimental result, particularly for lower leg muscles exposed to immobilization and growth, that muscle will adapt its optimum length to the length that is imposed [27,70,80]. Fig. 3 illustrates this concept for rat medial gastrocnemius muscle. The usual explanation for this phenomenon is that the number of sarcomeres is controlled in such a way that optimal overlap of myofilaments is made to coincide with the

![Diagram](Image)

Fig. 2. Slanted cylinder model of increase in fiber angle as a consequence of shortening. Due to the constancy of volume ($V_f = A_f l_f = A_{a-f} l_{a-f} \sin \gamma$) it can be shown that the fiber angle with the aponeurosis increases such that the increased fiber cross-sectional area $A_f$ can be accommodated on an unchanged area of attachment on the aponeurosis ($A_{a-f}$). If the aponeurosis has elastic properties it may shorten as force exerted on it is decreased ($\Delta l_{a-f}$). In order to still accommodate $A_f$, the fiber angle will have to increase even more. These processes are governed by the following equation:

$$\sin \gamma(\text{short}) / \sin \gamma(\text{long}) = \left( \sqrt{l_f(\text{long})} / \sqrt{l_f(\text{short})} \right) \left( l_{a-f}(\text{long}) / l_{a-f}(\text{short}) \right)$$

where $l_f$ represents fiber length and $l_{a-f}$ the length of the attachment area on the aponeurosis. If the fiber is allowed to bulge, a much larger fiber cross-sectional area can be accommodated.

interactions between fibers themselves and other constituent parts of muscle are neglected.

2.2. Phenomenological Hill models

Another important example of the effects of image forming on understanding of muscle is provided by the mechanical models of A. V. Hill [29]. These models pool all elastic properties of muscle, regardless of their anatomical location or level of muscular organization, in either series elastic or parallel elastic elements. All contractile properties not involving elasticity are concentrated in a contractile element, to which length–force and force–velocity characteristics are assigned. The model has been very successful by allowing a very much enhanced understanding of elastic contributions to the delivery of work by the muscle.

Geometric and Hill-type models have in common that muscle is modelled as being homogeneous with respect to fiber properties and/or muscle geometry (e.g. [5,7,9,18,22,23,35,36,45,46,55,60,79,81–83]). Therefore, they are to be considered as so-called lumped fiber models. Hill-type models are the most applied type of model but often the geometric effects described above are neglected.
imposed length of the muscle [70,80]. This idea was incorporated in a theory of control regarding number of sarcomeres in series [28]. Based on geometric models, an alternative idea was tested. The idea is that in pennate muscle changes of optimum length could also be brought about by length changes of the muscle as a consequence of contributions of increased or decreased fiber cross-sectional area [26]. In such a case there would be no necessity of changing number of sarcomeres in series within fibers. Experimental results show that at least for immobilization at short length [27] such ideas are tenable. It was estimated that fibers of very pennate medial gastrocnemius muscle were immobilized at short lengths comparable to those of less pennate soleus muscle. Despite that situation, the number of sarcomeres in series was not altered substantially in gastrocnemius but decreased very significantly (by more than 20%, Fig. 4a) in soleus [28]. In addition, during growth from young to young adult age, the number of sarcomeres in series within fibers increased by approximately 10% in soleus muscle. In contrast, no changes at all were found for the number of sarcomeres in series in gastrocnemius muscle [28]. These effects were attributed to the fact that, in less pennate muscle, adaptation of the number of sarcomeres in series is the only major mechanism for adaptation of muscle optimum length. In contrast, in very pennate muscle adaptation of optimum length has already occurred at the extracellular (organ) level: changing fiber diameter will have an effect on muscle length (Fig. 4b). In very pennate muscle, hypertrophy per se will increase muscle optimum length and atrophy will decrease it. As a consequence of atrophy-related changes in muscle optimum length, the following changes take place: during short length immobilization of pennate muscle sarcomeres, which initially were at low lengths, are returned towards their length of optimum overlap at the new muscle length, and the signal to change the number of sarcomeres is turned off. The fact that for gastrocnemius muscle (GM) the length range between optimum and active slack lengths was unchanged [27] (see also Fig. 3) is in accordance with this view.

2.4.1. Conclusions on geometric and phenomenological models

It is concluded that muscle geometry is functionally of very high importance, both for acute muscular properties and adaptation of these properties to altered conditions.

Clearly, simple models may contribute substantially to understanding of aspects of muscle function. It should be pointed out, however, that major limitations of these models also affect their application for other than heuristic purposes. One of these limitations is the assumption of homogeneity already indicated. Most definitely, the most limiting factor is not considering the degree of activation or designing models to describe effects in maximally activated muscle only. The wide use of such models is enhanced by the fact that most experiments describing physiological properties of whole muscle are performed under conditions of supramaximal stimulation of the peripheral nerve. As during in vivo activity muscle is not likely to be maximally active very often, modellers must deal with this problem in some way. Below we will consider the way this is usually done and contrast its effects with experimental results.

2.5. Modelling submaximal activation by scaling of muscular properties

A very practical way of dealing with situations of submaximally activated muscle is to assume that muscular
length–velocity–force properties are identical to those of maximally activated muscle apart from the fact that the force has to be scaled down. This means that properties normalized for force are identical for maximally and submaximally activated muscle. Usually, linear scaling is applied (Fig. 5). This approach is well known even though it is not always described explicitly when applied. A less well known but more confronting way of describing this approach is described below. As the physiological mechanism of submaximal activation of a fully recruited muscle is manipulation of firing frequency of muscle fibers, linear scaling of force can be performed by assuming that a linear force–firing frequency curve is applicable. In contrast, experimental force–stimulation frequency curves have a more sigmoidal shape. To fully describe the practice of linear scaling of force one should realize that a second and maybe more important assumption is made implicitly to obtain identical length–velocity–normalized force characteristics. The force–frequency characteristics have to be independent of muscle length. Fig. 6 shows a three-dimensional representation of normalized force as a function of firing frequency and length, as applied for rat GM on the basis of properties of maximally activated muscle. In the frequency–force planes linear relations are clearly visible. In the length–force planes clearly length range of active force generation is independent of degree of activation.

In skinned fiber the sarcolemma of muscle fibers is

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Fig. 5. Three-dimensional representation of the effect of linear scaling of force on length–velocity–force curves. The top left panel shows assumed properties under conditions of maximal activation. The bottom left panel shows a 50% scaled version of these characteristics. Note that after normalizing for optimum force, one curve (left panel) describes both 100 and 50% activation conditions. This indicates that the material properties of the muscle were unaltered.

Fig. 6. Linear scaling of force, as often applied in muscle modelling, represented as a function of muscle tendon complex length and firing frequency. The surface represents conditions of both maximal and submaximal activation. Force is normalized for optimum force at 100 Hz and length is expressed relative to optimum length. For a given length the firing frequency–force curve can be discerned. Note the linear character of this relation. Also note that if normalized for maximal force at each muscle length one linear curve would be obtained describing frequency force characteristics at all lengths. For a given frequency, length–force curves can be discerned. Note that if force would be normalized for optimum force of each length–force curve, one curve would be obtained describing length normalized force curves for all frequencies. As a consequence, optimum (arrows) and active slack length are unchanged at different frequencies.
removed by chemical treatment or mechanical procedures. In such preparations it is possible to manipulate the intracellular conditions by superfusion, using solutions containing different concentrations of calcium ions. This allows the study of the effects of calcium per se on exerted muscle force. Results are usually published as pCa–force curves for one or at best a few fiber lengths. These curves are the intracellular equivalent for steady state force–firing frequency curves. Results show that pCa-normalized force curves are sigmoidal in shape. The details of the sigmoid curve are very dependent on muscle length (e.g. [64–66]). This feature is referred to as a length-dependent calcium sensitivity of myofilaments. Calcium sensitivity is defined here operationally in terms of normalized force. This means that for a given calcium concentration a higher force, normalized for its value at maximal activation at that length, is obtained than at a lower length. To keep this definition explicit and clear it may be wise to refer to this phenomenon as “calcium sensitivity of (normalized) force” until the mechanism is more clearly understood.

Only Stephenson and Wendt [65] presented such findings also in terms of (partial) sarcomere length–force curves of skinned fibers (Fig. 7). This figure shows that with decreasing activation optimum length shifts to higher fiber lengths, whereas maximal sarcomere length of active force generation is unchanged. Recent experiments [3] on intact muscle fibers in which calcium concentration was measured as well as force also showed such effects for partial fiber length–force curves near optimum length. This study also showed that altered calcium concentrations were not found at different lengths, so that the effects must be related to the actual mechanisms of contraction. It has been suggested that a length-dependent affinity of troponin C may be involved in this phenomenon [3,65].

In their classic study Rack and Westbury [57] reported similar results for whole cat soleus muscle. Shortly thereafter, Close [11] reported that length normalized force curves for twitches are different than the curve for tetani.

When activated at fairly low frequencies, force is not constant in time but significant force ripples are present. This is caused by synchrony of recruitment of motor units in such experiments. For simplicity, representation of length force results in this article will be made for peak forces only. Fig. 8 gives such a representation for the results of Rack and Westbury [57]. This may be one of the most neglected results in twentieth century myology and biomechanics. Therefore, we conclude that an alternative approach of describing properties of submaximally active muscle is needed badly, and modellers should be encouraged to use it. At present, this author knows only one study in which it is unequivocally evident that attempts were made to incorporate such effects [22].

2.5.1. Conclusions on the classical approach

Despite the limitations of the classical approach described above, clearly much has been learned from using it. In particular, this is the case for mechanisms active in muscle that determine both acute and long-term properties. At the same time we should realize that such a classical approach is too limited for use in generating knowledge about properties of muscles during daily use.

3. Alternative approach

3.1. Effects of firing frequency and its history

We repeated the experiment of Rack and Westbury [57] for rat GM [59,60] and small bundles of its fibers [86]. Fig. 9a shows the principle of the protocol used in those studies for constant stimulation frequencies and an
example of force exerted in time as obtained at one muscle length. Fig. 10 shows the results of such experiments represented as a three-dimensional representation of normalized force as a function of firing frequency and length. Note the following features:

(a) Sigmoidal shapes of the curves in the frequency–force planes. The shape of these curves is very dependent on muscle length.
(b) With lower constant frequencies, optimum length is encountered at muscle lengths progressively higher than in maximally activated muscle.
(c) With lower constant frequencies active slack length moves substantially and progressively to higher muscle length. This shift tends to be somewhat smaller than the shift of optimum length, therefore length range between optimum and active slack length increases at lower constant frequencies.

It should be pointed out that the length force characteristics may no longer be what their name indicates but may also contain some velocity effects. This will happen [32] if a phase delay of sufficient magnitude is found between varying muscle force and consequent length changes due to viscoelastic properties of tendon and tendon plate. This may be true for the experimental data shown here. If it is, it is also likely to be the case for in vivo conditions.

Hatze [22] attempted to incorporate effects of firing frequency in his model by altering the slope of the force–frequency curve as a function of muscle length. However, it should be realized that such an action will not be fully successful as it has the effect of taking into account shifts of optimum length but not of active slack length. In such models, overestimation of force at lower lengths is a direct consequence. Recently, van Zandwijk et al. [76] were fairly successful in modelling submaxi-
Fig. 10. Three-dimensional representation of effects of constant stimulation frequency (CSF) and muscle tendon complex length on force of rat medial gastrocnemius muscle. Force is normalized for optimum force at 100 Hz stimulation and length is expressed relative to optimum length as encountered at this stimulation frequency. In the frequency–force planes the change of shape of the stimulation frequency–force curves can be clearly seen. Note the progressive shift of optimum length to higher lengths at lower stimulation frequencies (white arrows). At the lowest frequencies, which are physiologically most important, active slack length also shifts to higher lengths.

Fig. 11. Three-dimensional representation of effects of decreasing stimulation frequency (DSF) and muscle tendon complex length on the force of rat medial gastrocnemius muscle. Force is normalized for optimum force at 100 Hz stimulation and length is expressed relative to optimum length as encountered at this stimulation frequency. In the frequency–force planes the change of shape of the stimulation frequency–force curves can be clearly seen. Note the progressive shift of optimum length to higher lengths at lower stimulation frequencies (white arrows), but this shift is smaller than during CSF stimulation. At only the very lowest frequencies, active slack length also shifts a very limited amount to higher lengths.

The maximal force exerted by fully recruited rat GM during constant frequency stimulation (CSF) on the basis of twitch characteristics. It should be noted, however, that success was dependent on the model being optimized for individual variables of twitches and used to predict force of GM from individuals.

A different type of stimulation frequency experiment was also performed [58,59,86] in which stimulation frequency was decreased in a stepwise fashion during each isometric contraction (for the decreasing stimulation frequency (DSF) protocol, see Fig. 9b). Normalized force as a function of actual firing frequency and length is shown in Fig. 11. Note the following features:

(a) Sigmoidal shapes of the curves in the force frequency plane differ substantially with respect to those of constant stimulation frequencies.
(b) Shifts of optimum lengths to higher lengths with lower stimulation frequencies still occur albeit to a smaller extent. Therefore, the history of stimulation frequency of this particular protocol has the net effect of shifting optimum length back to somewhat lower lengths.
(c) The shift of active slack length to higher lengths with lower frequencies is only minimally present. It seems as if a memory persists of muscle being previously stimulated at high frequencies and almost maintaining active slack length accordingly.
(d) These changes cause force to be potentiated substantially at many lengths (Fig. 5 in [59]). In that study the authors normalized force for its value during maximal stimulation at each length, thereby drawing attention to extremely high normalized levels of potentiation at low muscle lengths that may be very important functionally because it allows force exertion many times higher than during constant firing frequencies. However, when these results are plotted as absolute force or normalized for force at the original (i.e. 100 Hz) optimum length (as is done in Fig. 12), clearly at higher lengths substantial potentiation occurs.

Initially, potentiation was defined operationally, i.e. in terms of increased force. However, more recently it has been described in terms of effects of phosphorylation of the myosin light chain (see review in [69]). The myosin light chain is an integral part of the cross bridge and it extends from the tip of the head to the neck region closer to the thick filament. Donation of a phosphate group from ATP is intimately related to the process of contraction. Considerably different ideas about the molecular effects of phosphorylation exist (e.g. [56]), but not about its increasing effects on force. Force increases are reported [54] to be linearly related to the amount of phosphorylation of the myosin light chain.

A more detailed analysis of these interesting phenomena is beyond the scope of the present paper. A more practical question that remains to be answered is how do these results of stimulation frequency experiments compare to those predicted by linear scaling of force for this muscle. Fig. 12 shows errors of linear scaling of force with respect to CSF and DSF results. The error curves are obtained by subtracting the curve of Fig. 6 from those of Figs 10 and 11 respectively. The results of this operation speak for themselves: very substantial
Fig. 12. Three-dimensional representation of differences in rat GM isometric force as obtained from linear scaling of force, constant (CSF) or decreasing (DSF) stimulation frequencies. Differences in force are normalized for optimum force at 100 Hz stimulation and length is expressed relative to optimum length as encountered at this stimulation frequency. The two figures on the left show the errors that would be made if linear scaling of force rather than experimental data of protocols regarding CSF (top panel) or DSF (bottom panel) would be used. At low lengths and frequencies negative errors of linear scaling compared to CSF and DSF protocols were not plotted. The figure on the right shows the difference in force between the DSF and CSF experimental data. In all three figures very substantial differences are encountered at many lengths and frequencies.

errors (up to 50% at some lengths in the lower frequency ranges) result for both conditions.

As indicated above Fig. 12 also shows differences in force between the particular DSF and CSF protocols used. Preliminary results (Bosch, Roszek and Huijing, unpublished observations) indicate that these effects may be quite specific for the particular protocols. A major challenge is found here for experimental physiologists and biomechanists alike to attempt to describe the effects of history of activation in such a general way that it can be applied for modelling of muscle and movement.

3.1.1. Conclusions on firing frequency and history

Due to the interaction between level of activation as well as its short time history and length force characteristics, linear scaling of force is not an adequate way of dealing with the problem of modelling submaximal activation if the model has to incorporate features of real muscle on this point. There is some hope that taking into account adequate features of excitation dynamics may allow incorporation of CSF effects. However, individual properties seem to play an important role in these effects and have to be taken into account. The effects of potentiation will further complicate such attempts. Describing such complicated effects as a function of muscle length seems to be indicated.

3.2. Effects of sustained isometric contraction (at constant stimulation frequency)

Many people are aware that, during sustained contraction, fatigue (e.g. [1,15]) will play a major role in determining how much force the muscle exerts at any point in time. It is common practice to define such fatigue operationally as the decrease in force seen in time during a contraction. Recently, Huijing and Baan [33] reported effects of fatigue on isometric length force characteristics during tetanic contraction, at almost maximal activation (100 Hz). Because of the finding of small shifts of optimum length but not of active slack length, we hypothesized these effects to be related to intracellular inhomogeneities of calcium concentration [31,32] and thus inhomogeneities of activation [1]. It should be noted that alternative hypotheses may have to
be considered as well. In any case these results indicate that, during sustained maximal contraction, isometric muscle length force characteristics are changing as a function of time. Few people realize that very complicated interactions between fatigue [1] and potentiation [69] are observed during sustained submaximal isometric contraction. It is argued [34] that the mechanisms of fatigue may even lead to an increase of force at higher muscle lengths (Fig. 13). Crucial to these arguments is that the maximal length of force exertion is considered a fixed property of muscle regardless of its degree of activation and potentiation. In the work on skinned fibers (see also Fig. 7) experimental evidence for this assumption is found, but the point requires further attention also for intact fibers and whole muscle. In any case, the net changes in force during an isometric contraction, even under conditions of constant firing rate, cannot be adequately assigned to be the consequence of fatigue [33,34]. Following similar arguments it is feasible that the effects of myosin light chain (MLC) phosphorylation (Fig. 13) may lead to a decrease of force [34] at certain lengths. Therefore, the use of operational definitions for these phenomena is likely to interfere with enhanced understanding rather than to facilitate it.

It should be noted that in vivo the central nervous system responds to short term sustained submaximal isometric contractions by lowering firing rates [6]. In such a case effects of potentiation, such as described for decreasing stimulation frequency protocols, should also be taken into account.

3.2.1. Conclusions on sustained contraction

From the results described in this section it becomes clear that what may seem to be a simple isometric contraction is already a very complex phenomenon during which length force properties alter in time by effects of fatigue and potentiation of force. We should consider it likely that dynamic muscular properties will also change with time. It should be noted, however, that in these experiments the relatively long duration of contraction was used compared to in vivo intermittent contractions that are encountered in cyclic movements. Therefore, one has to be careful with extrapolating the magnitude of these effects to the in vivo situation. Instead, this work

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**Fig. 13.** Schematic representation of alterations of isometric length-force curves as a consequence of two different mechanisms active during conditions of local muscle fatigue, potentiation by myosin light chain phosphorylation and distribution of sarcomere length in series within fibers. The alterations indicated are deduced from experimental data[32,57] on rat GM during sustained contraction. The magnitude of effects is very length dependent and may change to a direction that is in contradiction with operational definitions commonly used for fatigue (decrease of force in time) and potentiation (increase of force in time). Even during a sustained isometric contraction at constant firing frequency (CSF) the actual length–force properties would change dynamically in time and would at any time be determined by the net effect of the mechanisms illustrated here and most likely others as well.
should be used as an indication of mechanisms possibly active during in vivo movement.

3.3. Inhomogeneous fiber properties within a muscle

The problem of assumed homogeneity of fiber properties within a muscle is indicated above as one of the limiting factors playing a role in muscle modelling. It should be realized that this factor may also play a role in the interpretation of experimental results. For human muscles, joint angle moment characteristics were hard to explain on the basis of fiber lengths or number of sarcomeres in series within fibers (e.g. [25,37,39]). In addition, we know surprisingly little about which part of the length range of the length force curve is actually used by humans in vivo. Recently, Lieber, Friden and co-workers extended their previous experimental animal work dealing with the relationship of joint angle and sarcomere length [45,46,49] to architecture studies of human muscles [47] as well as invasive experiments on humans [48] (see also this issue).

The importance of knowledge about the joint angle–sarcomere length is evident. Many patients are hampered severely by limitations of joint excursion, which may be caused by consequences of faulty regulation of optimum length and/or length range of active force exertion. Therefore, it is very important to be aware of factors that should be taken into account in interpreting joint angle–sarcomere length data. Only fairly recently, some modellers interested in morphological details of active muscle have tried to study aspects of functional consequences of inhomogeneities in muscle (e.g. [8,16,24,76]) or apply finite element models to skeletal muscle [71–73,75]. Recent results presented by van der Linden et al. [21,72,73] will be discussed here. Fig. 14 shows an important feature of the model: initially, the muscle is modelled in the passive state with homogeneous properties just as in the geometric modelling. In similarity with geometric models, muscle fibers are represented by straight lines, but only for the initial passive state. As the muscle is activated a distribution of fiber length develops. The assumed homogeneity of the model muscle means that the muscle fibers contain the same number of sarcomeres in series. Therefore, fiber mean sarcomere length becomes distributed as well: different fibers within the active muscle have different mean sarcomere lengths. Such a distribution has been referred to as a secondary distribution of fiber mean sarcomere length [31,32]. Contraction also introduces curved fibers and aponeuroses. For the fibers the amount of curvature is dependent on the location within the muscle. We must conclude that mechanical interaction between the elements of the model makes the continued presence of homogeneous characteristics within the muscle impossible. Despite this added feature it was concluded [72,73] that muscle length force characteristics were not adequately described by the model: substantial differences for length force data remained. Clearly, factors of major importance are not included in the models used.

3.4. Primary inhomogeneities of fiber mean sarcomere length

For rat muscle circumstantial evidence regarding the existence of a distribution of fiber mean sarcomere length is available (e.g. [16,20,27,39,79,85,86]). The majority of this indirect evidence is based on the following finding. At muscle optimum length the mean sarcomere length of certain (groups) of fibers is not equal to sarcomere optimum length as expected from a simple homogeneous muscle. For example, GM active distal fibers reach optimum sarcomere length at muscle lengths 1.27 mm over optimum length [38]. This amounts to more than 10% of the length range between optimum and active slack lengths. For GM inhomogeneous with respect to mean sarcomere length of different fibers, models have been made to assess effects on length force characteristics [16]. For EDL muscle [8] a first effect was modelled in 1990.

Particularly for cat muscle, direct evidence has been accumulating in the last two decades that, at any muscle length, sarcomere length may be distributed [44,63]). This evidence is obtained from studying length force characteristics of whole muscle and single motor units stimulated through their isolated ventral roots. In some cat muscles (flexor digitorum longus muscle) the distribution is systematic for motor unit size (Fig. 15). However, for another cat muscle this could not be shown [44]. Note that if the distribution is regulated by motor unit size, muscle length force characteristics will be very much dependent on the degree of recruitment of motor units. More recently, direct evidence for distribution of length force characteristics of (groups) of motor units was also encountered for rat GM [12,13]: for that muscle bigger motor units tended to have their optimum length at higher muscle lengths than whole muscle optimum length. This means that to obtain total fiber force for a muscle its fiber length force curves do not add “in phase” with respect to fiber or sarcomere length. Instead, the addition should take place with the curves shifted a certain amount along the length axis with respect to each other (see also [32,34]). In such a muscle at muscle optimum length the grand mean of the sarcomere lengths should be equal to sarcomere optimum length. In a pen- nate muscle the situation is complicated by fiber length dependent angular effects described above.

3.5. Individual variation

In an experiment the functionally parallel-fibered semi-membranosus lateralis muscle (SMI) of the rat was examined [79]. Individual SMI muscles may show con-
Fig. 14. Changes of muscle geometry as modelled by a Finite Element Model of rat medial gastrocnemius muscle (GM). The top panel shows linearized geometry of the muscle as assumed to occur at optimum length in the passive state. The values for fiber as well as aponeurosis lengths and angles were experimentally determined for the most distal fiber and proximal aponeurosis of GM. The muscle is assumed to be fully homogeneous and muscle volume is constant. As the model muscle is activated a distribution of fiber lengths develops due to interactions between fibers as well as between fibers and aponeuroses. As this distribution was not present initially but developed as the muscle was activated, it is referred to as "secondary".

3.5.1. Conclusions on inhomogeneous properties

Inhomogeneous properties, particularly those related to primary and secondary distributions of fiber mean sarcomere length, have a sizable effect on length force characteristics of maximally active muscle but more importantly on not fully recruited muscle. Some evidence is available that such properties may be influenced by exposing the muscle to different conditions. In any case it is clear that a physiological individuality causes considerable variation between individuals in these phenomena. This will be very difficult to deal with in modelling unless model variables can be based on individual data.

3.6. Effects of previous shortening

It has been known for quite some time (e.g. [14]) that previous shortening leads to what is called a deficit in isometric force (Fig. 17). This means that, after shortening, the redeveloped isometric force does not attain values that are as high as that of a comparable isometric contraction at the target length. These effects may, for the major part, be ascribed to inhomogeneities of lengths of sarcomeres that are in series within one fiber [14]. However, some differences in force were also encountered in conditions in which no changes of such distribution could be shown [67]. On the basis of that finding
Fig. 15. Example of a distribution of motor unit optimum length with respect to whole muscle optimum length as found in cat flexor digitorum longus muscle[2]. Isolated motor units were stimulated supramaximally through their axons made accessible at the ventral roots of segments of the spinal cord. Length–force curves thus obtained were compared to those obtained under supramaximal stimulation of the whole peripheral nerve. Motor unit optimum lengths are distributed and in this case the distribution is systematic with respect to motor unit fiber type and thus size.

Fig. 16. Length–force characteristics of individual lateral semi-membranosus muscle of six rats. Force is normalized for optimum force of each individual muscle and length expressed relative to its optimum length. Substantial differences are found regarding length range between optimum and active slack lengths. These differences did not correlate with the number of sarcomeres in series within fibers. However, they did correlate highly and significantly with the amount of distribution of fiber mean sarcomere length \( r = 0.88 \). The muscles with the smallest length range showed no evidence of such a distribution, whereas those with the highest length range also showed evidence of the highest amount of distribution. It is evident that, regarding primary distribution of fiber mean sarcomere length, individual variation may have a sizable influence on length–force characteristics.

Fig. 17. Effects of previous shortening on length–force characteristics of rat medial gastrocnemius muscle (GM). The top panel shows length changes imposed on an individual GM muscle tendon complex as a function of time. Superimposed are length time trajectories of contractions that start at different lengths but end after isokinetic shortening at the identical target length. Note that shortening speed is identical regardless of initial length but range of shortening is varied (from 1 to 4 mm). One contraction, which remained isometric throughout activation, is used a reference (isom 0–0). The middle panel shows an example of force exerted as a function of time. After redevelopment of isometric force, a “force deficit”, relative to the fully isometric contraction, is seen in contractions that actually involved a concentric phase. The magnitude of the force deficit is also dependent on the amount of shortening that was imposed. Such experiments were performed for many target lengths. The bottom panel shows mean length–force curves for six muscles. Active muscle force and muscle length are plotted. Individual curves represent these characteristics for GM with equal previous shortening history. Clearly, force deficit is encountered at all target lengths but its magnitude is very much dependent on muscle length. Small differences in optimum length are found.
Classically, muscle force–velocity characteristics are determined in isotonic or isokinetic contractions. Generally, force–velocity curves based on isotonic contractions are considered more advantageous, as theoretically they do not contain effects of series elastic elements. However, the above indicates that force–velocity characteristics both for isotonic and concentric contractions are contaminated by length-history and or velocity-history effects in a way that seldom will reflect in vivo characteristics. This effect may very well be larger for isotonic contractions. Experimentally, such contractions have the added disadvantage that the history of shortening is not under the control of the experimenter, as it is determined by interaction of muscular length–force properties and imposed load.

The recent model reported by Barrata et al. [4,78] is a new effort to take such effects into account. However, this model is also based on experimental results involving isotonic contractions and is therefore likely to suffer from the lack of control of the determining factor: shortening history.

3.6.1. Conclusions on effects of previous shortening and its history

Previous shortening affects length force characteristics substantially. Its short-term history is also an important factor. The concepts of length–force and force–velocity characteristics need to be reconsidered in view of what is needed to understand performance in daily movement.
4. Summarized overall conclusions

The major conclusions are:

- Length–velocity–force characteristics are not unique properties of a muscle.
- These characteristics are not only strongly influenced by actual effects of recruitment, firing frequency, amount of shortening performed and velocity of shortening but also by the short time history of these factors.
- Therefore, length, velocity and activation cannot be considered as independent determinants of muscle functioning.
- For experimenters it is crucial to study muscular properties not just at one length but effects should be considered over the whole length range relevant for actual function.
- We are confronted with many indications of physiological individuality regarding these phenomena.

The effects for modelling are summarized below:

- Depending on their purpose, modellers should still select relatively simple models of muscle.
- However, chosen limitations should be made explicitly.
- Comparisons with experimental results are crucial for validation, but more so for generation of new ideas.
- As a consequence we should be focusing much more on differences rather than agreements between model and experimental results.

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