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Musculotendon forces derived by different muscle models

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The accuracy, feasibility and sensitivity of several different methods for calculating muscle forces during functional activities in humans were investigated. The upper extremity dynamic system was chosen, where the flexion–extension of elbow joint was studied. To counteract the redundant mechanisms we adopted optimization criteria with and without models of individual muscles according to their active and passive properties. Comparisons with known movements solved by inverse dynamics approach and optimization techniques provided similar results for all optimization criteria. Moreover, if muscle models with active and passive properties are included in these analyses, it is relatively easy to calculate muscle forces of both agonists and antagonists. These approaches may be used to provide input data for dynamic FEM stress analysis of bones and bone–implant systems.

Key words: inverse dynamics, muscle models, musculotendon forces

1. Introduction

Muscle forces are considered to be an important subject of considerations for orthopaedists, biomechanists, and physical therapists because joint contact forces, as well as muscle forces, must be estimated in order to understand joint and bone loading and pathology. The calculation of muscle forces generated during complex activities is not trivial. In this study, the use of several muscle models, with and without active force–length, force–velocity and passive force length properties, as well as inverse and forward dynamics approaches were studied. The technique of dynamic optimization allowed us to study the tendon and activation dynamics and to examine the redundant elbow joint problem as the control problem.

2. Methods

For this study the elbow joint musculoskeletal system was chosen. It consists of the following seven joint actuators: four flexors (biceps brachii long head (BIClh), biceps brachii short head (BICsh), brachialis (BRA) and brachioradialis (BRD)), and three extensors (triceps brachii long, medial and lateral heads (TRIlh, TRImh, and TRIlt)). Other elbow actuators were neglected. The elbow joint was selected because it gave a good visual demonstration and for the sake of simplification it is possible to say that the elbow motion is uniplanar and uniarticular. The elbow flexion–extension movement was executed without any motion in shoulder, hence all the elbow actuators were modelled as single joint actuators. This redundant musculoskeletal system was modelled with one degree of freedom at autonomus variable elbow flexion–extension angle.

2.1. Musculotendon models

Muscles are the actuators of the neuromusculoskeletal system that generates movement. The control of the complex musculoskeletal system is based on the

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understanding of physical principles of musculotendon actuator action. The muscle model used in this investigation is referred to as the Hill-type model. In figure 1, the musculotendon complex is presented as idealized mechanical objects [1], [2]. In contrast to the models used, for example, in [3] and [4], here the series elastic element (SEE) was neglected, because the energy stored in cross-bridges is expected to be very low compared with the total energy stored in the external and internal parts of tendon. The muscle is assumed to consist of two components: an active force generator and parallel passive component. The model for the active contractile component is based on the generally accepted notion that the active muscle force is the product of three factors: (1) a length–tension relation, (2) a velocity–tension relation and (3) the activation level. The passive component includes a parallel elastic element and passive muscle viscosity.

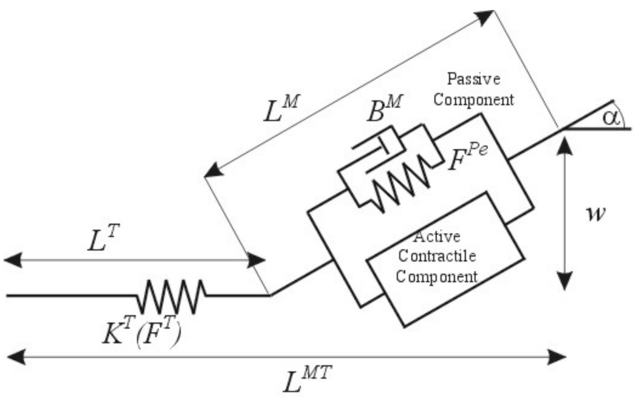

Fig. 1. The Hill-type model of musculotendon complex

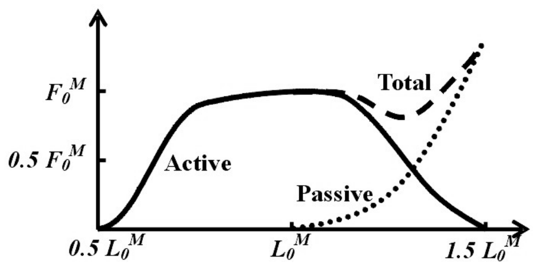

Fig. 2. The passive and active force–length relations

A theoretical explanation for the active force–length relation is based on the microscopic image of muscle and is elucidated by the sliding filament theory [5]–[7]. This theory offers an explanation for a generally accepted notion that when a muscle is completely tetanized, the active force displays a "parabolic" dependence on fiber length in a nominal region, $L^M \in \langle 0.5L_0^M ; 1.5L_0^M \rangle$, with maximum force when $L^M = L_0^M$. This nominal region is an ideal case and in practice can be different for every single muscle. The passive force–length relation displays the "exponential" dependence when muscle is lengthened more than optimum muscle length (equation (1), figure 2).

The active force–length relation for muscles has been constructed as parabolic function (equation (2)) that fits the data reported by [8], [6]. This curve is then scaled to provide a description for specific muscle:

$$F_0^M f_L^{pe} = F_0^M (L^M / L_0^M)^3 \exp(8(L^M / L_0^M) - 12.9)\,, \quad (1)$$

$$F_0^M f_L^{\mathrm{act}} = F_0^M (1 - ((L^M / L_0^M - 1)/0.5)^2)\,. \quad (2)$$

For the force–velocity relation under concentric and isometric conditions, the hyperbolic relation (the Hill equation) was used:

$$F^M = F_0^M f_v = F_0^M \frac{v_0^M - v^M}{v_0^M + c v^M}\,. \quad (3)$$

For the force–velocity properties under eccentric conditions the modified Hill equation (4) proposed by MASHIMA et al. [9] was applied:

$$F^M = F_0^M f_v = F_0^M \frac{2v_0^M - b' + v^M \dfrac{a'}{F_0^M}}{v_0^M - b'}\,. \quad (4)$$

The muscle force–velocity relation under eccentric, isometric and concentric conditions when muscle is fully activated is shown in figure 3.

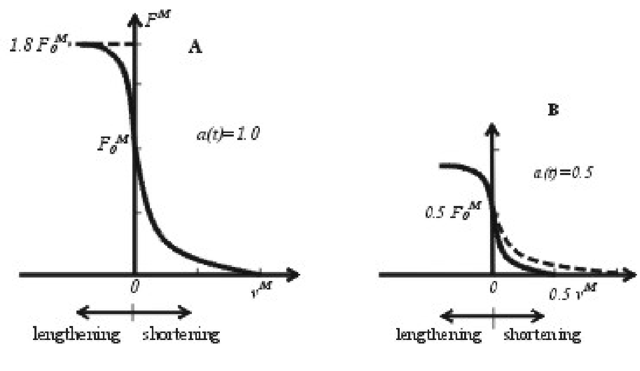

Fig. 3. The force–velocity relation when muscle is fully activated (A) and when the activation level is $a(t) = 0.5$ (B)

Both active muscle properties (force–velocity and force–length properties) must be scaled by activation level. It is assumed that the passive muscle velocity and muscle viscosity effect are small, then a total muscle force is the sum of the passive and active forces

$$F^M = F_0^M (f_L^{\mathrm{act}} f_v a(t) + f_L^{\mathrm{pa}}) \cos(\alpha)\,. \quad (5)$$

When the tendon dynamics and both passive and active properties are included, the muscle model is expressed by the first-order differential equation

$$\dot{F}^T = K^T(F^T)\,(\dot{L}^{MT} - \dot{L}^M / \cos(\alpha)), \tag{6}$$

where K^T is the tendon stiffness [4]. In order to describe the dependence of muscle activation level $a(t)$ on excitation level, the activation dynamics was used in the form of the first-order differential equation

$$\dot{a} = (u^2 - ua)/\tau_{\text{rise}} + (u - a)/\tau_{\text{fall}}, \tag{7}$$

where $u(t)$ is the excitation level of muscle at the time t, and τ_{rise} and τ_{fall} are the rise and decay constants of muscle activation [10].

Some authors use the EMG-driven models, where the experimentally collected, processed and normalized EMG signal is used as an activation signal. The muscle models are sometimes used in a much simpler form, without dynamical properties and force–length and force–velocity properties, only the driving signal be included (equations (8) and (9)). The differences between these models and the computational approaches will be discussed below.

2.2. Experimental data collection and inverse dynamics

Elbow flexion and extension movements were recorded with the 6-camera 60Hz VICON Motion Analysis system for two movement speeds (slow, 1.1 rad/sec; fast, 2.8 rad/sec) and two loading conditions (unloading and loading with a 4.2 kg bar-bell). Simultaneously the electromyographic activity (EMG) of the elbow joint actuators (BRD, BIClh, TRIlh, and TRIlt) was recorded using bipolar surface electrodes. Unprocessed EMG data during maximum voluntary isometric contraction (MVC) were also collected from the same muscle. EMG data for MVC were collected when the muscles fibres subjected to measurements had assumed the optimum length L_0^M.

Raw electromyograms during flexion–extension movement activities and maximum voluntary isometric contraction were bandpass-filtered (20–500 Hz), offset, rectified and smoothed (using a RMS window of 75 msec). Processed EMG signals were normalized, i.e., divided by the processed MVC values (for each muscle separately). The same EMG signals as these recorded for BIClh were associated with the BRA and BICsh actuators and the averages for TRIlh and TRIlt signals were associated with TRImh, because the muscle were very close and it is possible to assume their similar function in elbow flexion–extension. The elbow joint net moment M_{net}, which represents the sum of moments from all joint actuators, both flexors and extensors, was solved using inverse dynamics.

Inertia properties were calculated using an algorithm based on three inputs, i.e., human weight, height and gender, for details see [11]. The positions of anatomically significant limit points (e.g., muscle attachments) were taken from the Mayo’s study of anthropometric data [12]. These data were then scaled using the length of brachium and antebrachium from the measured subject. Additionally, the positions of attachments, muscle volume, mass and physiological crossectional area (PCSA) were scaled using the circumference of brachium and antebrachium.

2.3. Raw EMG-driven models and static optimization

One of the possibilities of estimating the force in a single muscle is the use of EMG as a driving signal. Here, the suitable muscle models are based on the Hill-type muscle model (figure 1). The estimated force does not depend on (imperfect) joint torque calculation via inverse dynamics and it is computationally simple enough to be potentially applied in a real time. The disadvantages of these types of models lie in the assumptions associated with the input data such as an EMG signals and the muscle parameters of the model. The equation

$$F^M = F_0^M a(t) \tag{8}$$

represents the non-physiological EMG-driven muscle model [13], based only on the maximum isometric muscle force $F_0^M = PCSA \cdot \sigma$, where $\sigma = 31.8$ Ncm^{-2} is a specific muscle tension [14] and the activation signal $a(t)$ is a recorded, processed and normalized EMG. Next equation

$$F^M = F_0^M f_L^{\text{act}} a(t) \tag{9}$$

represents the muscle model more physiological than (8), where f_L^{act} is an active force–length factor [15]. Equation (5) is the basis for a physiological EMG-driven model [16] and considers the factors in terms of force–velocity f_v, force–length f_L^{act} and activation level $a(t)$ of contractile muscle component, force–length relation f_L^{pe} of passive muscle component and

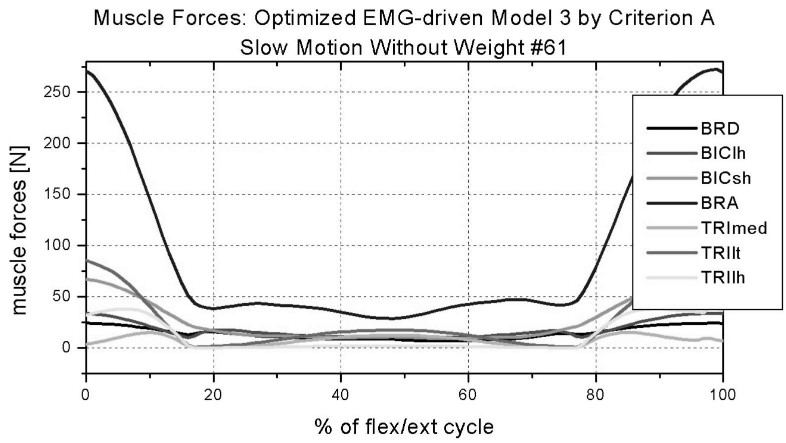

Fig. 4. The estimated elbow actuators forces during flexion–extension cycle by muscle model equation (5) and optimization criterion equation (10)

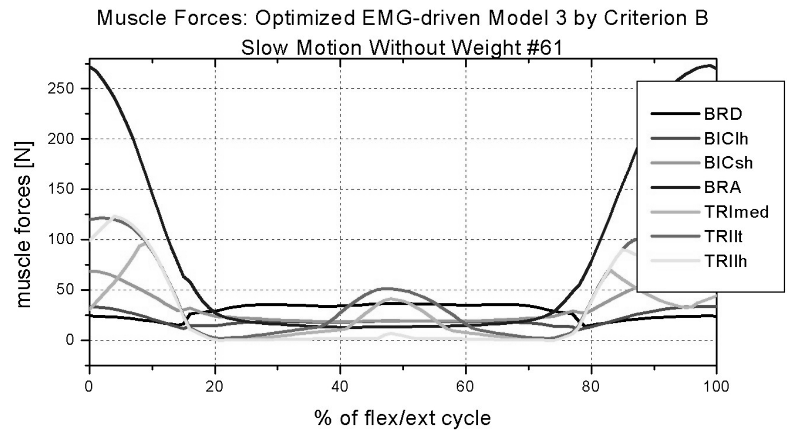

Fig. 5. The estimated elbow actuators forces during flexion–extension cycle by muscle model equation (5) and optimization criterion equation (11)

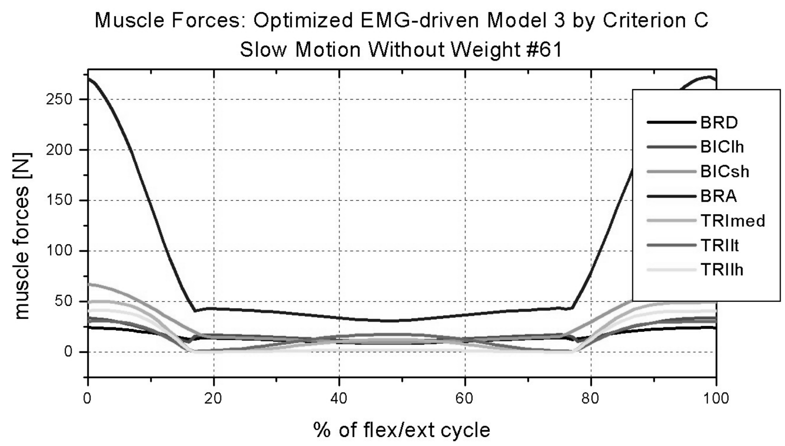

Fig. 6. The estimated elbow actuators forces during flexion–extension cycle by muscle model equation (5) and optimization criterion equation (12)

penalty angle α. This model corresponds to a full Hill-type muscle model without tendon dynamics and muscle viscous properties.

All inputs to these three models ((5), (8), (9)) are obtained under the same experimental conditions. Usually it is necessary to apply any optimization technique to the “optimal” processing of the EMG’s because the net joint moments calculated based on estimated muscle forces by means of the raw EMG-driven models with normalized EMG’s as driving signals and the net joint moments calculated using inverse dynamic technique are different. If the optimization of the driving input parameter EMG is used, the results obtained can be better. Activation signal should be optimized because during its recording both processing and interpretation may include many errors. Therefore, all three muscle models were applied to the static optimization technique. The influence of three different optimizing criteria was studied: 1. Minimization of activation squared [17], equation (10). 2. Minimization of muscle stress cubed [18], equation (11). 3. Minimization of maximum activation squared, equation (12):

$$J = \sum_{i=1}^{n} a_i^2 \,, \tag{10}$$

$$J = \sum_{i=1}^{n} \left(\frac{F_i^M}{PCSA_i} \right)^3 , \tag{11}$$

$$J = \max \left(\sum_{i=1}^{n} (a_i)^2 \right). \tag{12}$$

In all three cases, the optimization problem was to minimize the objective function J = min over $i = 1 - n$ actuators, $n = 7$, with the equality constraints

$$M_{\text{net}} - \sum_{i=1}^{n} F_i^M \times r_i = 0 \,, \tag{13}$$

where r_i is the moment arm of the i-th muscle to the elbow joint, M_{net} is the elbow net moment. The additional inequality constraint is that all of the musculotendon forces must be positive ($F_i^M \geq 0$) because muscles cannot produce compressive forces.

3. Results

Each EMG-driven model used without optimization technique gives the values of the net joint moment that differ from these obtained based on inverse dynamics approach. Optimization technique was used to process and set up the weight of activation level $a(t)$ for each muscle. If the model is simple ((8) and (9)), the values of the estimated forces are zero for elbow extensors (co-contractors) and they do not correspond to the measured EMG signals.

A “physiological” muscle model (5) includes also a passive force–length factor and due to this passive property it always gives co-contractors forces with non-zero value (see, for example, figures 4, 5, 6 which show the results calculated by means of static optimization approach constrained by the net joint moment from the specific movement condition – the slow motion without weight). This statement is valid for each of three optimization criteria used (equations (10), (11), (12)), and do not agree with other muscle models in which all passive and active muscle properties are not included.

4. Discussion

The main goal of this study was to deal with the possibilities of estimating musculotendon forces in multiple muscle systems. The sense of an optimum method for solving this problem with respect to its practical usage was studied as well. The musculotendon forces will be used as input data for FEM stress analysis of bones, therefore simple solution should be maintained and the data obtained must be as authentic as possible.

A human musculoskeletal system includes redundant multiple-muscle system allowing the performance of motion. In order to simulate and calculate muscle forces in terms of engineering problem, this redundant system must be optimized.

Two “simple” ways for muscle force calculation in redundant multiple-muscle system are based on: static optimization and known kinematics (inverse dynamics technique) without taking account of physiological and morphological properties. Static optimization and inverse dynamics technique with manifestation of muscle forces is based on the Hill-type model, which includes passive and active muscle properties. It is also possible to express muscle force taking account of tendon dynamics and the properties of musculotendon, expressed by the first-order differential equation. The redundant multiple-muscle system as regards the dynamic properties of musculotendon must be solved by using the dynamic optimization technique, which is not trivial. It is possible to solve such a system using the activation dynamics, and solving the forward motion of this system is a classical control problem.

The necessary constraints for muscle modelling are input data concerning such physiological and morphological properties of muscle as positions of muscle attachments and significant points of body segments, $PCSA$, penalty angle α_0, tendon length L^T, optimum muscle length L_0^M, maximum isometric force F_0^M, etc. Most of these parameters are experimentally estimated (measured), especially in the case where the results (the calculated muscle forces) will be used as inputs to general calculations of FEM stress analysis of bones.

The question is: which estimated muscle forces are better and physiological? Can we get real muscle forces by optimization? The results are much better when a muscle model is based on muscle physiology.

4.1. Is the static optimization a suitable method?

Thus, when the forces generated by all joint muscles, contractors and co-contractors are needed to be known, the raw static optimization methods alone are not sufficient. If the muscle function is represented by equation which defines both active and passive muscle properties, as for example, a Hill-type muscle model in [16] and [19], the co-contractor forces with non-zero values can be obtained.

The co-contractors are the muscles being usually able to lengthten, and activation is usually small. The passive muscle component and tendon can generate force when the co-contractor length is greater than optimum muscle length. The stiffness of the passive component of musculotendon depends on muscle length and has exponential character. The co-contractor forces cannot be equal to zero if co-contractors are lengthened more than the optimum muscle length. Therefore, static optimization can be a suitable technique for muscle forces estimation, but the muscle mechanics must be described by equation which takes into account both active and passive components, for example, equation (5).

Here it is found that by this technique both contractors and co-contractors forces can be estimated.

4.2. The necessity of EMG data collection

The EMG-driven method gives muscle forces with non-zero values because all the muscles, contractors and co-contractors, are almost all time activated. In the case of the co-contractors, the measured activation is very small, less than 10% of normalized EMG. The EMG-driven methods usually make use of the muscle model as regards an active muscle properties or both active and passive muscle properties.

One of physiological driving factors of muscle force is EMG, being processed as muscle activation level a_i (t) (EMG normalized by MVC value). The main problem of these methods is the processing of the normalized EMG signal to muscle activation signal. If the normalized EMG signal is used as activation signal for muscle models, in many cases the joint moments calculated from the forces estimated by raw EMG-driven models are different from the net joint moments calculated by using inverse dynamics approach. The elbow joint moments calculated from forces based on raw EMG-driven models are usually much smaller compared to moments from inverse dynamics technique. Therefore, the muscle forces estimated by using raw EMG-driven models should be optimized, or the activation (normalized EMG) signal in the Hill-type model is optimized.

In this study, the combined technique, i.e., the EMG-driven model with static optimization approach, was used for muscle force estimation in elbow problem. At first, the activation input into EMG-driven model was: i) normalized raw EMG, or ii) EMG signals processed and modified by optimization, or iii) unknown variable which was calculated from optimization. The variant, where an unknown activation signal was calculated from optimization, was easier (a complicated experimental measurement of EMG is necessary) and gave convenient results. Therefore finally, the EMG signals collected experimentally were not used for muscle force estimation. The muscle model in terms of all active and passive properties, such as active and passive force–length relations, force–velocity relation and activation, model equation (5), expresses most of the physiological muscle properties.

Some authors, [19], [16], use the Hill-type models with the processed EMG as an activation signal (EMG-driven models). The question arises: Is a prime necessity to know the muscle activation signal if we want to arrive at the muscle forces, especially if the driven signal (processed EMG) is optimized?

The role of the muscle activation signal is to control an active muscle component. The processed EMG signal is appropriate to be used as activation signal for the first approach only. The findings of this study and the answer to the above question are that for the EMG-driven models with the optimization technique it is not necessary to know muscle activation and recorded and processed EMG, omitting its difficult experimental collection for some muscles.

All of these models are exactly valid for the accepted simplifications and assumptions only. Real forces can differ from simulated forces and can be obtained only by direct measurement [20]. Direct measurements of muscle forces are invasive and generally impractical.

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References

[1] MARTIN C.F., SCHOVANEC L., *Muscle mechanics and dynamics of ocular motion*, Journal of Mathematical Systems, Estimation and Control, 1998, 8, 1–15.
[2] MARTIN C.F., SCHOVANEC L., *The control and mechanics of human movement systems*, Progress in Systems and Control Theory, 1999, 25, 173–202.
[3] PANDY M.G., ZAJAC F.E., *Optimal muscular coordination strategies for jumping*, Journal of Biomechanics, 1991, 24, 1–10.
[4] ZAJAC F.E., *Muscle and tendon: properties, models, scaling, and application to biomechanics and motor control*, Critical Rewiews in Biomedical Engineering, 1989, 17, 359–411.
[5] FUNG Y.C., *Biomechanics: Mechanical Properties of Living Tissues*, Springer-Verlag, Ltd., Berlin 1981.
[6] HUXLEY A.F., *Cross-bridge action: Present views, prospects, and unknowns*, [in:] W. Herzog (editor), *Skeletal Muscle Mechanics*, J. Willey and Sons, Ltd., Chichester, 2000.
[7] MCMAHON T.A., *Muscles, Reflexes, and Locomotion*, Princeton University Press, Princeton, New Yersey, 1984.
[8] GORDON A.M., HUXLEY A.F., *The variation in isometric tension with sarcomere length in vertebrae muscle fibers*, Journal of Physiology, 1966, 184, 170–192.
[9] MASHIMA H., AKAZAWA K., HUSHIMA H., FUJII K., *The force–load–velocity relation and the viscous-like force in the frog skeletal muscle*, Japanese Journal of Physiology, 1972, 22, 103–120.
[10] PANDY M.G., ANDERSON F.C., HULL D.G., *A parameter optimization approach for the optimal control of large-scale musculoskeletal systems*, Journal of Biomechanical Engineering, 1992, 114, 450–460.
[11] ZATSIORSKI V.M., *Kinetics of Human Motion*, Human Kinetics, Champaign, 2002.
[12] VEGER H.E.J., YU B., AN K.N., ROZENDAL R.H., *Parameters for modeling the arm*, Journal of Biomechanics, 1997, 30, 647–652.
[13] PRITULSKI B.I., TADAO I., ALBRECHT A.M., GREGOR R.J., *Is coordination of two-joint leg muscles during load lifting consistent with the strategy of minimum fatigue?*, Journal of Biomechanics, 1998, 31, 1024–1034
[14] CHENG E.J., BROWN I.E., LOEB G.E.D., BRAND R.A., *Virtual muscle: a computational approach to understanding the effects of muscle properties on motor control*, Journal of Neuroscience Methods, 2000, 101, 117–130.
[15] ZHENG N., FLEISIG G.S., ESCAMILLA R.F., BARRENTINE S.W., *An analytical model of the knee for estimation internal forces during exercise*, Journal of Biomechanics, 1998, 631, 963–967.
[16] LLOYD D.G., BESIER T.F., *An EMG-driven muskuloskeletal model to estimate muscle forces and knee joint moments in vivo*, Journal of Biomechanics, 2003, 36, 765–776.
[17] KAUFMAN K.R., AN K.N., LITCHY W.J., CHAO E.Y.S., *Physiological prediction of muscle forces.* II. *Application to isokinetic exercise*, Neuroscience, 1991, 40, 765–804.
[18] CROWNINSHIELD R.D., BRAND R.A., *A physiologically based criterion of Musile force prediction in locomotion*, Journal of Biomechanics, 1981, 14, 793–801.
[19] BUCHANAN T.S., LLOYD D.G., MANAL K., BESIER T.F., *Neuromusculoskeletal modeling: Estimation of muscle forces and joint moments and movements from measurements of neural command*, Journal of Applied Biomechanics, 2004, 20, 367–395.
[20] KOMI P.V., SALONEN M., JARVINEN M., KOKKO O., *In vivo registration of Achilles tendon forces in man*. I. *Methodological development*, International Journal of Sports Medicine, 1987, 8, 3–8.