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Microscopic and Macroscopic Mechanics of Musculotendon Actuation
근육작용에 대한 미시적 또는 거시적인 메커니즘

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Key Words : 기계 일(Mechanical Work), 근육 작동기(Musculotendonr Actuator), 근육수축(Muscle Contraction), 근육 활성화(Muscle Activation), 힘-길이-속도(Force-Length-Velocity)

Abstract : 본 논문은 근수축을 이용한 근육 작용의 운동학을 다룬다. 첫째, 근육 해부학이 미시적 관점의 운동학에 대하여 검토되었으며, 다음으로 거시적 관점의 근육 운동학이 작동 이론을 설명하기 위해 공식화 되었다. 그리고 작동 모델을 평가하기 위해 시뮬레이션 결과가 제시된다. 본 연구는 마이크로미터 거리에 따라 생물학적 분자 운동이 어떻게 거시적 운동으로 바뀌는지를 설명하고 있다. 마지막으로 근육 모델이 거시적/미시적 작동용으로 여러 가지 역학의 형태로 이용될 수 있다는 것을 보인다.

1. 서 론

A striking feature of living cells is their ability to generate various forms of motions. These movements are developed on the molecular level by protein molecules (or biological motors) in the interior of a cell. The protein complexes are responsible for muscle contraction, intracellular transports, and cell mobility[1~2]. In addition, the microscopic muscle contraction underlies many essential functions such as locomotion, breathing, hearting beating, and regulation of blood pressure. A muscle physiology literature contains numerous reports identifying the relationship among force, length, and velocity during muscle contractions[3~5]. The actomyosin can be modeled as a biological actuator whose output force is a function of length and velocity with level of neural activation[6].

Two classic papers by Hill[7] and Huxley[8] provided basic theory for the crossbridge muscle mechanics. Hill-type approach still remains the most popular form of lumped-element model for the whole muscle. The sliding filament theory was tested by measuring the tension-length relationship[9]. In addition, the more adjustment had been made on the length-tension and force-velocity properties[10]. There exists a large body of literature describing the macroscopic dynamics of musculotendon unit[11~13]. Recently, there have been major advances in the understanding of how skeletal muscle develops mechanical force. Nowadays, technology is reaching into ever smaller dimensions. Musculotendon actuator has been considered as nature's premier living biological generators of force, displacement, work, and power. Such a biological actuator would have the potential to be used various fine motion control fields[14] , such as robot, micro-system, and nanotechnology[15~16]. Thus, fine mechanical movement could be powered by tiny protein machines known as biomolecular motors. The applications of muscle molecular motors within an engineering system can take several forms. The aim of this paper is to elucidate the mechanics on force-length-velocity properties for musculotendon actuation.

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2. Modelling for musculotendon actuators

2.1 Molecular basis for muscle contraction

Molecular muscle motors are remarkable cellular machines that convert chemical energy generated by the hydrolysis of ATP (adenosine triphosphate) into mechanical work (force×distance). Three different types of muscle tissues exist in the human body; cardiac, smooth, and skeletal. Cardiac muscle makes up the wall of the heart, used to pump blood through circulatory system. Smooth muscle is found in the walls of all the hollow organs of the body (except the heart).

Before developing actuation models, an understanding of the underlying anatomy and physiology is required. Muscle fibers (diameter: 10~100μm) are just the building blocks for muscle, and are made up of bundles of contractile muscle called myofibrils. As depicted in Fig.1, each sarcomere consists of one set of thick myosin filaments and two sets of thin actin filaments, and its length can be from 1.5~3.5μm long, depending upon contractile state. Under low salt conditions, the actin (mass: 42 kDa) can be isolated as a soluble protein and is roughly dumb-bell shaped. Based on sliding filament theory[8~9], these thick and thin filaments are linked at regular intervals by crossbridges formed from extensions of the myosin molecules to produce mechanical force. A myosin molecule (length: ≈160 nm, mass: 530 kDa) is long, rod-shaped, and has a projection at one end. Several hundred such molecules are arranged in a sheath to form one long myosin myofilament. There are 300-400 actin molecules in a single actin myofilament.

As a myosin head is activated from a neural signal, the arrangement shown in Fig. 1 causes the Z-disks (or Z-lines) to move towards each other. Thick filaments are held to the Z-disk by titin, an elastic protein. During the muscle contraction, the thin filaments anchored at the Z disks are pulled in over the thick filaments so that each sarcomere shortens and generates forces. The A band (or zone) does not change its length,

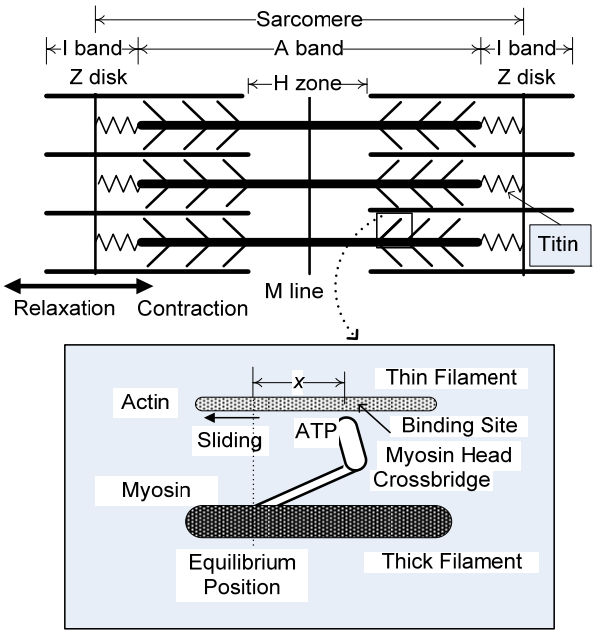

Fig. 1 Illustrative representation of the sarcomere as elemental contractile unit

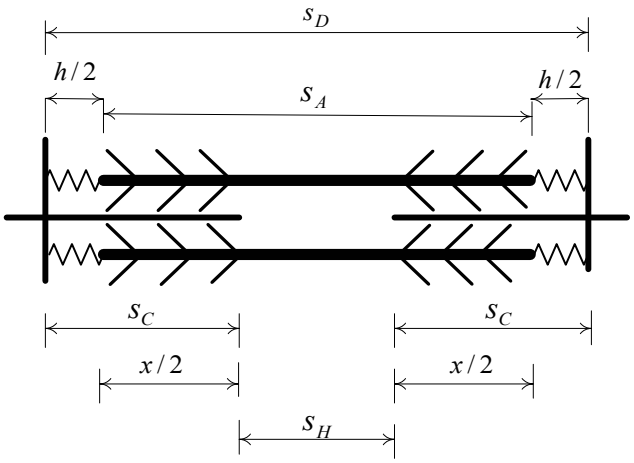

Fig. 2 Schematic diagram of lengths in a sarcomere, with actin and myosin filaments

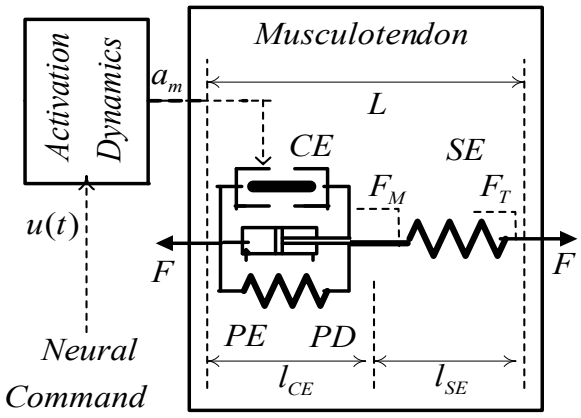

Fig. 3 Diagrammatic representation of musculotendon actuation model

though the distance between Z-lines lessens, and I and H bands narrow.

As shown in Fig. 2, the sliding distance by contraction is calculated as

$$x = s_A - s_H = 2s_c - h \tag{1}$$

Note that x is undefined for a configuration, in which a crossbridge is not attached the actin filament.

2.2 Modelling Musculotendon Forces

The interaction of the actin and myosin filaments when forming crossbridges gives rise to the large-scale, featuring macroscopic physical properties; the force-length and force-velocity relationships. One of the simplest models of muscle fibers, from a mechanical standpoint, is Hill's three component models[7]; the contractile element (CE) where it describes all happening in crossbridges, the parallel elastic element (PE) where it represents the connective tissue sheaths, and the parallel damping element (PD) where it characterizes the viscosity effect.

A twitch is a quick and single contraction caused by an action potential, analogous to an impulse function. A twitch results in relatively little force being produced by the muscle because the action potential does not release enough Ca^{2+} to form a considerable number of crossbridges.

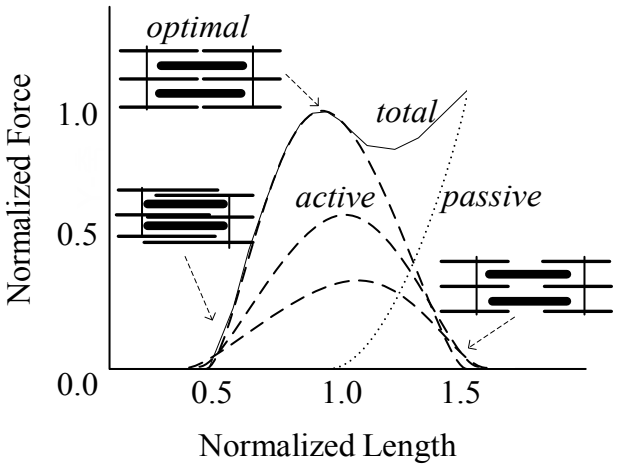

Fig. 4 Normalized force (F_M/F_{MI}^m)-length (l_M) property of various muscle lengths: active (dashed), passive (dotted), and total (solid)

As shown in Fig. 3, each musculotendon actuator is represented as three element muscle fibers in series with an elastic tendon. Thus, the whole muscle can be thought of as having an active part which generates tension when activated by neural stimuli, and a passive part that applies a resistive force when stretched beyond a resting length.

Muscle cannot be activated or relaxed instantaneously. The delay between muscle excitation (u) and activation (a_m) is usually modeled as a first-order process. Thus the contraction dynamics characterize how muscle activations from the motor neurons are transformed into the active forces in CE[12]. The transform from the neural input to a measure of muscle activation is given by

$$\frac{da_m(t)}{dt} + \frac{[\rho_{AD} + (1-\rho_{AD})u(t)]}{\tau_A} a_m(t) = \frac{u(t)}{\tau_A} \tag{2}$$

where ρ_{AD} is the ratio of the time constant for activation to deactivation, $0 \le \rho_{AD} \le 1$, and u(t) is a parameter describing the neural signal to contractile muscle, $0 \le u(t) \le 1$. When the muscle is fully activated (i.e., $u(t)=1$), the τ_A can be a time constant described in equation (2).

The elongation (or strain) of the muscle fiber is defined as follows:

$$\epsilon_{CE} = l_M - 1 \tag{3}$$

where the normalized length is given by $l_M = l_{CE}/l_{CE}^0$. Then the length at which active force peaks F_{MI}^m is coupled to the level of muscle activation as

$$l_{CE}^o(t) = l_{EC}^o[\eta(1 - a_m(t)) + 1] \tag{4}$$

where η is the percent change in the optimal fiber length. As illustrated in Fig. 4, the three dashed curves are representing the active forces depending upon activation levels. Note that the optimal fiber length is longer as the activation decreases.

In practice, the force-length relation represented by the active part is given by

$$F_A^m = a_m(t) f_{CE}(l_M) F_{MI}^m = a_m(t)\left[1 - w_{CE}^{-2}(l_M - 1)^2\right] F_{MI}^m \quad (5)$$

where the parameter w_{CE} has been introduced to regulate the width of the length-tension curve. Similarly, the muscle fiber forces developed by the passive parts are given by

$$F_p^m = \left[f_{PE}(l_M) + f_{PD}(l_M)\right] F_{MI}^m = q_p e^{\{c(l_M - 1) - d\}} F_{MI}^m + d_p l_M F_{MI}^m \quad (6)$$

where c and d are constants; the coefficients q_p and d_p have no physical interpretation; $l_M = l/l_{CE}^0$ is the strain rate. The force factors (f_{PE} and f_{PD}) in equation (6) represent the parallel elastic element and the damping element, respectively. For a given length, the total tensions developed within the muscle fiber are then the sum of the contributions from the active and passive muscle tissues

$$F_M(t) = F_A^m(t) + F_p^m(t) \quad (7)$$

The active contractile force or movement is transmitted through to the tendon. Then the tendon force is given by

$$F_T(t) = w_{SE}^{-2}(l_T - 1)^2 F_{MI}^m \quad (8)$$

where l_{SE}^o is the slack (unloaded) length of the tendon with $l_T = l_{SE}/l_{SE}^0$ and $\dot{l}_T = \dot{l}_{SE}/l_{SE}^o$, and w_{SE} is a parameter for regulating the width of the length-tension curve. Passive elements in the muscle consist of material that does not actively generate force on its own. Rather these materials exhibit tension when strained by external forces.

In contrast, since the total tension ($F_M + F_T$) given in equations (7) and (8) is constant in the isotonic contraction, the following relation should be satisfied

$$\left.\frac{dF_M}{dl_M}\right|_{\dot{l}_M} \dot{l}_M + \left.\frac{dF_M}{dl_M}\right|_{\dot{l}_M} \ddot{l}_M + \frac{dF_T}{dl_T}\dot{l}_T = 0 \quad (9)$$

where the model is assumed to be fully activated, i.e., $a(t)=1$

During muscle contraction, the amount of heat liberated was measured by the temperature change within a muscle[11]. If there is also a change in length, a mechanical work is performed. According to the first law of thermodynamics, the total energy consumption (E) is equal to the heat (Q), plus the work (W):

$$E = Q + W, \; with \;\; W = \int F_M \cdot dl_{CE} \quad (10)$$

Accordingly, the total rate of the muscular energy is given by

$$\frac{dE}{dt} = (\alpha + F_M) \cdot V = \beta(F_{MI}^m - F_M) \quad (11)$$

where a and β are the empirical constants that are used to match the shape of the hyperbolic curve. As described in equation (11), the rate of the heat production ($a \cdot V$) plus the rate of mechanical work performed ($F_M \cdot V$) depended linearly on the load. Thus, the generic form of force-velocity relation is given by the following hyperbolic function:

$$F_M = \frac{F_{MI}^m \cdot \beta - \alpha \cdot V}{V + \beta} \quad (12)$$

When the shortening velocity is zero, then $F_M = F_{MI}^m$. From equation (12), the maximum velocity of shortening occurring at $F_M = 0$ leads to $V_m = \beta F_{MI}^m / a$. As the load increases, the shortening speed decreases. Additionally, the normalized form can be described by

$$V_{NV} = \frac{1 - F_{NF}}{1 + F_{NF}/z} \quad with \;\; z = \frac{\alpha}{F_{MI}^m} = \frac{\beta}{V_m} \quad (13)$$

where V_{NV} and F_{NF} are normalized velocity (V/V_m) and force ($F_M = F_{MI}^m$), respectively, and z is a shape parameter. Consequently, the normalized mechanical power is given by:

Table 1 Simulation data for human muscle

parameter	value	parameter	value
l_{CE}	0.085 [m]	V_m	0.51 [m/s]
l_{SE}^0	0.265 [m]	w_{CE}	0.500
l_{CE}^0	0.090 [m]	w_{SE}	0.030
F_{MI}^m	7500 [N]	z (nominal)	0.250

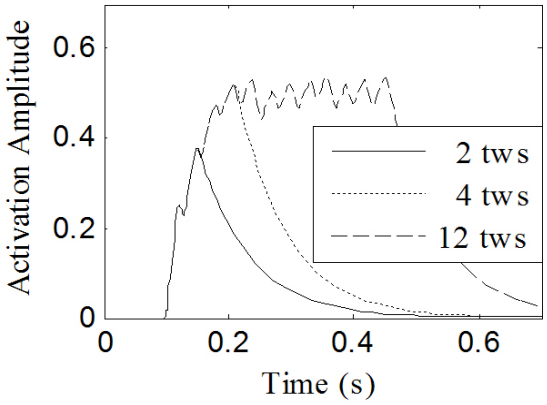

Fig. 5 Activation characteristics with successive twitches (tws)

$$H_{NP} = F_{NF} \cdot V_{NV} = \frac{zF_{NF}(1-F_{NF})}{z+F_{NF}} \tag{14}$$

Based on equations (12), the time rate of the muscle length yields

$$\frac{dL}{dt} = \frac{\beta(F_{MI}^{m} - F_M)}{F_M + \alpha} + \frac{dl_{SE}}{dt} \tag{15}$$

3. Numerical simulation analysis

Skeletal muscle motor is the biological actuator used for controlling movement in a wide variety of situations. In this section, the simulation results are presented to evaluate the actuation theories. First, Table 1 summarizes key data for musculotendon unit [16]. Then the time course of muscle responses is illustrated in Fig. 5, due to successive excitation twitches of amplitude 0.2 with each separation 30ms apart.

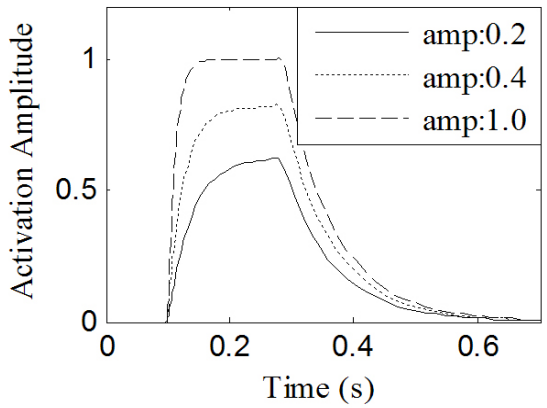

Fig. 6 Activation characteristics due to activation levels (fused tetanus)

If a second stimulus is excited before a muscle relaxes the muscle will shorten further, strengthening more tension than a simple twitch, called summation. In case of temporal summation, the second peak is higher than the first because the further influx of C^{2+} upholds a second contraction, which is added to the preceding contraction. In this test, the time constant τ_A is set to 12 ms, and the ratio of time constant ρ_{AD} is set to 0.15. It is important to recognize that the curve for activation rises rapidly to each maximum after the stimulus and then that for deactivation falls down in a little bit slowly.

As shown in Fig. 6, if many stimuli are repeated at a sufficiently high rate, then the muscle will not relax between each stimulus but rather will remain in a smooth continuous contracted state called tetanus. In this experiment the muscle has been given 12 twitches 15ms apart in which the separation is shorter than that in Fig. 5. In fact, tetanus gives the maximum tension, featuring about 4 times higher than a simple twitch (isometric contraction). Now, the relationship between tension and length has been compared at various muscle lengths (see Fig. 7). Total forces are then the sum of the forces developed in both the active and passive muscle tissues. For the illustration purposes, the formula is found with the following values for the passive force:

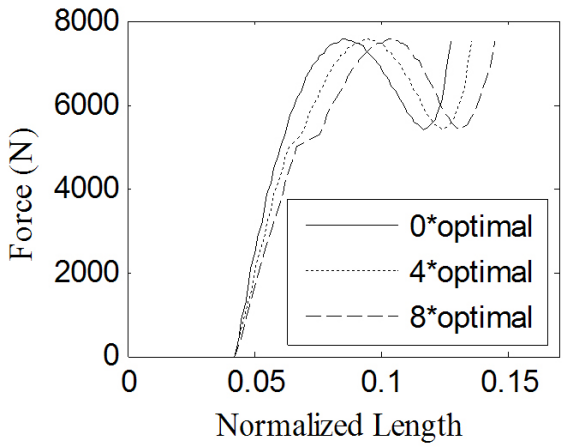

Fig. 7 Tension-length relationship of a skeletal muscle fiber

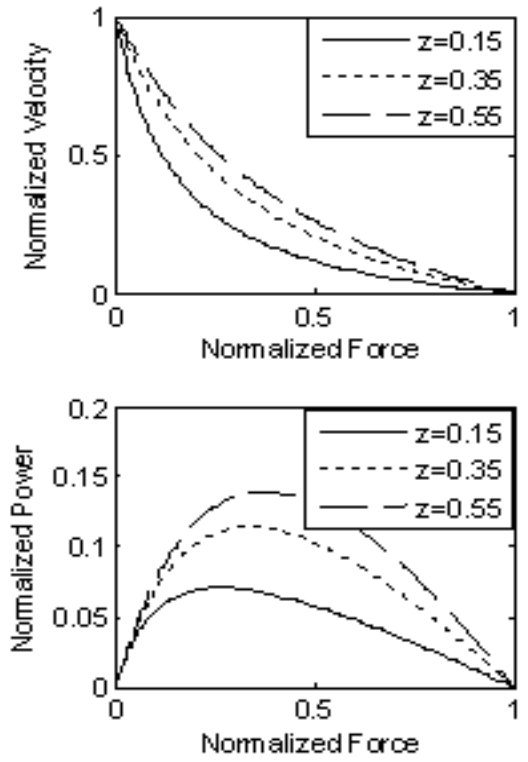

Fig. 8 Dimensionless relationships between force-velocity (upper) and power (lower)

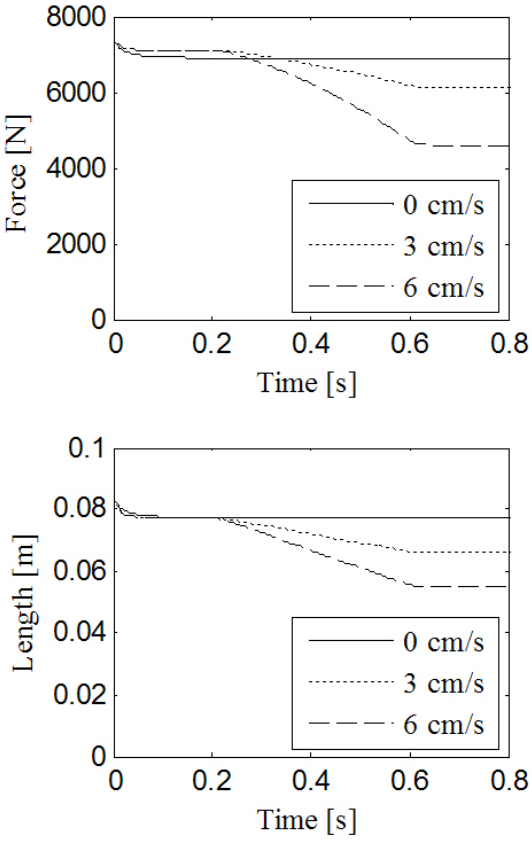

Fig. 9 Isometric force (upper) and length (lower) responses due to ramp change in length

$F_P^m = 1.0e^{(10(l_M-1)-5)}F_{MI}^m$ ignoring the damping term.

It is noted that the slack length is given in terms of optimal length (l_{CE}^o). Based on equations (13) and (14), the shapes of force-velocity (-power) curves are reasonably well (but not uniquely) described in Fig. 8, where the results are normalized with respect to V_m and F_M^{Im}. A reasonable envelope for the shape parameter z is given by $0.15 \leq z \leq 0.55$. It should be noted that the power H_{NP} has a maximum when the load and velocity are about one third of their maximum values.

In the study, the changes in force are described when a decrease of length is imposed on a muscle fiber during contraction. Assuming that the muscle is maximally activated, the isometric force at the corresponding l_{CE} has been calculated from the force-length relationship of CE.

As shown in Fig. 9, the ramp shortening has been imposed at 2 sec with magnitudes of 0, 3, and 6 cm/s. It is noted that dl_{CE}/dt is negative for shortening. Since the total length remains constant, the contractile element can only be shortening by stretching the series elastic element.

5. Conclusions

There has been tremendous interest in understanding biomolecular motors to incorporate them into small novel devices. At first, the micro-mechanism of crossbridge model has been presented to describe the essential actuation theory. The activation mechanics characterizes how the motor neurons stimulate the active forces based on twitch contraction. Next, the force-length response has been presented due to ramp shortening of contractile element. In addition, the force-velocity relation has been analyzed during muscle contraction. This study offers some microscopic and macroscopic aspects to further understand the musculotendon actuator. Finally, the musculotendon model developed can be exploited in various actuators for fine motion control.

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