The Variation of Action Potential and Impedance in Human Skeletal Muscle during Voluntary Contraction

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Liao, T.-J. and Nishikawa, H. *The Variation of Action Potential and Impedance in Human Skeletal Muscle during Voluntary Contraction*. Tohoku J. Exp. Med., 1994, 173 (3), 303-309 — We used an insulated acupuncture needle to deliver a 20 kHz alternative current to detect the changes on the electromyogram (EMG) and deep muscle impedance. We found that impedance increased (with a duration of 224.1 ± 75.3 msec) and then decreased (with a duration of 293.4 ± 97.3 msec) after motor unit action potential (MUAP) firing during isometric voluntary contraction (VC) of the tibialis anterior muscle (TAM). We divided the positive peak into pattern I (with a latency range of ≥ 30 msec and ≤ 75 msec) and pattern II (with a latency range of > 75 msec and ≤ 140 msec). These patterns of change corresponded to the contraction times of tension caused by the electrical stimulation and VC. These impedance changes may be caused by a change in the extracellular and intracellular fractions, and may reflect the variance of tension intensity in the muscle tissue. ——— voluntary contraction; action potential; impedance; insulated acupuncture needle

Impedance changes in muscle during contraction have been studied by Dubussion (1936), who reported that not strictly isometric contraction of a muscle is accompanied by a lowering of impedance, which is proportional to the shortening of fibers. Bozler (1935) also reported that the impedance increased on contraction; it also increased in iodoacetic acid rigor, and decreased in chloroform rigor. These findings suggested that the impedance change varies with the conditions (mechanical shortening or chemical process) in the living muscle. For two decades, the impedance has been continuously recorded and directly used for the calculation of the extracellular space (Pelligrino et al. 1981; Korf and Postema 1988). Although these impedance changes have been described, the relationship between muscle activity and impedance change is still unknown. Herein, we examined the relationship between the muscle activity and impedance change of the tibialis anterior muscle (TAM) during voluntary contraction (VC).

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We conclude that the impedance increases and then decreases after motor unit active potential (MUAP) firing immediately, and according to the change of latencies we divided these variances into two patterns. Each pattern may be dominated by a different muscle fiber with a different contraction time, respectively. Therefore, by recording muscle impedance changes we determined the type of muscle fiber and the rough variation of the muscle tension.

MATERIALS AND METHODS

Studies were conducted on 12 adult volunteers (male, mean 20 years) who were neurologically normal. They sat in a comfortable chair and put their foot on floor naturally in an electrically shielded room, and adjusted their posture so that no muscle activity was observed in the EMG monitored on an oscilloscope.

The recording electrode which is a sterile insulated acupuncture needle (200 $\mu\text{m}\phi$, coated with formvar exposed tip about 1 mm), was inserted into the right Tusanli acupoint (S 36, one finger breadth lateral to the lower border of the tuberositas tibiae, about 9 cm below the knee joint) (Stux and Pomeranz 1987). After needle insertion, the needle was manipulated until the fastest rise-times were secured in the main spikes of the lowest threshold MUAP (Warmolts and Engel 1972) during VC. When starting the experiment, the subjects maintained balances of their heels on the floor and then lifted up the dorsal foot from the floor to less than 5 degrees as the lowest VC, 5 to 10 degrees as a mild VC, and more than 10 degrees as a vigorous VC. The lowest VC was performed to produce MUAP firing, and as soon as the subject heard the burst sound (monitor by an oscilloscope, VC11; Nihon Kohden, Tokyo, and a speaker) VC was stopped. VC was performed for 10 min (per 10 sec a time) of each subject.

In the recording procedure, we measured TAM impedance, EMG and *zeta* potential changes simultaneously during VC. For these measurements we delivered an alternating current of 100 nA at a frequency of 20 kHz by a locally

Fig. 1. Block diagram of the *zeta* potential, EMG and impedance measurements. I-V converter, current-voltage converter; HPF, high-pass filter; BPF, band-pass filter; LPF, low-pass filter; Rect, full wave rectifier; Amp, amplifier; Osc, oscillator (supplied 20 kHz sine wave); N, insulated acupuncture needle (as a leading electrode); S, surface electrode (as a reference).

made device; this current does not cause any activation of the muscle (Fig. 1) (Liao 1992). In this device, a 20 kHz carrier was put on the surface of TAM by the surface electrode as a reference. These three signals of 20 kHz carrier, EMG and needle moving (named as *zeta* potential) will pass the tip of the insulated needle electrode and input to a current-voltage converter during VC, then a low-pass filter (10 Hz), a band-pass filter (10 Hz to 5 kHz) and a high-pass filter (5 kHz) were used to separate the *zeta* potential and the EMG from the 20 kHz carrier, and then to be amplified or rectified (full wave) then to be amplified and recorded as *zeta* potential, EMG and impedance. The power spectrum range of the EMG was approximately between several Hz and 3 kHz, with the most desirable frequency range for recording being 20 to 200 Hz (John 1979), and the *zeta* potential (Goton 1957) of the electrode movement was less than 2 Hz in this study. In this experiment, MUAP activated from the lowest VC up through mild VC or vigorous VC were observed, All of these signals were recorded by a data recorder (XR-50; TEAC, Tokyo), and only one single discharge was analyzed by VC-11 with a QP-110J program (Nihon Kohden).

RESULTS

In the above experiments, MUAP was activated from the minimal to the mild or vigorous effort which caused impedance changes. Usually, the impedance changes are described as an upward (increase) and then a downward (decrease) deflection with a certain latency after firing MUAP (Fig. 2), and these peak latencies and peak amplitudes were different in each volunteer. The maximum procedure of this impedance change ended within 1 sec, and the duration of the positive waves was always shorter than the negative one. A steady rhythmic low frequency (range from 6 to 10/sec) MUAP was activated on minimal to mild VC (Fig. 3a-c). In the period of VC, the *zeta* potential was always recorded and changed only of the initial point and end of the VC. The impedance appeared with a delay time after EMG firing. Different positive peak values of impedance were plotted and showed that many of the components were distributed in the

Fig. 2. Relationship between action potential and the muscle impedance recorded simultaneously from TAM during VC.

AP, action potential; A, amplitude of positive wave; D, duration of positive wave; DI, duration of negative wave; L, delay time; LI, positive peak latency.

Fig. 3. The *zeta* potentials (upper traces), EMGs (middle traces) and impedances change (bottom traces) recorded simultaneously from TAM during the lowest VC (a) and mild VC (b, c).

The former shows a single and the latter shows two rhythmic low frequency MUAP.

TABLE 1. *The data component of impedance recorded from TAM during VC*

Data item	mean $\pm$ s.d.	<i>n</i>
Delay time (msec)	8.0 ± 6.0	130
Positive peak amplitude (Ω)	110.5 ± 94.0	130
Positive peak latency (msec)	66.3 ± 21.3	130
Positive wave duration (msec)	224.8 ± 75.3	130
Negative wave duration (msec)	293.4 ± 97.3	123

The values were mean and s.d. of delay time after MUAP firing, positive peak amplitude, peak latency, wave duration, and negative wave duration recorded from TAM during VC.

short latencies area (Fig. 4). Most of the impedances changed from the range of 30 Ω to 300 Ω during VC. Table 1 shows the average value and standard deviation of positive peak amplitude, peak latency, wave duration, delay time after MUAP firing and negative wave durations. Upward impedance increased with a delay time of 8.0 ± 6.0 msec after MUAP firing, and the average duration of negative waves was longer than that of the positive waves. Fig. 5 shows the distribution of latency percentage content (from Fig. 4). Short latencies (with a latency range of ≥ 30 msec and ≤ 75 msec) showed a high percentage content, and long latencies (with a latency range of > 75 msec and ≤ 140 msec) showed a low percentage content.

DISCUSSION

The size principle (Henneman 1957) is applied to both slow and rapid VCs in man (Desmedt and Godaux 1977), and according to this principle type 1 muscle fibers (red muscle) are fired under mild VC and type 2 (white muscle) are fired under vigorous or sudden VC (Warmolts and Engel 1972). In the case of mild VC in this study, the impedance duration was longer than the firing interval that

Fig. 4. The peak amplitude distribution of each positive latency of impedance recorded from TAM during VC ($n = 130$).

Fig. 5. The percentage content of each positive peak's latency of impedance recorded from TAM during VC.

produced the impedances fusing together (Fig. 3b and 3c). These MUAP and impedance change patterns resembled the relationship between MUAP and contraction time (Buchthal and Schmalbruch 1970; Majima 1978). The electrical impedance of cells and tissues is largely determined by the properties of the surface membrane and these properties in turn depend on the activity of the cell as a whole (Bozler 1935). In this study we just measured the relative impedance change, before, during and after VC. Because any pressure put on the muscle or change of muscle shape will cause an impedance change (Liao 1992), we offset the direct current level and always kept the same impedance level before and after VC.

The simplest explanation for the increase of impedance on contraction would therefore be that it is an increase of the volume concentration of the cells due to swelling (Bozler 1935). When mechanical shortening is completely avoided, the

impedance changes are effected by the chemical processes known to occur in the muscle (Dubussion 1936). In this study, whether these impedance changes are effected by a chemical process or not during VC remains unknown. However, because the evidence to support this suggestion is lacking, the *zeta* potential change and MUAP firing are always observed at the same time in the process of VC. These *zeta* potential changes indicate that the tip of the needle electrode moved in the muscle during VC, for this moving will cause *zeta* potential production (Goton 1957). These findings suggest that the mechanical shortening causes the impedance change.

Most of the individuals in this study showed an impedance increase then decrease, and a few showed an impedance decrease then increase. This may be because the VC is not sufficiently strict to offer a perfect isometric condition. In strictly isometric conditions the contraction is accompanied by a rise in impedance (Dubussion 1936). Nevertheless, we found that the impedance change patterns (duration and amplitude) resembled each other whether there was increase then decrease or decrease then increase. This positive muscle impedance change may reflect the change of muscle tension at the same time, which is caused by a variance in the extracellular and intracellular fractions, because the mechanical shortening may cause cell volume concentration during VC. VC was performed within a short time (10 min) so that the cell swelling causing an impedance change would not need to be considered. The *zeta* potential change is always observed after the end of the VC. This may suggest that the muscle relaxes suddenly and causes a negative impedance change.

The different types of change of muscle impedance might be caused by the different muscle fibers that attend the contraction. TAM contains two different fibers; a fast-twitch-glycolytic fiber (FG fiber) and a slow-twitch-oxidative fiber (SO fiber) (Buchthal and Schmalbruch 1970; Peter et al 1972; Nagata 1984). The contraction time of SO fiber is longer than 60 msec, and that of the FG fiber is shorter than 60 msec (Buchthal and Schmalbruch 1970). In this study, we observed a short latency with a high percentage content (76.9%, 100 of 130 samples) of impedance changes in the range of ≥ 30 msec and ≤ 75 msec, and a long latency with a low percentage content (23.1%, 30 of 130 samples) of impedance changes in the range of > 75 msec and ≤ 140 msec during VC (Fig 4). These values are in contradiction to the distribution rate of 73.4% and 26.6% for FG fibers and SO fibers of human TAM respectively (Nagata 1984). According to these findings we divided the impedance change components of ≥ 30 msec and ≤ 75 msec into pattern I, > 75 msec and ≤ 140 msec into pattern II, and suggested that the FG fiber will activate the former and SO fiber activate the latter impedance change.

The difference in impedance latency and amplitude in each pattern may be caused by the different intensity of VC and the location of electrode, because movement of the electrode or slight increase in the stimulus caused a new fiber

bundle to be activated (Buchthal and Schmalbruch 1970). The negative impedance change may reflect relaxation phase of the muscle change. By means of recording muscle impedance change we may determine the type of muscle fiber and the rough variation of the muscle tension.

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