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Electrophysiological evidence of doubly innervated branched muscle fibers in the human brachioradialis muscle

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Abstract

Objective—Motor-unit action potentials (MUAPs) with unstable satellite (late-latency) components are found in EMG signals from the brachioradialis muscles of normal subjects. We analyzed the morphology and blocking behavior of these MUAPs to determine their anatomical origin.

Methods—EMG signals were recorded from the brachioradialis muscles of 5 normal subjects during moderate-level isometric contractions. MUAP waveforms, discharge patterns, and blocking were determined using computer-aided EMG decomposition.

Results—12 MUAPs with unstable satellite potentials were detected, always two together in the same signal. Each MUAP also had a second unstable component associated with its main spike. The blocking behavior of the unstable components depended on how close together the two MUAPs were when they discharged.

Conclusions—The latencies and blocking behavior indicate that the unstable components came from branched muscle fibers innervated by two different motoneurons. The satellite potentials were due to action potentials that traveled to the branching point along one branch and back along the other. The blockings were due to action-potential collisions when both motoneurons discharged close together in time.

Significance—Animal studies suggest that branched muscle fibers may be a normal characteristic of series-fibered muscles. This study adds to our understanding of these muscles in humans.

Keywords

Electromyography; Motor unit action potential; Satellite potential; Polyneuronal innervation; Branched muscle fiber

Introduction

EMG signals from the human brachioradialis muscle contain pairs of motor-unit action potentials (MUAPs) that exhibit interdependent shape irregularities (Lateva et al., 2002). These MUAPs have volatile components that are delayed or blocked when both MUAPs occur within a specific time interval of each other. A detailed analysis shows that the volatile potentials are produced by shared muscle fibers that are innervated by the motoneurons of both motor units

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at two widely separated endplates (Lateva et al., 2002; 2003). The delay and blocking are due to refractoriness or collision when both motoneurons try to excite the fiber at the same time. Brachioradialis thus appears to be a human example of series-fibered animal muscles in which muscle fibers with multiple widely separated endplates have also been described (Katz and Kuffler, 1941; Jarcho et al., 1952; Zenker et al., 1990; Duxson and Sheard, 1995).

This paper describes another type of irregular MUAP in the normal brachioradialis muscle, namely, MUAPs with volatile satellite potentials. Satellite potentials (or late-latency components) are occasionally seen in other normal human muscles (Lang and Partanen, 1976; Stålberg et al., 1996; Finsterer and Mamoli, 1997). Although in diseased states satellite potentials can occur before or after the main MUAP component, regular satellite potentials in normal human muscles always occur after the terminal wave of the MUAP—the deflection that marks the extinguishment of the main action potential (AP) volley at the muscle/tendon junction (Lateva and McGill, 1999). This is consistent with their being produced by retrograde propagation in non-innervated muscle fibers that are in electrical continuity with innervated fibers at the muscle/tendon junction (Lateva and McGill, 1999). Here we analyze the morphology and blocking behavior of MUAPs with irregular satellite potentials to determine their anatomical origin.

Methods

Intramuscular EMG signals were recorded from the brachioradialis muscle during voluntary isometric contractions in 5 normal subjects (3 males, 2 females, aged 29 to 48). None of the subjects had any history of neuromuscular disease or muscular trauma. The experimental procedures were approved by the Stanford University Committee on the Use of Human Subjects in Research and conformed to the Declaration of Helsinki. Each subject gave informed written consent.

The subjects sat comfortably with the right arm supported in 165° of elbow flexion and neutral forearm rotation. The brachioradialis muscle was identified by palpation during resisted elbow flexion, and the axis of the muscle from the elbow crease to the palpable distal muscle/tendon junction was marked. All distance measurements are given with respect to the elbow crease with positive indicating the distal direction and negative the proximal direction.

EMG signals were recorded simultaneously from six intramuscular electrodes inserted at regular intervals along the muscle axis. Signals were recorded in a monopolar montage with respect to a surface reference electrode placed over the distal muscle tendon. The ground electrode was placed on the back of the hand.

The subjects were instructed to flex the elbow against manual resistance provided at the wrist by one of the examiners. EMG signals were collected in 20-s-long epochs during several low- and moderate-level contractions for each subject. Auditory feedback was provided to guide the subjects in maintaining a constant level of contraction. The signals were amplified (Nicolet Viking, Madison, WI, USA) with filter settings of 5 Hz and 5 kHz, sampled at 10 kHz, and stored on a computer for further analysis.

The EMG signals were digitally high-pass filtered at 1 kHz and were decomposed into their component MUAP trains by an experienced investigator using the EMGLAB program (McGill et al., 2005). Pairs of MUAPs that exhibited interdependent shape irregularity were identified. Separate templates were used for stable and volatile MUAP components to track their configuration from discharge to discharge.

Blocking windows were plotted for each irregular MUAP pair. This was done by pairing each discharge of one MU with the nearest discharge of the other MU and plotting the presence or

absence of the volatile components as a function of the interval between the two discharges. The intervals were measured between the points on the MUAP waveforms at which the first volatile components usually occurred. If a MUAP had no detectable component other than the volatile components themselves, then the interval was measured to the first volatile component.

For each irregular MUAP, the latencies of the volatile components and the terminal wave were determined as in Lateva and McGill (1999; 2001). First, the mean MUAP waveform was averaged from the unfiltered signal using the identified discharge times as triggers. The high-pass-filtered signals were also averaged over those discharges in which the volatile components were present and those in which they were blocked. Because the inter-component latencies exhibited considerable jitter (Lateva et al., 2003), only those occurrences in which the inter-component latency was within ± 0.05 ms of the mean value were included in the averages in order to prevent blurring. These averages were then subtracted to obtain the average of the volatile components. The first deflection of the MUAP waveform from the baseline was taken as the MUAP onset, and the latencies of the other components were measured from this point. The latency of the terminal wave was measured to the peak of the terminal deflection of the MUAP waveform. Some MUAPs did not have detectable terminal deflections. The latencies of the volatile components were measured to their peaks in the high-pass filtered signal.

The architectural organization of the MUs was also estimated. For each MU, the MUAP waveforms at the different recording sites along the muscle axis were determined by spike-triggered averaging, using the identified discharge times as triggers. The MU's mean muscle-fiber conduction velocity was estimated from the slope of the regression line between the latency of the MUAP spike at the different recording sites and the distance of the sites along the muscle axis. For some MUs, the regression lines were not reliable due to poor signal-to-noise ratio at some recording sites. In these cases, the conduction velocity was assumed to equal the mean value estimated for the other MUs in the same muscle. The locations of the MU endplate and muscle/tendon junction were then estimated from the locations of the electrodes, the latencies of the MUAP components, and the conduction velocity (Lateva and McGill, 2001).

Results

A total of 188 MUs were detected. 22 pairs of MUAPs were found to exhibit interdependent shape irregularity. In 16 of these pairs, each MUAP had a single volatile component that could be explained in terms of shared muscle fibers as previously described (Lateva et al, 2002). In the other 6 pairs, each MUAP had two volatile components: one associated with the main MUAP complex, and one that occurred later as a satellite potential. This report focuses on these 6 pairs. Examples are shown in Figs. 1-3. The component latencies are listed in Table 1.

Each of the MUAPs with volatile satellite potentials always occurred in one of three configurations: either both volatile components were present, both were blocked, or the earlier one was present and the later one (i.e., the satellite) was blocked. The fourth possible configuration—the earlier component blocked and the later one present—was never observed. (Figs. 1A,B, 2A,B, and 3A,B).

The presence or absence of the volatile components in a particular discharge always depended on the proximity of the nearest discharge of the other MUAP (Figs. 1C, 2C, 3C). In general, whenever there was a sufficiently large interval between the two MUAPs, both volatile components of both MUAPs were present. Whenever the MUAPs occurred within a certain interval of one another, both volatile components of the later MUAP were blocked. Whenever they occurred within an even closer interval, the later volatile components of both MUAPs were blocked. For example, for the pair shown in Fig. 1C (pair 1 of Table 1), whenever the

interval between MUAP 1.1 and the nearest discharge of MUAP 1.2 was between -22.1 and -11.8 ms (with negative meaning that MUAP2 came first), both volatile components of MUAP 1.1 were blocked. Whenever the interval was between -11.8 ms and $+11.8$ ms, the later volatile components of both MUAPs were blocked. Whenever the interval was between $+11.8$ and $+24.8$ ms, both volatile components of MUAP 1.2 were blocked. Otherwise no components were blocked.

The irregular MUAP pairs fell into three distinct classes based on the characteristics of the volatile components and the blocking behavior. The pair in Fig. 1 (pair 1 of Table 1) is illustrative of the first class. In this class, the earlier volatile component of each MUAP had the same size and shape as the later volatile component of the other MUAP, and the latency between the volatile components was the same in both MUAPs. These findings are explained if the volatile components from both MUAPs came from a single branched muscle fiber that was innervated on each branch by one of the two motoneurons (Fig. 1D). The electrode was located between the endplates and the branching point. For each MUAP, the first volatile component was produced by an AP in the branch innervated by its own motoneuron and the second by a retrograde AP on the other branch. This explains why the first volatile component of MUAP 1.1 resembled the second volatile component of MUAP 1.2 and vice versa, and why the latency between the components was the same in both MUAPs.

The blockings occurred whenever APs from the two endplates collided. If the collision took place between one of the endplates and the electrode (or if a nerve impulse arrived at the endplate while it was still refractory from the passage of an AP from the other endplate), then both volatile components from that endplate were blocked and both volatile components from the other endplate were present. If the collision took place between the electrode and the branching point, then the first volatile components of both MUAPs were present, but the second volatile components were both blocked.

The second class of irregular MUAPs is illustrated in Fig. 2 (pair 4 of Table 1). Here the earlier volatile component of each MUAP resembled the later volatile component of the other MUAP, as in the first class, but, unlike the first class, the latency between the volatile components was considerably different. These observations are explained by the branched fiber configuration shown in Fig. 2D. In this case the branching point was on the opposite side of the endplates from the electrode. For each MUAP, the earlier volatile component was produced by an AP on the branch innervated by its own motoneuron, and the later volatile component by an AP on the other branch. The latency between the volatile components was different because of the different distances of the endplates from the electrode. Whenever APs collided on the loop between the endplates, the later volatile components of both MUAPs were blocked. Whenever a nerve impulse reached an endplate while it was still refractory, both volatile components from that endplate were blocked.

The third class of irregular MUAPs is illustrated in Fig 3 (pair 6 of Table 1). This pair differed from the others in that the earlier volatile components of the two MUAPs were similar to each other, rather than to the later component of the other MUAP. The later components were also similar to each other. The blocking behavior was also somewhat different from the other classes. These MUAPs were only observed to occur in two configurations: with both volatile components present or with both of them blocked. These observations are explained by the configuration shown in Fig. 3D. In this case, a branched fiber was doubly innervated on one branch and not innervated on the other. The electrode was located between the endplates, but very close to endplate 2. The volatile components of MUAP 6.1 were produced by the distally propagating AP (traveling toward the right in the figure) on the innervated branch and the retrograde AP on the other branch. The volatile components of MUAP 6.2 were produced by the proximally propagating AP on the innervated branch and the retrograde AP on the other

branch. An AP collision between endplate 1 and the electrode blocked both volatile components of MUAP 6.1. Whenever a nerve impulse arrived at endplate 2 while it was refractory, both volatile components of MUAP 6.2 were blocked. It should also have been possible for APs to collide between the electrode and endplate 2, blocking only the later volatile component of MUAP 6.2. This situation was not observed, presumably because the electrode was very close to endplate 2 and so such collisions were rare.

If the presented branched-fiber configurations are correct, then certain relationships should exist between the inter-component latencies and the widths of the blocking windows, since both are determined by the propagation times between various points along the fiber. These relationships are illustrated schematically in Fig. 4, where d_1 – d_3 are propagation times, i is the time associated with the initiation of the action potential, and r is the duration of the refractory period (see Appendix for further details). In particular, for the MUAPs in classes 1 and 2, the latencies between the volatile components should equal the widths of the left and right sections of the central blocking window: $L_{a1-b1} = W_3$ and $L_{a2-b2} = W_2$. Furthermore, for all classes, the difference between the latencies of the first volatile components should equal half the difference between the widths of the left and right blocking windows: $L_{a1} - L_{a2} = (W_1 + W_2 - W_3 - W_4)/2$. Moreover, for class 1, the central blocking window should be symmetric: $W_2 - W_3 = 0$, and for class 2, the difference between the widths of the left and right parts of the central blocking window should equal the difference in the latencies of the first volatile components: $L_{a1} - L_{a2} = (W_2 - W_3)/2$. Table 2 shows that these values did in fact closely agree in the observed MUAPs.

The relationships in Fig. 4 also show that the widths of some of the blocking windows should equal r , the duration of the muscle-fiber refractory period. These values, which are marked with asterisks in Table 1, ranged from 2.1 to 3.6 ms (mean 2.8 ms), in line with previous reports (Farmer et al., 1960; Mihelin et al., 1991; Lateva et al., 2002). The relationships for class 1 also provide these formulas for the initiation time: $i = L_{a1} - (W_1 - r)/2$ and $i = L_{b1} - (W_2 - r)/2$. For class 3, the first of these equations also applies, while the second formula becomes $i = L_{b1} - W_3/2$. The values of i computed by these formulas are listed in Table 1. They ranged from 0.8 to 1.2 ms (mean 1.0 ms).

The architectural arrangement of the MUs is shown in Table 3. The locations of the two endplates on the branched fiber, the branching point, and the distal muscle/tendon junction were estimated from the location of the recording electrode, the latency of the terminal wave, the conduction velocity, and the derived values of d_1 , d_2 , and d_3 . The value 2.8 ms was used for r , and the value 1.0 ms was used for i . For the MUs with distal branching points, the branching points were located considerably proximal to the distal muscle/tendon junction. It was not possible to estimate the location of the proximal muscle/tendon junction because the electrode montage did not record the proximal terminal wave.

Most of the time that irregular MUAPs (either with or without satellite potentials) were detected from a pair of MUs, the MUAPs recorded simultaneously from the same pair of MUs at other recording sites did not exhibit irregularities. This is to be expected if the irregularities were caused by single muscle fibers, since it is unlikely that another electrode would be close enough to the same single fiber to record its activity. MUAP pairs 1 and 2, however, did come from the same two MUs at different recording sites. As shown in Table 3, the irregularities at each site were consistent with a branched fiber with the same endplate and branching-point locations. Although it is possible that both electrodes in this case did record from the same branched fiber, it is probably more likely that they recorded from two different, but anatomically similar, branched fibers.

Discussion

The irregular MUAP pairs in the brachioradialis muscle all involved satellite potentials that exhibited intermittent blocking. The component latencies and blocking behavior in each pair could be precisely explained by assuming that the satellite potentials were produced by a single branched muscle fiber that was innervated by both motoneurons. The late components were due to APs that propagated to the branching point along one branch and back along the other. The blockings occurred whenever the APs from the two endplates collided. Three different branched-fiber configurations were observed, differing with respect to whether the endplates were on the same branch or on different branches, and whether the branching point was distal or proximal to the endplates.

Other branched-fiber configurations might be expected to exist as well. A branched fiber could be multiply innervated by the same motoneuron, either on different branches or at different sites on the same branch. This case would be virtually impossible to detect electrophysiologically since the APs from the two endplates would always collide at approximately the same point, and so the MUAPs recorded at any single point along the fiber would always have a consistent configuration. A branched fiber could also be innervated by more than two motoneurons. In this case, the blocking behavior would be more complex, and some components might be blocked most of the time. None of the irregular MUAPs we analyzed fit into this category.

Irregular satellite potentials have not been previously reported in any other normal human muscles, to the best of our knowledge. The six MUAP pairs from five subjects reported here are the only such pairs we found in a wider study of hundreds of MUAPs from 18 subjects. Some of the MUAPs in the wider study had stable satellite potentials that did not exhibit blocking. These may have come from branched fibers that were only singly innervated, or from branched fibers that were co-innervated by motoneurons that were not recruited during the investigated contractions.

Stable satellite potentials are occasionally observed in other normal human muscles. The latencies of satellite potentials in the normal brachial biceps and tibialis anterior muscles indicate that they are due to retrograde AP propagation in recurrent muscles fibers that branch at the muscle/tendon junction (Lateva and McGill, 1999). These recurrent fibers may be the result of longitudinal muscle-fiber splitting (Swash and Schwartz, 1977, 1984; Loughlin, 1993).

The branching points in brachioradialis did not all occur at the muscle/tendon junction. The branching points of MUAP pairs 1, 2, and 6 were located approximately midway between the endplate and the distal muscle/tendon junction. These branching points must therefore have occurred either along the length of the fiber, or at the end of a fiber that terminated intrafascicularly. The other three branching points were located from 24 to 41 mm proximal to the elbow crease. We were not able to estimate where they occurred with respect to the muscle/tendon junction. Given the broad origin of brachioradialis, it is possible that these branching points were located at the proximal muscle/tendon junction.

The human brachioradialis, like sartorius and gacilis (Heron and Richmond, 1993), and latissimus dorsi (Snoble et al., 1998), is known to have intrafascicularly terminating muscle fibers and multiple endplate zones (Feinstein et al., 1955). This suggests that brachioradialis, like many long, parallel-fibered animal muscles, has a series-fiber architecture. Series-fibered muscles are composed of bands of fibers that do not extend all the way from tendon to tendon, but rather terminate intrafascicularly to form interdigitating myomyomal junctions (Chanaud et al., 1991; Hijikata and Ishikawa, 1997; Young et al., 2000). The lack of a tendinous insertion can explain the lack of detectable terminal waves in some of the MUAPs we observed. A series-

fibred architecture allows long muscles with long excursions to be made up of shorter, more electromechanically efficient, muscle fibers. Series-fibred muscles have multiple innervation zones along their lengths to supply the fibers in the various bands. They also have some long fibers, perhaps descended from the primary myotubes, that extend through and receive innervation in more than one endplate zone (Duxson and Sheard, 1995; Lateva et al., 2002).

Intrafascicularly branching muscle fibers may be a normal feature of series-fibred muscle architecture. Such fibers have been reported in developing guinea pig sternomastoid muscles (Paul et al., 2004); in adult rat plantaris muscles (Tamaki et al., 1992); and in adult frog lumbrical, tibialis anterior, and semitendinosus muscles (Brown et al., 1982). The branching may represent a mechanical connection important for force transmission when not all the fibers are directly connected to tendon (Trotter et al., 1995; Young et al., 2000). The distinct branches are presumably innervated separately during development. The processes that normally prevent polyneuronal innervation within a single endplate zone apparently do not operate over the longer distance between the endplates on the separate branches. The purpose of the electrical continuity between the branches remains unknown.

Appendix

This section derives the relationships shown in Fig. 4. Consider panel 1. The diagram on the left shows a branched muscle fiber innervated at endplates e_1 and e_2 . Line el marks the location of the electrode, and the variables d_1 , d_2 , and d_3 represent the propagation times for an action potential to travel between the various points.

The middle diagram shows the morphology of the two MUAPs, with o_1 , a_1 , and b_1 representing the onset and first and second volatile components of MUAP 1 and o_2 , a_2 , and b_2 representing the onset and first and second volatile components of MUAP 2. The latency of the first volatile component equals the propagation time from the endplate to the electrode (d_1) plus a fixed amount i that corrects for the fact that the onset is measured at the time at which the MUAP first departs the baseline in the unfiltered signal and the volatile potential is measured at its peak in the high-pass-filtered signal. The latency between the first and second volatile components equals the propagation time from the electrode to the branching point and back (d_3), which, in this case, is the same for both MUAPs.

The diagram on the right shows the blocking behavior. Let t_1 and t_2 be the times at which nerve impulses arrive at the two endplates. The time plotted along the abscissa represents the difference between the times at which the corresponding muscle-fiber action potentials arrive at the electrode (or would arrive if they were not blocked), namely $t = (t_2 + d_2) - (t_1 + d_1)$. For a collision to occur, the two action potentials must arrive at a particular point at the same time, i.e., $t_1 + T = t_2 + d_1 + d_2 + d_3 - T$, where T is the propagation time from e_1 to the collision site. Combining these equations gives:

$$t = 2T - 2d_1 - d_3$$

For example, if a collision takes place on the top branch at the location of the electrode (i.e., $T = d_1$), then $t = -d_3$. This marks the left boundary of the central blocking window. If a collision takes place at e_1 (i.e., $T = 0$), then $t = -2d_1 - d_3$. The fiber is refractory if the action potential from e_2 arrives up to r ms earlier, where r is the refractory period. Thus the left boundary of the left blocking window is $t = -2d_1 - d_3 - r$. The other boundaries can be determined in a similar way.

The relationships in the other two panels can be derived along these same lines. Note that the variable d_3 is defined slightly differently in each case. The equation for the abscissa of the blocking diagrams for panel 2 is $t = 2T - d_1 + d_2 - d_3$, and for panel 3 it is $t = 2T - 2d_1$.

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Fig. 1.

First class of volatile satellite components. (A) and (B) show MUAP pair 1 from Table 1. The top three traces show several superimposed occurrences of the MUAPs (after high-pass filtering) in each of the three observed configurations: both volatile components present (top traces), satellite blocked (second traces), and both volatile components blocked (third traces). Both MUAPs also have a stable component that was present in every discharge. (In MUAP 1.1, the stable component and first volatile component are superimposed.) The fourth traces show the averaged volatile components. The fifth traces show the averaged MUAP waveforms. o_1 and o_2 are the MUAP onsets, L_a the latency of the first volatile component, L_{a-b} the latency between the volatile components. The terminal waves are not seen. (C) Blocking diagram. Each circle represents one observed pair of discharges of the two MUAPs, with the abscissa indicating the temporal separation between them (negative meaning that MUAP 1.2 came first). Open circles signify that the indicated components were blocked, closed circles that no components were blocked. (D) Schematic diagram of a branched muscle fiber innervated by two motoneurons at endplates e_1 and e_2 . The location of the electrode is indicated by the dotted line. The black arrows indicate an action potential from e_1 producing the volatile components of MUAP 1.1 as it passes and re-passes the electrode. The white arrow shows an action potential from e_2 that collides with the other action potential at x . The location of the collision depends on the relative timing of the nerve impulses. Collisions can only occur between the two endplates.

Fig. 2. Second class of volatile satellite components. (A) and (B) show MUAP pair 4 from Table 1, as in Fig. 1. (C) Blocking diagram, as in Fig. 1. (D) Schematic diagram of the branched fiber configuration.

Fig. 3.

Third class of volatile satellite components. (A) and (B) show MUAP pair 6 from Table 1. Only two configurations were observed: both volatile components present (top traces) and both volatile components blocked (second traces, B only). MUAP 6.1 had no large stable components, and so was only detected when both volatile components were present. (C) Blocking diagram, as in Fig. 1. The gap results from the undetected discharges when both volatile components of MUAP 6.1 were blocked. (D) Schematic diagram of the branched fiber configuration.

Fig. 4.

Diagram showing the expected relationships between the component latencies and blocking windows for the three classes of MUAP pairs. Left: fiber architecture, with endplates e_1 and e_2 , electrode el , and propagation times d_1 , d_2 , and d_3 . Middle: timing of the volatile components a_1 , b_1 , and a_2 , b_2 , with respect to the MUAP onsets o_1 and o_2 . i is the time associated with initiation of the MUAP. Right: blocking windows. r is the refractory period.

Table 1

MUAP morphology and blocking behavior.

Pair	MUAP	Component latencies (ms)				Blocking windows (ms)			<i>i</i>
		L_a	L_b	L_t	L_{a-b}	W_1, W_4	W_2, W_3		
1	1.1	4.6	16.5	21.0	11.9	10.3	11.8	0.9	
	1.2	6.0	17.9	20.0	11.9	13.0	11.8	0.9	
2	2.1	9.0	13.1	21.0	4.1	18.4	4.1	1.2	
	2.2	10.6	14.7	20.0	4.0	22.1	4.1	1.0	
3	3.1	11.3	28.8	20.0	17.5	2.8*	16.8		
	3.2	11.0	27.6	20.0	16.6	3.6*	16.3		
4	4.1	11.4	30.6	21.0	19.2	2.5*	31.0		
	4.2	5.8	36.1	13.5	30.3	2.5*	19.6		
5	5.1	8.7	37.0	20.0	28.3	2.1*	27.5		
	5.2	10.0	36.3	20.5	26.3	2.7*	28.7		
6	6.1	7.0	25.5	16.5	18.5	16.6	0.0	0.6	
	6.2	0.8	19.0	19.5	18.2	3.1*	0.0	0.8	

Latencies: L_a = earlier volatile component, L_b = later volatile component (satellite), L_t = terminal wave, L_{a-b} = latency between earlier and later volatile components. Blocking windows: W_1 = leftmost window (both components of MUAP1 blocked), W_2 , W_3 = left and right parts of center window (both satellite components blocked), W_4 = rightmost window (both components of MUAP2 blocked). In each pair, W_1 and W_2 are listed for MUAP1, and W_3 and W_4 for MUAP2.

* indicates a value that is expected to equal the muscle-fiber refractory period. *i* = estimated time associated with MUAP initiation (see appendix).

Table 2
Relationships between inter-component latencies and blocking-window widths.

Pair	Class	L_{aI-bI}	$\sim$	W_3	L_{a2-b2}	$\sim$	W_2	$L_{aI}-L_{a2}$	$\sim$	$\frac{1}{2}(W_L-W_R)$	$\sim$	$\frac{1}{2}(W_2-W_3)$
1	1	11.9	$\sim$	11.8	11.9	$\sim$	11.8	-1.4	$\sim$	-1.4	$\sim$	0.0*
2	1	4.1	$\sim$	4.1	4.0	$\sim$	4.1	-1.6	$\sim$	-1.8	$\sim$	0.0*
3	2	17.5	$\sim$	16.3	16.6	$\sim$	16.3	0.3	$\sim$	-0.2	$\sim$	0.2
4	2	19.2	$\sim$	19.6	30.3	$\sim$	31.0	5.6	$\sim$	5.7	$\sim$	5.7
5	2	28.3	$\sim$	28.7	26.3	$\sim$	27.5	-1.3	$\sim$	-0.9	$\sim$	-0.6
6	3							6.2	$\sim$	6.7		

Values separated by $\sim$ are expected to be equal. Values marked with * are expected to equal 0. $W_L = W_I + W_2$, $W_R = W_3 + W_4$.

Table 3

Anatomical parameters.

Pair	MUAP	CV (m/s)	location (mm distal to elbow crease)			
			electrode	endplate	branch point	distal tendon
1	1.1	4.8	40	22	69	120
2	1.2	4.8		15		110
	2.1	4.8	60	22	70	120
	2.2	4.8		14		110
3	3.1	4.5	60	14	-24	104
	3.2	4.5		15		—
4	4.1	4.6	60	12	-32	109
	4.2	4.6		38		100
5	5.1	4.6	60	25	-41	117
	5.2	4.6		19		113
6	6.1	3.6	20	-2	53	—
	6.2	3.6		21		91