Amplitude cancellation reduces the size of motor unit potentials averaged from the surface EMG

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Keenan, Kevin G., Dario Farina, Roberto Merletti, and Roger M. Enoka. Amplitude cancellation reduces the size of motor unit potentials averaged from the surface EMG. J Appl Physiol 100: 1928–1937, 2006.—The purpose of the study was to evaluate the influence of selected physiological parameters on amplitude cancellation in the simulated surface electromyogram (EMG) and the consequences for spike-triggered averages of motor unit potentials derived from the interference and rectified EMG signals. The surface EMG was simulated from prescribed recruitment and rate coding characteristics of a motor unit population. The potentials of the motor units were detected on the skin over a hand muscle with a bipolar electrode configuration. Averages derived from the EMG signal were generated using the discharge times for each of the 24 motor units with lowest recruitment thresholds from a population of 120 across three conditions: 1) excitation level, 2) motor unit conduction velocity; and 3) motor unit synchronization. The area of the surface-detected potential was compared with potentials averaged from the interference, rectified, and no-cancellation EMGs. The no-cancellation EMG comprised motor unit potentials that were rectified before they were summed, thereby preventing cancellation between the opposite phases of the potentials. The percent decrease in area of potentials extracted from the rectified EMG was linearly related to the amount of amplitude cancellation in the interference EMG signal, with the amount of cancellation influenced by variation in excitation level and motor unit conduction velocity. Motor unit synchronization increased potentials derived from both the rectified and interference EMG signals, although cancellation limited the increase in area for both potentials. These findings document the influence of amplitude cancellation on motor unit potentials averaged from the surface EMG and the consequences for using the procedure to characterize motor unit properties.

Spike-triggered averages can be derived from the surface electromyogram (EMG) by triggering off the discharge times of a single neuron and averaging into the concurrently recorded surface EMG signal. The size of potentials derived from the EMG signal using the discharges of single motor units as trigger events has been used to measure motor unit synchronization (51, 52, 72), to estimate the number of motor units in a muscle (49, 68), to identify motor units during intramuscular recordings (34, 36), and to assess cross talk between muscles (12, 26).

The technique of spike-triggered averaging relies on a number of assumptions (58, 59), including 1) a fixed delay between the trigger and the response of interest; 2) the absence of correlated activity between the response and other signals; 3) a response waveform that is identical for each trigger event; and 4) an adequate number of trigger events to extract the response. When the technique is applied to averaging from the surface EMG, some of these assumptions may be violated. For example, synchronization of motor unit discharges (72), which can alter the amplitude of the EMG signal (45, 73, 75), may influence the averaged potential. Moreover, the common strategy of deriving averages from the rectified EMG signal introduces problems related to the nonlinear relation between the size of the response and the amplitude of the EMG signal (5, 30, 52) and does not obviate a loss of signal due to cancellation (17).

Computational models of EMG signals have been used to characterize the sensitivity of the surface EMG to selected parameters (6, 13, 25), including amplitude cancellation and motor unit synchronization (39, 45, 73), and have been used to evaluate spike-triggered averaging (4, 27, 30, 74). The purpose of the study was to evaluate the influence of selected physiological parameters on amplitude cancellation in the simulated surface EMG and the consequences for spike-triggered averages of motor unit potentials from the interference and rectified EMG signals. The amount of cancellation was altered by varying excitation level, motor unit conduction velocity, and motor unit synchronization in a population of motor units (39). Results demonstrate the extent to which changes in physiological properties violate the basic assumptions of spike-triggered averaging and influence the size of potentials averaged from the interference or rectified EMGs. A preliminary account of these findings has been published in abstract form (40).

METHODS

The study involved computer simulations that were based on a model of a population of motor units (28) with the addition of a surface EMG model that incorporated muscle fibers with a finite length and a volume conductor with skin, subcutaneous, and muscle layers (25). These models simulate surface-detected motor unit potentials based on the known physiological properties of the first dorsal interosseus muscle. Simulations involved four main steps: 1) specification of the recruitment and discharge times of a population of 120 motoneurons in response to different levels of excitation; 2) generation of motor unit potentials from the number, location, and conduction velocities of the simulated muscle fiber action potentials for each motor unit; 3) simulation of the surface EMG by summing the trains of motor unit potentials; and 4) calculation of the spike-triggered average.
average from the EMG 50 ms before and after each action potential of the reference motor unit. Because the EMG signal is influenced by the distribution of motor unit locations, 20 libraries of 120 motor unit potentials based on random location of motor units were generated for each simulated condition. The area of potentials averaged from the interference, rectified, and no-cancellation EMGs were computed for the 24 motor units with the lowest recruitment thresholds in each library and compared with the area of the original motor unit potential. The no-cancellation EMG, which can be generated using computer simulations but generally cannot be obtained during an experiment, was formed by rectifying the motor unit potentials before they were summed together (17).

**Surface EMG Simulations**

The model was implemented in MATLAB version 6.5 (The MathWorks, Natick, MA). The basic parameters in the model were similar to those published previously (39) and are summarized in Table 1. The distributions of properties across the motor unit pool were based on the size principle (32), and these included innervation number, recruitment threshold, motor unit territory, and conduction velocity of motor unit potentials.

**Motor unit properties.** The simulated muscle had a circular cross section with a radius of 8.67 mm that was derived from physiological cross-sectional areas calculated by Keen et al. (38). The number of muscle fibers was 66,000, based on an average fiber diameter of 56 μm (19), a muscle radius of 8.67 mm, and an estimation that the noncontractile tissue accounted for 20% of the cross-sectional area (33). These values were similar to those in the original model by Fuglevand et al. (28) (71,747 muscle fibers and a muscle radius of 7.5 mm). To match the exponential relation between the number of motor units and motor unit force (41, 43, 53) and the high correlation between innervation number and tetanic force of a motor unit (8, 37), the number of fibers innervated by a single motoneuron increased exponentially from the smallest to the largest motor unit. Innervation numbers in the present study ranged from 26 to 2,510 fibers, based on the 100-fold range of twitch forces in first dorsal interosseus that have been estimated by spike-triggered averaging (53, 70) and intra-muscular microstimulation (22).

**Activation characteristics.** Activation of the motor unit pool was modeled as a ramp-and-hold function, with a 1-s ramp increase in excitation to a mean level that was constant for 2,500 s. Isometric contractions of this duration were necessary to obtain 20,000 trigger events to generate all spike-triggered averages, thereby satisfying the fourth assumption related to the technique of spike-triggered averaging (see Introduction): namely, that an adequate number of trigger events exists to extract the waveform of the response. However, fewer trigger events are typically used in experimental measurements (64, 72).

The distribution of recruitment thresholds for the motoneurons was determined from an exponential function with many low-threshold neurons and progressively fewer high-threshold neurons (18, 55). Each motor unit began discharging at 8 pulses per second (pps) once excitation exceeded the assigned recruitment threshold of the unit, and discharge rates increased linearly with increased excitation; the rate was 3 pps per 10% increase in excitation. Excitation corresponded to the simulated net synaptic input onto the motoneuron pool (31), and it was assumed that all neurons received the same level of excitation. As a consequence, the simulated input-output functions of the neurons matched the well-established relations between discharge rate and injected current (42). Maximal excitation was denoted as the level of input necessary to bring the last recruited motoneuron to its assigned peak discharge rate, and values of excitation were expressed as a percentage of this maximum. As described by Fuglevand et al. (28), the first recruited unit had a maximal discharge rate of 35 pps, whereas peak discharge rate decreased linearly with increasing recruitment threshold, with the last recruited unit assigned a peak discharge rate of 25 pps. The last unit was recruited near 50% maximal excitation. The discharge rate was modeled as a random process with a Gaussian distribution, with the coefficient of variation for discharge rate set at 20%.

**Generation of motor unit potentials.** The volume conductor was an anisotropic and inhomogeneous medium representing the muscle, subcutaneous (1.5-mm-hick), and skin (1-mm-thick) tissues. Ultrasound measures were used to determine the thickness of subcutaneous and skin tissues used in the model (39). Each parallel tissue layer was homogeneous, and the conductivities of each layer were the same as in Farina and Merletti (25): muscle was anisotropic and more conductive in the longitudinal than the transverse fiber direction (conductivity ratio = 5), whereas the subcutaneous and skin tissues were isotropic. The conductivity ratio was 20 between skin and fat layers and 0.5 between fat and muscle. The analytical description of the intracellular action potential was based on Dimitrova and Dimitrov (64, 72), and 0.5 between fat and muscle. The analytical description of the intracellular action potential was based on Dimitrova and Dimitrov (64, 72), with the time course of the intracellular action potential divided into three parts: the rising phase was approximated as described by Rosenfalck (60), the beginning of the repolarization phase was described by one-half a cubic spline, and the negative afterpotential was represented by an exponential function.

The distribution of the motor unit territories within the muscle (35, 50) was determined by randomly selecting the x-y coordinates corresponding to the center of each motor unit territory within the circular cross section of the muscle. The fibers of a motor unit were randomly scattered in the motor unit territory (67), with a density of 20 fibers/mm² (3), and interdigitated with fibers belonging to many other units. The territories of the largest motor units, therefore, were greater than those of the smallest motor units (7). To restrict the distribution of the fibers of a motor unit within the cross section of the muscle (7, 67), motor unit territories were modeled to be circular (10). The radius of the motor unit territory was based on the density of 20 fibers/mm² and the assigned innervation number of the motor unit. However, when a portion of the motor unit territory was constrained by the muscle boundary, the territory of the unit was modified to fit the muscle cross section, and fiber density was changed to accommodate the required number of motor unit fibers within the new territory. The constraint on motor unit territory had little effect on fiber density for small motor units, but resulted in a greater fiber density for the largest motor units, which is consistent with experimental findings (3, 37). In previous versions of the model (28, 73), motor unit territories were restricted to be circular and within the muscle; thus the largest motor unit territories overlapped within the center of the muscle, resulting in high fiber densities. In the present study, average muscle fiber density

### Table 1. Description of model parameters

<table>
<thead>
<tr>
<th>Model Parameters</th>
<th>Values</th>
</tr>
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<tbody>
<tr>
<td>Muscle properties</td>
<td></td>
</tr>
<tr>
<td>Muscle cross section (radius)</td>
<td>8.67 mm</td>
</tr>
<tr>
<td>Number of motor units</td>
<td>120</td>
</tr>
<tr>
<td>Number of muscle fibers</td>
<td>66,000</td>
</tr>
<tr>
<td>Number of fibers in a motor unit (range)</td>
<td>26–2,510</td>
</tr>
<tr>
<td>Skin thickness</td>
<td>1.5 mm</td>
</tr>
<tr>
<td>Subcutaneous tissue thickness</td>
<td>1 mm</td>
</tr>
<tr>
<td>Average fiber length</td>
<td>40 mm</td>
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<tr>
<td>Innervation zone (location along fiber from proximal to distal attachment)</td>
<td>40%</td>
</tr>
<tr>
<td>Innervation zone and tendon spread</td>
<td>5 mm</td>
</tr>
<tr>
<td>Conductivity ratios</td>
<td></td>
</tr>
<tr>
<td>Skin and subcutaneous tissue</td>
<td>20</td>
</tr>
<tr>
<td>Subcutaneous tissue and muscle</td>
<td>0.5</td>
</tr>
<tr>
<td>Muscle—longitudinal and transversal to fiber direction</td>
<td>5</td>
</tr>
<tr>
<td>Detection system</td>
<td></td>
</tr>
<tr>
<td>Electrode shape and diameter</td>
<td>Circular, 4 mm</td>
</tr>
<tr>
<td>Interelectrode distance</td>
<td>10 mm</td>
</tr>
<tr>
<td>Center of detection system (location along fiber from innervation zone to distal attachment)</td>
<td>50%</td>
</tr>
</tbody>
</table>
was \(-280 \text{ fibers/mm}^2\), and constraining the motor unit territories resulted in a more uniform fiber density throughout the muscle than in previous studies.

The surface-detected motor unit potential comprised the sum of the action potentials of the muscle fibers belonging to the motor unit. Based on experimental measures (39), average fiber length was 40 mm, and the center of the innervation zone was located at 40% of the length of the fibers from the proximal attachment. The end plate and insertion of each fiber into the tendons varied randomly within the motor unit (uniform distribution) over a range of 5 mm, matching the narrow distribution found using surface electrode arrays over the first dorsal interosseus (39, 61). The 120 motor units had a mean and Gaussian distribution of conduction velocities of $4 \pm 0.4 \text{ m/s}$ (24, 71), with the smallest motor units assigned the slowest conduction velocities (1). The simulated signals were detected with a bipolar electrode configuration and circular electrodes (4-mm diameter) to replicate the methods commonly used to record EMG of the first dorsal interosseus muscle (16, 29, 64, 74). The bipolar electrodes were located halfway between the innervation zone and the distal tendon, with 10 mm between the centers of the two electrodes. Motor unit potentials and the EMG signal were computed at 4,096 samples/s.

### Simulated Conditions

Selected conditions were simulated to determine their influence on the area of potentials averaged from the surface EMG. Conditions were selected (Table 2) that have been reported to alter the amount of cancellation (27, 39, 73), and each condition was varied independently (default values shown in Table 2) to address the following two questions.

**Question 1:** Do changes in the number of active motor units and the durations of their potentials influence the area of potentials derived from the interference and rectified EMGs? Because variation in the number of active motor units and the duration of their potentials do not violate the assumptions required for accurate spike-triggered averaging (see Introduction), the expectation was that potentials averaged from the interference EMG would accurately reproduce the area of the original motor unit potential. However, variation in the number of active motor units and potential durations does influence EMG amplitude and amplitude cancellation (39, 54) and were expected to have an effect on potentials extracted from the rectified EMG. To alter the number of active motor units, three levels of excitation were simulated: 5, 10, and 15% of maximal excitation (Table 2). These levels of excitation resulted in average full-wave rectified EMG amplitudes (aEMG; mean of 2,500 s) that were 1, 5, and 9% of maximal aEMG, respectively, and involved the activation of 24, 52, and 68 motor units at mean discharge rates of 8.5, 9.6, and 10.9 pps, respectively. These low levels of excitation were chosen to mimic the range commonly examined in experimental studies (65, 72, 10.9 pps, respectively). These low levels of excitation were chosen to mimic the range commonly examined in experimental studies (65, 72, 10.9 pps, respectively). These low levels of excitation were chosen to mimic the range commonly examined in experimental studies (65, 72, 10.9 pps, respectively). These low levels of excitation were chosen to mimic the range commonly examined in experimental studies (65, 72, 10.9 pps, respectively).

Muscle fiber conduction velocities, which can decrease during sustained contractions (23, 66, 76), range from 3 to 5 m/s (see review in Ref. 2). To vary the durations of the motor unit potentials, which are inversely related to motor unit conduction velocity (47), mean conduction velocity varied from 2.5 to 5 m/s at 5% maximal excitation (Table 2). Average motor unit potential duration for the 120 motor units across the 20 random motor unit locations was 16.9, 10.2, and 8 ms at mean conduction velocities of 2.5, 4, and 5 m/s, respectively.

**Question 2:** Does motor unit synchronization influence the area of potentials derived from the interference and rectified EMGs? Based on prior reports (30, 52), the expectation was that motor unit synchronization would increase the area of potentials averaged from the rectified EMG, but not the interference EMG. Motor unit synchronization was examined by using an established scheme (69, 73) that adjusted the timing of selected action potentials to impose a temporal association between action potentials discharged by different motoneurons (56). Briefly, four levels of motor unit synchronization were imposed at 5% maximal excitation by applying a function that selected between 5, 10, 20, and 30% of the action potentials discharged by each motor unit to be synchronized with the discharges of six other motor units. Discharges of the reference motor unit were randomly selected, and when a discharge of another randomly selected motor unit occurred within $\pm 30$ ms of the discharge by the reference motor unit, this discharge time was aligned with the reference unit with a Gaussian distribution that had an SD of 1.67 ms centered on the reference unit. This process was repeated until the discharges of six other motor units were aligned with the discharge of the reference motor unit for each discharge of the reference motor unit selected for this adjustment, and ad seriatim for all motor units in the pool. Motor unit synchronization was quantified with the common input strength (CIS) index, which indicates the number of synchronous discharges above chance in the cross-correlation histogram constructed from the discharges of pairs of motor units divided by the duration of the trial (57). CIS values were calculated and averaged for every pair of motor units active (24 motor units, 276 comparisons). The result of imposing the four levels of motor unit synchronization was average CIS values of 0.5, 1.0, 1.5, and 2.0 (Table 2), which match the range of values observed experimentally for first dorsal interosseus (46, 63).

### Data Analysis

**Spike-triggered averaging procedure.** For each simulated condition, average potentials were computed from the interference, rectified, and no-cancellation EMGs (Fig. 1). The following procedures were used to compute the area of the averaged potentials: 1) 2,500 s of surface EMG were simulated for each condition and 20,000 trigger events were used to generate each spike-triggered average; 2) spike-triggered averages were generated for the 24 motor units with the lowest recruitment thresholds across the 20 libraries of motor unit locations for each of the simulated conditions; 3) each spike-triggered average had a duration of 100 ms centered around the positive peak of the reference motor unit potential (solid vertical gray line; Fig. 1B); 4) mean baseline level of motor unit activity for each spike-triggered average was calculated as the mean EMG amplitude in arbitrary units (AU) for the first 40 ms in each spike-triggered average; and 5) area of the averaged potentials was calculated as the absolute value of the spike-triggered average from 10 ms before the positive peak to 10 ms after the negative peak in each spike-triggered average (between dashed vertical gray lines; Fig. 1B), after first subtracting the mean baseline level. Twenty thousand trigger events were used so that the original motor unit potential could be extracted from the spike-triggered average, even for the smallest motor unit potentials.

**Amplitude cancellation index.** Amplitude cancellation has previously been quantified by comparing the amplitude of the surface EMG before and after all motor unit potentials were rectified (17, 39). In the present study, an amplitude cancellation index was calculated for each reference motor unit to quantify the portion of its signal that was lost in the interference EMG due to overlap with other motor unit potentials. The following steps were used to compute the amplitude cancellation index: 1) the aEMG amplitude was calculated for the train of

### Table 2. Description of simulated conditions

<table>
<thead>
<tr>
<th>Simulated Conditions</th>
<th>Values</th>
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<tbody>
<tr>
<td>Excitation level</td>
<td>5*, 10, and 15% of maximal</td>
</tr>
<tr>
<td>Mean motor unit conduction velocity</td>
<td>2.5, 4*, and 5 m/s</td>
</tr>
<tr>
<td>Motor unit synchronization (average CIS)</td>
<td>0*, 0.5, 1.0, 1.5, and 2.0</td>
</tr>
</tbody>
</table>

CIS, common input strength. *Default condition.
surface-detected motor unit potentials; 2) the difference between the aEMG before and after removing the potentials of the reference motor unit was subtracted from the value derived from step 1; and 3) the value from step 2 was divided by the value from step 1 and multiplied by 100 to normalize the amplitude cancellation index for each motor unit. The rationale for the index was that, if the decrease in aEMG after removal of the potentials of the reference motor unit equals the average EMG amplitude of the full-wave-rectified motor unit potential train, this would indicate that no cancellation occurred between the potentials of the reference motor unit and other motor unit potentials. However, the decrease in aEMG after removal of the reference motor unit potential train was always less than the aEMG amplitude of the reference motor unit potential train, with the amplitude cancellation index indicating the amount of cancellation for each motor unit.

RESULTS

Selected parameters that influence cancellation between the opposite phases of motor unit potentials were adjusted to examine their effect on the area of potentials averaged from the interference and rectified EMGs.

Excitation Level

The influence of excitation level on the area of the averaged potentials was examined at 5, 10, and 15% maximal excitation (Fig. 2), which were associated with EMG levels that ranged from 1 to 9% of maximum. When using 20,000 trigger events, the area of one representative surface-detected motor unit potential at 5, 10, and 15% maximal excitation was accurately extracted from the interference and no-cancellation EMGs. In contrast, the area of the original motor unit potential was underestimated by the average derived from the rectified EMG and was nearly absent at 15% maximal excitation (Fig. 2E). The areas of averaged potentials from the interference, rectified, and no-cancellation EMGs are depicted for motor units 1–24 across 20 libraries of random motor unit locations at 5, 10, and 15% maximal excitation in Fig. 2, B, D, and F, respectively. At each excitation level, there was a linear relation between the areas of the original motor unit potentials and those extracted from the interference and no-cancellation EMGs ($r^2 > 0.99$; slope of the regression line $\sim 0.98$). However, the area of the potentials averaged from the rectified EMG was less than that of the original motor unit potentials. For example, the potentials derived from the rectified EMG using the representative surface-detected motor unit potential in Fig. 2 (area = 17.0 AU·ms) decreased relative to the original potential by 32, 64, and 75% at 5, 10, and 15% maximal excitation, respectively, which compared with the reductions for the largest motor unit potential (area = 102.4 AU·ms) of 13, 23, and 38% relative to the original potential. Although the absolute change in the area of the potentials averaged from the rectified EMG was greatest for the largest potentials (Fig. 2F), the percent change in area was smallest.

Cancellation in the Rectified EMG

To examine the role of cancellation in the potentials averaged from the rectified EMG, the decrease in its area relative to the original motor unit potential was compared with the amplitude cancellation index for each motor unit potential (see METHODS). The amplitude cancellation index and the percent decrease in the potential averaged from the rectified EMG relative to the original motor unit potential were linearly related for motor units 1–24 across 20 libraries of random motor unit locations at the three levels of excitation (Fig. 3). The percent decrease in the potential averaged from the rectified EMG relative to the original potential seldom exceeded 80%, because random fluctuations in the baseline level of the rectified spike-triggered average were similar to the area of the reference surface-detected potential at high levels of amplitude cancellation. This effect was most obvious for small potentials at higher excitation levels (e.g., Fig. 2E). The association between the amplitude cancellation index and the decrease in the size of the potential extracted from the rectified EMG can also be calculated by comparing the potential averaged from the no-cancellation and rectified EMGs, as indicated by the top two traces in Fig. 2, A, C, and E. Because the area of the original motor unit potential was similar to the potential averaged from the no-cancellation EMG, but was underestimated from the average from the rectified EMG, the decrease in the potential derived from the rectified EMG was attributable to cancellation between overlapping positive and negative phases of action potentials.
Motor Unit Conduction Velocity

At 5% maximal excitation, changing the mean motor unit conduction velocity altered the area of the potentials derived from the rectified EMG, but not the interference and no-cancellation EMGs at each excitation level, but it was underestimated for the rectified EMG. The area of the potential derived from the rectified EMG progressively declined as excitation increased from 5 to 15% (from A to E). B, D, and F: the area of the potentials averaged from the interference, rectified, and no-cancellation EMGs were calculated for motor units 1–24 across 20 random motor unit locations at 5, 10, and 15% maximal excitation, respectively (480 data points for each excitation level), and compared with the area of the original surface-detected potential. The area of the surface-detected potential for all motor units was successfully extracted by averaging from the interference and no-cancellation EMGs, but not by averaging into the rectified EMG. The underestimation with the rectified EMG was greatest for large potentials. The data for the potentials extracted from the no-cancellation EMG are indicated with a linear regression line ($r^2 > 0.99$; slope $-0.98$).

Motor Unit Synchronization

Potentials averaged from the EMG at 5% maximal excitation were generated with motor unit synchronization imposed at average CIS values of 0, 0.5, 1.0, 1.5, and 2.0. For two representative motor units, potentials averaged from the interference EMG [spike-triggered average 1 (STA 1) and 2 (STA 2)] are shown on the same scale at varying levels of motor unit synchronization (CIS values = 0, 1, and 2; Fig. 5A). With no motor unit synchrony, the area of STA 1 was 2.2 AU·ms, and synchronization increased the area to 37.3 AU·ms (CIS = 1.0) and 62.6 AU·ms (CIS = 2.0). Without motor unit synchrony, the area of STA 2 was 25.4 AU·ms, and synchronization increased the area to 40.8 AU·ms (CIS = 1.0) and 58.7 AU·ms (CIS = 2.0). The percent change in area of potentials averaged from the interference EMG was typically greatest for the smaller motor unit potentials (e.g., compare the increase in STA 1 vs. STA 2 in Fig. 5A). Similar effects were observed for potentials derived from the rectified EMG (Fig. 5B).

Although the area of potentials averaged from the interference and rectified EMGs at 5% maximal excitation increased to a similar extent with motor unit synchronization, the magnitude of the increase was less than the increase for potentials extracted from the no-cancellation EMG. For example, the average area for the surface-detected potentials of motor units 1–24 across 20 random motor unit locations was 9.9 AU·ms, which increased to 29.8 and 46.8 AU·ms for potentials averaged from the interference EMG at an average CIS of 1 and 2, respectively. In contrast, potentials derived from the no-cancellation EMG increased to 40 and 66.1 AU·ms, respectively. Consequently, the effect of synchronization on the areas of the potentials averaged from the interference and rectified EMGs was reduced by cancellation.
Further analysis was performed on experimental data (Fig. 6) to examine the possibility that potentials averaged from the interference EMG recorded over first dorsal interosseus could increase at greater force levels, possibly due to motor unit synchronization, as suggested for the trapezius muscle by Westad and Westgaard (72). This possibility was examined in a competitive weight lifter, who had previously been identified as having high levels of motor unit synchronization with an average CIS of 2.4 (46). Spike-triggered averages were generated using data collected by Moritz et al. (55), which involved recording the surface EMG and the discharges of the same motor unit across a range of force levels in first dorsal interosseus. Spike-triggered averages derived from the interference and rectified EMGs were generated by the motor unit discharges at average force levels of 2 and 7% maximal voluntary contraction (Fig. 6), and the area of the potentials was computed from experimental data, as described for the simulated data. Consistent with the findings of Westad and Westgaard (72), the area of potentials averaged from the interference EMG increased with force level (7.6 to 14.0 mV/ms). Potentials derived from the rectified EMG also increased with force level (5.5 to 9.25 mV/ms).

**DISCUSSION**

The purpose of the study was to evaluate the influence of selected physiological parameters on amplitude cancellation in the simulated surface EMG and the consequences for spike-triggered averages of motor unit potentials from the interference and rectified EMG signals. Cancellation between opposing phases of motor unit potentials influenced the area of potentials averaged from both the interference and rectified EMGs, but the effect varied across conditions. An increase in excitation level and a decrease in mean motor unit conduction velocity decreased the area of potentials derived from the rectified EMG, but not those from the interference EMG. The decrease in area of potentials extracted from the rectified EMG was quantitatively related to the amount of amplitude cancellation in the EMG signal. Furthermore, motor unit synchronization resulted in similar increases in the area of potentials obtained from the rectified and interference EMGs, but cancellation limited the increase in area for both potentials.

**Effect of Amplitude Cancellation**

The influence of amplitude cancellation on potentials averaged from the rectified EMG is depicted in two ways in the
The rectified EMG was examined. Results of the present study range investigated experimentally, and its influence on the EMG signal (30, 52). Excitation level was varied over the derived from the rectified EMG based on the variance of the descriptions that quantified the expected value of the potential 

Fig. 6. Potentials averaged from the interference (A) and rectified (B) EMGs increased with target force in a subject with high levels of motor unit synchronization. STAs were generated from experimental data previously collected (56) in a subject with high levels of motor unit synchronization (average CIS level = 2.4) (46). The subject performed an isometric contraction of the first dorsal interosseus muscle at different force levels, and a single motor unit was identified with an intramuscular EMG recording. Potentials were extracted from the surface EMG recorded with a bipolar electrode configuration triggered by the discharges of the single motor unit at 2 and 7% maximal voluntary contraction (MVC) (821 and 858 triggers, respectively). The areas of the potentials obtained from the interference and rectified EMGs were measured between the dashed vertical lines in a manner identical to that used for the simulated data. The size of potentials derived from the interference and rectified EMGs at the higher force level increased, consistent with the findings of Westad and Westgaard (72).

The increase in area of the potentials derived from the interference EMG with motor unit synchronization was limited by amplitude cancellation. The amount of cancellation at 5% maximal excitation was substantial; the area of potentials obtained from the interference EMG were 25.5 and 29.2% less than those derived from the no-cancellation EMG at mean CIS level of 1 and 2, respectively. The magnitude of the cancellation depends on the extent to which timing variability introduces overlap between the opposite phases of the synchronized potentials, and timing variability is influenced by such motor unit properties as axonal conduction velocities (15, 44), activity-dependent axonal excitability (11), and muscle-fiber con-

Influence of Motor Unit Synchrony

Although motor unit synchronization increased the size of potentials averaged from the rectified EMG, consistent with the increase predicted by Milner-Brown et al. (51, 52), synchronization also increased the area of the potential derived from the interference EMG (Fig. 5). It is generally assumed that the potential extracted from the interference EMG will yield the waveform of the surface-detected motor unit potential, because the positive and negative phases of the potentials of the synchronized motor units will cancel one another. However, this effect depends on the durations of the synchronized potentials relative to the variability in the timing of the synchronized potentials. Hamm et al. (30) reported a minimal contribution of a synchronized potential (~0.5 ms duration) to an average derived from an interference neurogram after adding timing variability (SD 0.25 ms, Gaussian distribution, their Fig. 2). In the present study, the duration of surface-detected motor unit potentials averaged ~10 ms at a mean conduction velocity of 4 m/s, which is similar to the duration of potentials detected experimentally in first dorsal interosseus (72, 75). Timing variability of the synchronized motor unit potentials was simulated with an SD of 1.67 ms to match the variability in the width of the peak in histograms formed by cross-correlating the discharges of pairs of motor units (62). Because the durations of the motor unit potentials in first dorsal interosseus are larger than the timing variability between surface-detected motor unit potentials, the potentials of synchronized motor units contribute to the potentials averaged from the interference EMG.

The increase in area of the potentials derived from the interference EMG with motor unit synchronization was limited by amplitude cancellation. The amount of cancellation at 5% maximal excitation was substantial; the area of potentials obtained from the interference EMG were 25.5 and 29.2% less than those derived from the no-cancellation EMG at mean CIS values of 1 and 2, respectively. The magnitude of the cancellation depends on the extent to which timing variability introduces overlap between the opposite phases of the synchronized potentials, and timing variability is influenced by such motor unit properties as axonal conduction velocities (15, 44), activity-dependent axonal excitability (11), and muscle-fiber con-
Implications for Experimental Findings

Although the simulations in this study were limited in scope to spike-triggered averages generated at low levels of excitation for specific surface EMG recordings, the study has practical implications for the interpretation of experimental results. The current results agree with Westad and Westgaard (72), for example, that motor unit synchronization can influence the area of potentials averaged from the interference EMG (Fig. 5), but may contradict their conclusion that the amount of synchronization necessarily changes with contraction intensity. The increase in the area of the potential averaged from the interference EMG with increased contraction intensity, which does not occur in the absence of motor unit synchrony (Fig. 2), may be a consequence of the reference potential being synchronized with larger motor unit potentials as contraction intensity increases. For example, the increase in the size of the potentials extracted from the interference EMG of a subject with high levels of motor unit synchronization as he increased target force (Fig. 6) may have been caused by an increase in the number of active motor units and not an augmentation of motor unit synchronization.

Amplitude cancellation and motor unit synchronization likely decrease the effectiveness of the spike-triggered averaging technique to quantify various motor unit properties, including estimates of motor unit number (9, 68) and size (14). There is discussion over which method is most appropriate to identify motor unit properties: stimulation of the motor nerve or spike-triggered averaging during voluntary contractions (21, 48). The present results suggest that the ability to identify motor unit properties by spike-triggered averaging is limited due to amplitude cancellation and motor unit synchronization and imply that the use of motor nerve stimulation may be a more appropriate approach. The influence of amplitude cancellation and motor unit synchronization on potentials averaged from the rectified EMG is difficult to quantify due to the many factors that can influence the amount of cancellation. Potentials averaged from the interference EMG are less equivocal, although the estimated areas are influenced by motor unit synchronization.

In summary, the decrease in area of potentials averaged from the rectified EMG was linearly related to the amount of amplitude cancellation in the interference EMG, which varied with different motor unit properties. Motor unit synchronization resulted in similar increases in potentials extracted from the rectified and interference EMG for the conditions simulated in this study. These findings indicate limitations in the estimation of motor unit properties based on potentials averaged from the surface EMG.

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REFERENCES


