Influence of motoneuron firing synchronization on SEMG characteristics in dependence of electrode position

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Received 26 December 2000; accepted in final form 29 May 2001

Kleine, Bert U., Dick F. Stegeman, Daniela Mund, and Christoph Anders. Influence of motoneuron firing synchronization on SEMG characteristics in dependence of electrode position. J Appl Physiol 91: 1588–1599, 2001.—The frequency content of the surface electromyography (SEMG) signal, expressed as median frequency (MF), is often assumed to reflect the decline of muscle fiber conduction velocity in fatigue. MF also decreases when motor unit firings synchronize, and we hypothesized that this effect can explain the electrode-dependent pattern in our previous recordings from the trapezius muscle. An existing motoneuron (MN) model describes the afterhyperpolarization following a spike as an exponential function on which membrane noise is superimposed. Splitting the noise into a common and an individual component extended the model to a MN pool with a tunable level of firing synchrony. An analytical volume conduction model was used to generate motor unit action potentials to simulate SEMG. A realistic level of synchrony decreased the MF of the simulated bipolar SEMG by ~30% midway between endplate position and tendon but not above the endplate. This is in accordance with experimental data from the biceps brachii muscle. It was concluded that the pattern of decrease of MF during sustained contractions indeed reflects MN synchronization.

Surface electromyography; short-term synchrony; fatigue; electromyogram modeling; biceps brachii muscle

Surface electromyography (SEMG) is often used to study muscle electrophysiology and central motor drive in fatiguing exercises (10, 43). A fatigued motor unit (MU) generates less force per motoneuron (MN) firing event. This decline must be compensated for by higher firing rates and/or recruitment of other MUs. Therefore, the amplitude (root mean square, RMS) of the SEMG will increase with fatigue if the force is kept constant. With fatigue at high isometric forces, the concentration of extracellular ions changes (potassium in particular; Ref. 39), and the muscle fiber conduction velocity decreases. This results in a compression of the spectrum of the SEMG and a reduction of its median frequency (MF) (27, 45). The increased spatial length of the intracellular action potential with decreasing velocity will lead to increased amplitudes. Although this is partially counteracted by an increased spatial dispersion, it could contribute to the increasing RMS with fatigue (42). Although the MF depends on the position of the recording electrode relative to the fibers (22, 36), conduction velocity and MF decrease by almost the same percentage in electrically evoked responses (26), a relationship that is confirmed by simulation studies (42). However, in voluntary contractions, the MF drops more than the muscle fiber conduction velocity (11, 21, 31). In the biceps muscle, a decrease of MF is also found at forces as low as 15% of maximum voluntary contraction (MVC), although the conduction velocity is unchanged during fatigue (20). These authors concluded that changes in the firing pattern, especially synchronization, of the MNs must be responsible for the spectral shift to lower frequencies. Short-term synchrony between two MNs, i.e., more firings at (almost) the same time than expected by chance, is caused by synaptic input that is common to both MNs. The synchronizing input could be assumed to be common to all MNs of a pool. Corticospinal cells have widespread projections to the pool of MNs supplying a muscle (29), and short-term synchrony is absent in patients after a contralateral stroke (7, 8). Synchrony also increases with force (16) and in an attention-demanding task (37). To overcome fatigue, the central drive to a muscle has to increase, and as fatigue progresses more synchrony due to increased corticospinal activity could be expected. Furthermore, during fatigue the input that a MN receives from muscle afferents decreases (9), requiring even more supraspinal input.

An increase of firing synchrony with fatigue has not been directly proven because of several methodological limitations. Measurement of synchrony requires the stable and accurate recording of single MU activities. At low forces, the recording could be done with high-density SEMG (18). However, this method cannot be applied with high forces to decompose the complex interference pattern. Needle electrodes can be employed to record MU firing patterns during fatigue, but
they tend to get out of place with long and forceful contractions.

Indirect evidence for synchronization comes from simulations of MN firing patterns and the corresponding SEMG (12, 44). In these simulations, synchrony was induced by shifting a certain percentage of the discharges to be simultaneous with a reference MN. Although these models allow useful predictions, they do not attempt to describe the physiological mechanism of short-term synchrony.

An SEMG model connecting a pool of MNs to MU action potentials (MUAPs) was developed to predict the effect of synchrony on MF at different electrode positions. The present study extended the model MN of Matthews (23) to synchronize the firings of a pool. Matthews described the membrane potential of a MN after the discharge of an action potential by an exponential function. The spike phase and the early part of afterhyperpolarization (AHP) are not considered, because no new spikes can be generated in this refractory phase, and the course of the early part of the AHP is not influencing the occurrence of the next spike. During a steady isometric contraction, each spinal MN in the active pool receives an inflow of predominantly excitatory postsynaptic currents. The finite number of neurons innervating the MN and their discrete discharges produce a fluctuation of the net input. Matthews described the depolarization caused by the constant inflow as an offset of the membrane potential on which fluctuations were superimposed. The fluctuations were described as white noise that was filtered by using the time constant of the membrane. This approach separates the net excitatory drive from its variability, although they cannot be separated experimentally.

The synaptic input to a particular MN comes from many different sources that could have different distributions within the MN pool. One extreme would be input that is unique for a particular MN and does not reach the other MNs. The other extreme corresponds to a branching of the axons to form an evenly distributed projection to the whole MN pool. These two sorts of input lead to the extension of Matthews’ (23) model in the present study: Synchrony was induced by splitting the noisy input into a “common” and an “individual” component. The common noise represents the input that is shared by all MNs of the pool. Peaks in the common noise discharge all MNs of the pool. The individual component of noise varies between MNs and represents the input that is unique to a particular MN. The firing sequence of each MN of the pool of 300 MNs was used to generate a MUAP train in which the specific MUAP was generated with an analytical volume conduction model (1) that accurately predicts MUAPs of the biceps muscle (32). The MUAP trains of all MUs were added to simulate a SEMG signal.

In a recent study of the trapezius muscle (19), our laboratory described the topography of RMS and MF during fatiguing shoulder elevation with 50% MVC. Apart from the expected dependence of MF on electrode position, the normalized change of MF was position dependent as well. Above the endplate zone, the monopolar MF decreased more than it did closer to the fiber end. By contrast, the bipolar MF fell less at the endplate but decreased more midway between endplate and muscle fiber end.

It was hypothesized that synchronization, simulated by increasing the percentage of common input to the MN pool, can explain the electrode-dependent changes of SEMG characteristics during fatigue.

To confirm the above findings, a multichannel SEMG study on the biceps muscle contracting at 20% MVC was conducted. This particular muscle was selected because the volume conduction model was constructed and validated for this muscle (32). A moderate force level was measured to minimize the effect of conduction slowing. Central fatigue, which was expected, should synchronize firing.

METHODS

SEMG recordings. Ten healthy volunteers (5 female, 5 male), mean age 24 yr (range 22–28), gave their informed consent to participate in the study. The subjects sat in front of a table. The position of the arm was such that the angle in the elbow joint was 90°. In this way, isometric elbow flexion force was measured by a wrist belt attached to a strain gauge sensor. The forearm was supinated to reduce the influence of the brachioradialis muscle. Three trials of MVC were performed, and the best attempt was accepted as MVC if the force differed by <10% between trials. After application of SEMG electrodes, the subject sustained a force of 20% MVC for 15 min. All subjects were able to maintain the target force for that period.

Thirty-two channels of SEMG were recorded from a rectangular 4 × 8 electrode grid with an interelectrode distance (IED) of 1 cm. The Ag/AgCl electrodes (diameter 5 mm) were placed in an elastic cuff around the upper arm. Care was taken to align the columns of eight electrodes parallel to the muscle belly. The distal electrode row was placed at the transition between muscle and tendon. With the elbow at 90°, the electrode grid covers about the distal two-thirds of the muscle belly. The endplate zone is therefore in the proximal third of the grid, as was confirmed by the resulting RMS profiles. The monopolar signals (referenced to the olecranon) were amplified, filtered between 10–700 Hz, and analog-to-digital converted with a resolution of 2.44 μV/bit (range 12 bit) and a rate of 3,906 s⁻¹-channel⁻¹ (Biovision, Wehrheim, Germany). The signals were stored on hard disk for subsequent off-line processing.

SEMG processing. The SEMG processing steps were essentially the same as in a previous experiment in the trapezius muscle (19). SEMG intervals of 1,048 ms (4,096 samples) were selected every 30 s during the measurement of 15 min. For each channel the calculation included the following steps. After baseline shifts were removed by subtracting the signal mean and digital low-pass filtering (400 Hz, second-order Butterworth), the mean amplitude (RMS) of each interval was calculated. After the linear trend was removed from the signal, the power spectrum was estimated by using the fast Fourier transform algorithm. The MF was calculated. By use of a linear regression on all the data points, the intercept of the RMS (RMS₀) and the RMS after 15 min of fatiguing exercise at 20% MVC (RMSᵢ) were calculated. The normal-
ized change of RMS was then calculated as \((\text{RMS}_F - \text{RMS}_0)/\text{RMS}_0\). Similarly, \(\text{MF}_0\), \(\text{MF}_F\), and the normalized change of MF \([(\text{MF}_F - \text{MF}_0)/\text{MF}_0]\) were calculated from linear regression. Bipolar montages were constructed by subtracting the signals recorded monopolarly from adjacent electrodes in the fiber direction (distal-proximal). In addition to bipolar montages with an IED of 1 cm, montages with an IED of 2 cm were calculated by skipping one electrode. The same procedure as above was applied to the bipolar SEMG signals. RMS, MF, and their normalized change were averaged across subjects for each monopolar or bipolar channel. The standard error of mean was calculated.

**Simulation of motoneuron firing behavior.** In the MN model introduced by Matthews (23), the time course of the membrane potential contains an AHP starting after each spike and approaching the threshold exponentially (Fig. 1, gray line). Noise is superimposed on this average membrane potential trajectory. White noise with a sampling interval of 1 ms (Fig. 1, top traces) is generated from a Gaussian distribution with zero mean and a standard deviation (SD) of one and is fed into an impulse response of a simple first-order passive low-pass filter with a time constant of 4 ms, the latter to simulate the decay of postsynaptic potentials. After such filtering, performed as described by Matthews, the high-frequency content of signal decreased (Fig. 1, third trace) and the SD increased to 1.58.

Because intracellular recordings from MNs cannot be performed in humans, the absolute amplitude of the AHP and amplitude of the membrane noise are unknown. However, it was shown by Matthews (23) that the model output depends only on the amplitude of the AHP relative to the noise level. Therefore, the SD of the filtered membrane noise that is put into the model is defined as 1 noise unit (NU). Voltages in the MN model are then expressed in NU instead of millivolts. From comparison of intracellular recordings from cat lumbar MNs, it was estimated that 1 NU corresponds to \(-2 mV\) (23). However, in such recordings, the noise differs between MNs and is dependent on the depth of anesthesia.

A new spike is generated by the MN as soon as the threshold is reached. After the spike, the MN membrane potential is reset to the AHP envelope. Here an AHP starting at \(-20\) NU and decaying with a time constant of 30 ms was used (Fig. 1, bottom trace). These values were found to describe the firing pattern of biceps MUs accurately (23). The input to a MN is introduced by shifting the whole membrane trajectory by a constant voltage. The firing rate of MN was calculated as \(1/(\text{mean interspike interval})\). As done by Matthews, intervals longer than 300 ms were excluded because slow and irregular firing can be a problem in experimental recordings. The relation between input voltage (in NU) and firing rate as estimated from simulated spike trains of 30 s duration is given in Fig. 2A. The firing rate cannot be lower than 3.3 Hz and was set to zero when no interspike intervals shorter than 300 ms were present. This causes a sudden increase of the firing rate in Fig. 2A. A negative input voltage indicates that the average AHP will not reach the threshold. Firings can still be induced by noise peaks. For the estimation of the topographical pattern of SEMG characteristics, the model was run with mean inputs of 6 and 10 NU. An input of 6 NU induces a firing rate of 14.8 Hz, which is within normal values for the biceps contracting with 10 and 30% MVC (5). Between the MNs of the pool, the input was varied randomly according to a Gaussian distribution with a SD of 0 (no variation), 1, or 2 NU but was constant within each MN over the simulated time. Adding input voltage to AHP of the model MN is equal to lowering the threshold. From a physiological point of view, the distribution of input voltages simulates a MN pool with different thresholds.

**Synchrony between motoneuron firings.** Short-term synchrony is generated by synaptic input that is common to a pair or a group of MNs (8). The above model MN was therefore slightly modified to allow for two sorts of noisy input. One part of the membrane noise is common to all MNs of the pool (simulating branched input), and another part was unique for each MN. A weighted average of the individual noise and the common noise was calculated with the required

![Fig. 1](http://jap.physiology.org)
percentage of common input as weighting factor. Thus, for the condition “25% common input” to the MN pool, the noise signal was composed of 25% of the common noise signal and 75% of the individual noise signal for each MN. The combined noise was then filtered with a time constant of 4 ms as in the nonsynchronization case. The SD of the filtered noise was kept at 1 NU.

The amount of synchronous firing of a pair of MNs can be estimated from their cross-correlation histogram (8). Short-term synchrony refers to a higher number of nearly simultaneous firings than expected by chance. Nordstrom et al. (28) define the common input strength (CIS) as the number of extra simultaneous firings per second, i.e., the area of the peak in the cross-correlation histogram above baseline, normalized to the duration of the recording. The baseline was estimated as the mean number of spikes within a latency window of 10–50 ms before the reference spike (37). The number of extra simultaneous spikes was the total number of spikes within 10 ms before or after the reference spike minus the number of spikes expected according to the baseline count. CIS is not dependent on firing rate and is therefore superior to normalization to the number of firings (28). Because CIS can be estimated for any pair of spike trains and the percentage of common input is a property of the particular MN-pool model presented here, the relationship between these measures is not straightforward. The dependence of CIS on the percentage of common noisy input is given in Fig. 2B. At low forces, a CIS of ~1 s\(^{-1}\) is expected, but it can increase to 2.5 or 3.8 s\(^{-1}\) at forces of 50 and 100% MVC, respectively (16). CIS can only be used to measure the synchrony between pairs of MNs. To measure synchrony in a MN pool, Yao et al. (44) introduced the population synchrony index (PSI). First, the number of coincident impulses, i.e., how often two, three, or more MNs fire within the same millisecond, is calculated and weighted with a binomial factor. Then, the PSI is calculated by subtracting the number of coincident impulses expected due to chance with normalization to this number. Therefore, PSI provides a normalized value of the total number of coincident impulses for all MNs in excess of that expected due to chance. (For more details on the derivation of PSI, see Ref. 44.) The PSI for a pool of 300 MUs is given in Fig. 2C. In their simulation, Yao et al. considered a PSI of 1 as moderate synchrony and a PSI of 3 as high synchrony. An example of a firing pattern of 50 MNs receiving an input of 6 NU without and with synchrony induced by 50% common noise is given in Fig. 2D and E, respectively.

**Simulation of MUAPs.** The analytical volume conduction model of Blok (1) was used to simulate the potential distribution generated by an MU at the skin. In this model, the volume conductor is described by three cylindrical layers representing an inner muscle compartment (radius 3.6 cm), a subcutaneous fat layer (2 mm), and a skin layer (2 mm). The length of the cylinder was set to 25.6 cm. The conductivity of skin was 0.75 S/m and that of fat was 0.05 S/m. The radial conductivity of the anisotropic muscle tissue was 0.1 S/m, and the axial conductivity was 0.5 S/m.

The bioelectric source was simulated as a muscle fiber aligned parallel to the axis of the cylinder. The intracellular action potential was calculated according to Rosenfalck (35). It propagated with a velocity of 4 m/s toward both tendons. With histological methods, the average innervation ratio of the biceps muscle was estimated to be 750 muscle fibers per unit (6). Because of the skewed distribution of MU sizes within a muscle, most MUs are smaller than average (17) and all MUs were simulated as if consisting of 500 muscle fibers.

**Fig. 2.** Firing patterns generated by a pool of model MNs. A: increase of firing rate with input voltage. B: common input strength (number of extra synchronous discharges per second) of an MN pair at different percentages of common input. C: population synchrony index of a pool of 300 MNs with increasing common input. In B and C, firing rates of the MNs varied from 9 s\(^{-1}\) (black) to 12 s\(^{-1}\) (gray) and 15 s\(^{-1}\) (black line with ●). D: 2 s of the firing pattern of a pool of 50 MNs firing independently at 14.8 Hz (input 6 NU). E: synchronized firing of the same 50 MNs with 50% common noisy input.
fibers. The motor endplate of each fiber was at the axial middle of the cylinder and was allowed to vary with an SD of 3 mm between fibers. The muscle fibers had a length of 10 cm (5 cm in both directions from the endplate), and the position of the fiber end at the tendon varied with an SD of 4 mm. The onset of the action potential varied by an SD of 0.2 ms between fibers. The MUAP output of the volume conduction model had a sampling rate of 2,000 s⁻¹. The model parameters used here are taken from Roeleveld et al. (32) or from Blok (1).

The model calculated MUAPs for MU depths of 6–25 mm below cylinder surface in steps of 1 mm. The MUAP was stored with a spatial resolution of 1 mm in the longitudinal direction and ~2 mm along the circumference of the cylinder. To simulate the whole muscle, 300 MUs were randomly positioned within the muscle compartment at a depth of at least 6 mm from the surface and not deeper than 2.5 cm from the electrode position (Fig. 3). To simulate some physiological variability in the position of endplate regions and muscle fiber ends, the MUAPs were also randomly shifted longitudinally with an SD of 5 mm. The MUAP at the recording electrode was taken from the model run with the appropriate depth and relative electrode position along the circumference. An electrode diameter of 5 mm was simulated by averaging all simulated positions closer than 2.5 mm. For each of the 300 MUs, the MUAP at positions between ~7 cm and 7 cm (resolution 2 mm) from the middle of the cylinder (the average endplate position) was stored. Similarly, bipolar MUAPs were generated by subtracting the monopolar MUAPs from positions with an IED of 1 or 2 cm.

Simulated surface EMG interference pattern. From the firing pattern of 300 MNs and their corresponding 300 MUAPs, an SEMG interference pattern was generated by adding the MUAPs at the appropriate points in time generated by the MN model. With histological methods, the number of MUs in the biceps muscle was estimated to be 774 (6). This is in sharp contrast with a MU number estimate of 109 when using electrophysiological methods (25). The difference could be explained by the fact that the MUAPs from deep and distant MUs are small and could be missed during the incremental stimulation. Therefore, the electrophysiological method is more likely to underestimate the true number of MUs. Because not all MUs will be recruited at 20% MVC, a number of 300 MUs was used in the model. The number of MUs was not a critical parameter because a model-run with 100 MUs showed almost equal results. To estimate the MF and the RMS, 15 s of SEMG were generated. After 4 s were skipped from the beginning, 10 epochs of 2,048 samples were used to calculate MF and RMS, as was done for the measured data. The means of MF and RMS were plotted as a function of recording electrode position. The influence of synchrony was analyzed by plotting the difference in MF between the synchrony and the no-synchrony conditions normalized to the no-synchrony condition (normalized change). For the accurate calculation of the power spectrum at some selected electrode positions, 60 s of SEMG were generated and the power spectrum was estimated by averaging over 50 epochs with 2,048 samples.

RESULTS

Experiment. The RMS of the biceps muscle SEMG recorded in an elbow flexion of 20% MVC is given in Fig. 4. The highest monopolar RMS was measured just proximal from the middle part of each electrode row (Fig. 4A). The bipolar RMS was relatively low in that region (Fig. 4, B and C). The region of low bipolar RMS corresponds to the endplate position of the MUs, where action potentials, propagating in opposite directions, partially cancel. For the medial electrode rows, the endplate region is less clearly distinguished. During 15 min of fatiguing contraction, the RMS increased at all electrode positions. The monopolar RMS increased by 87–128%, whereas the bipolar RMS increased even by ~200% at some electrodes.

The monopolar MF was relatively independent of electrode position in the nonfatigued biceps (Fig. 5A). With fatigue, the monopolar MF decreased at all positions but more prominently at the distal electrodes (Fig. 5D). The bipolar MF was high in the endplate region, lowest between endplate and tendon, and higher again at the most distal recording positions (Fig. 5, B and C). This pattern was more prominent with 2 cm IED than with 1 cm IED. As above for the RMS, the pattern was more obvious in the lateral electrode rows. Changes of bipolar MF were weakest at the endplate region and about two times stronger midway between endplate and tendon.

Simulation. With a common input of 50%, a CIS in the physiological range was obtained (Fig. 2B). By using these model parameters, a PSI of ~3 was reached, a value that was considered to be high synchrony by Yao et al. (44). When the firing patterns in Fig. 2, D and E, are compared, the synchrony of firing is obvious. Synchronization increased the amplitude substantially (Fig. 6, A, B, D, and E). In the power spectrum, synchronous firing increased the power at low frequencies (Fig. 6, F, G, I, J, and K). When the range of firing rates was narrow (the same threshold for all MNs), there was a prominent peak in the spectrum at the firing rate and its first harmonic (Fig. 6J). The increase in the low frequencies depends on electrode position and montage. It is almost absent for bipolar signals from the endplate zone (Fig. 6, C and H).
Figures 7 and 8 demonstrate the influence of synchrony for all electrode positions that were simulated. The model run with a mean input of 6 NU and a SD between MNs of 2 NU, corresponding to a mean firing rate of 15.5 Hz (SD 4.2), is shown. The monopolar RMS was highest above the endplate region and increased most strongly with synchronization there (Fig. 7, A and D). At the same position, the bipolar RMS was low and did not increase with synchrony. A strong increase of the bipolar RMS was found between endplate region and tendon (Fig. 7, B, C, E, and F). The monopolar MF was lowest at the endplate position and increased toward the fiber end (Fig. 8A). Synchronous firing decreased the monopolar MF by 10–15% above the endplate region but by only ~5% above the fiber end (Fig. 8D). The bipolar MF was highest at the endplate...
region and lower between endplate end tendon (Fig. 8, B and C). Synchrony did not influence the bipolar MF directly above the endplate position but decreased the bipolar MF by ~30% proximal and distal from that position. The influence of synchrony on bipolar MF was slightly stronger with an IED of 1 cm than with 2 cm IED (Fig. 8, E and F).

With a mean input of 6 NU and SD of 0, 1, or 2 NU, the mean ± SD firing rates were 14.8 ± 0.1, 14.8 ± 2.0, and 15.5 ± 4.2 Hz, respectively. A mean input of 10 NU increased the firing rates to 24.8 ± 0.2, 25.0 ± 3.2, and 26.1 ± 8.2 Hz. Variability of firing within each MN caused a small variation of firing rates between MNs, even with a uniform input to the MN.
pool. The slight changes of mean firing rate with variable input were caused by the non-Gaussian distribution of firing rates in the MN pool. As expected, there was almost no difference in mean ± SD of firing rates between 0 and 50% common input. Firing rate or firing rate distribution hardly influenced initial MF or the induced changes in MF. Regardless of firing rate, 50% common input decreased the MF by almost the same amount.

**DISCUSSION**

An analytical volume conduction model (1) was connected to a MN model (23) to simulate the SEMG pattern over a whole muscle. The firings of the MN pool were partially synchronized by noisy input that was common to all MNs. The profile of RMS and MF that was predicted by the simulations was in accordance with experimental results from the trapezius muscle.
In the biceps muscle, the highest bipolar MF was found above the endplate region (Fig. 5, B and C), a finding that also confirms results from vastus lateralis and tibialis anterior muscles (15, 36). In the trapezius muscle (19) the pattern of monopolar MF was qualitatively inverse to that of the bipolar MF, which is similar to the model output presented here. The monopolar MF measured in the biceps muscle, however, does not exhibit any clear-cut pattern. No relation to the endplate zone, as defined by a low bipolar RMS, was found. This unexpected finding could be explained by electrical activity from deep or distant sources that only affect the monopolar signal. Whereas bipolar signals are mainly influenced by sources close to the electrodes, monopolar signals reflect deep sources and far-field potentials as well (41). Roeleveld et al. (33) compared the monopolar and bipolar RMS of the biceps muscle. At low to moderate forces the highest monopolar RMS was more distal than the lowest bipolar RMS. This finding was explained by a more distant position of the motor endplates of deep MUs. In the simulation presented here, the endplate position was allowed to vary between MUs, but not systematically with depth. However, with a difference of the endplate position of ~2 cm, which would be consistent with the data of Roeleveld et al. (33) and Scholle et al. (38), still some systematic variation of the MF had to be expected. Another possible explanation could be activity from other muscles than the biceps. Agonists of the biceps in elbow flexion are the brachialis and brachioradialis muscles. Although measurements were performed in a supinated hand position to minimize the contribution of the brachioradialis, this cannot be excluded. The proximal muscle-tendon transition of the brachioradialis is between the electrode grid and the reference electrode. The extinction of propagating action potentials at the muscle fiber end will generate far-field potentials (34) that are almost equal at all electrodes of the present montage. Synchrony is not restricted to MUs of the same muscle. MUs of agonistic muscles tend to fire in synchrony as well (30). Far-field potentials from brachioradialis MUs synchronized with other brachioradialis MUs and also with biceps MUs could therefore contribute to the strong and topographically uniform decrease of monopolar MF.

Synchronization produced a substantial increase of RMS for both the monopolar and the bipolar signal. Synchrony aligns the MUAPs, and they will add to produce high amplitudes. The increase in RMS that is found in fatigue has therefore been attributed not only to an increased firing rate and/or recruitment, but also to synchronization.

In the fatiguing contraction at 20% MVC, the change of MF depends on electrode position. With fatigue at high forces, the muscle fiber conduction velocity decreases and MF declines. When MU firings are syn-
Synchronization of motoneuron firing influences SEMG

In the present simulations, the firing rate between the 300 MNs of the pool could be varied. When the firing frequency of all MNs was set to the same value, there was a sharp peak in the spectrum at this frequency (Fig. 6J, black curve). As can be expected, this peak was even more prominent with synchronous firing (Fig. 6J, gray curve). With a large firing rate variation between MNs, the peak in the spectrum becomes smaller and broader, but the amount of variation did not influence the spatial distribution of MF over the simulated muscle. The dependency of MF on electrode position and synchrony remains. In a fatiguing contraction at moderate force the MU firing rate increases. To exclude the possibility that this influences the topographical distribution of MF, the model was run with an input of 10 NU and common inputs of 0 and 50%. When compared with an input of 6 NU, the increased firing rate had almost no influence on the MF and the change in MF induced by 50% common input. Taken together, changes in firing rate and firing rate distribution across MNs have no influence on MF.

Under physiological conditions, firing rate modulation and recruitment are linked. Because of the higher muscle fiber conduction velocity of high-threshold MUs, MF increases as force rises from low to moderate levels. Another change that could occur with fatigue is a derecruitment of fatigued MUs with simultaneous recruitment of fresh MUs (“alternation” or “rotation”). This possibility cannot be excluded for fatigue at 20% MVC. However, in the previous experiment on the trapezius muscle (19) the force was 50% MVC and a substantial role of recruitment is excluded when almost all MUs are active already from the beginning.

Fig. 8. MF of the SEMG generated by the model as a function of recording electrode position. An electrode position of 0 cm corresponds to the mean motor endplate position. The muscle fiber-tendon transition was at +5 and −5 cm. A–C: absolute values of MF for asynchronous firing (dotted), 25% (black), and 50% (gray) common input. B–D: change of MF, normalized to the condition of asynchronous firing. A and D: monopolar SEMG. B and E: bipolar SEMG with 1 cm IED. C and F: bipolar SEMG with 2 cm IED. Input to the 300 MNs of the pool was 5 ± 2 NU.
The same pattern was found for bipolar MF in the fatigued biceps muscle and in the trapezius muscle. These data suggest that in a fatiguing contraction the slower conduction decreases the MF at all electrode positions, whereas synchronization decreases MF dependent on electrode position. Both effects together can readily explain the experimental findings. With respect to electrode locations in bipolar kinesiological SEMG, it can be recommended from these results as well to place electrodes midway between endplate region and muscle-tendon transition (13, 14), where the influence of synchrony on the spectrum is strongest. In situations in which a separation between influences from muscle fiber conduction velocity and from synchrony is wanted, multichannel recordings are expected to give relevant clues.

In the present model, synchrony was induced by one common input signal to the whole MN pool, whereas the individual noisy input was unique to each MN. An assumption that seems more realistic would be the existence of many parallel input channels that send branches of their axons to a subgroup of MNs. Each MN then receives a weighted average of all input channels, the weighting factors representing the relative strength of the connections. Despite the simplified assumptions, the model presented here can predict the experimental data. The concept of one common input can be supported by experimental data. The corticospinal projection seems to be the major source of short-term synchrony, because in patients with a relatively selective lesion of the corticospinal tract after a stroke, short-term synchrony of MN firing is reduced or absent (7, 8). Recordings from corticospinal neurons in monkeys show that these neurons innervate a large part or the whole MN pool (29). A set of corticospinal neurons that all send projections to all MNs is well described by one channel of common input. Most studies of short-term synchrony recorded from hand muscles. A lower but still significant degree of synchrony was also found in the biceps brachii muscle (7). Probably, MN firings are already weakly correlated from the beginning and synchronize progressively as the corticospinal drive increases to compensate for peripheral fatigue. As to the presence of an individual input that is unique to a MN, one could think of spinal interneurons that project more focally (although not likely to only one MN). Their high number and different weighting would produce an input that does not correlate (or only weakly) between MNs.

Although the input to the pool of MNs by itself was always white random noise, i.e., did not prefer low frequencies, with short-term synchrony the power of SEMG increased at frequencies around the firing rate. When motor cortex activity is recorded simultaneously with either SEMG or single MU activity, significant corticomuscular coherence is found at specific frequencies, indicating that the inflow to the MN pool is modulated by cortical rhythms (2, 3, 24). The common drive behavior of MUs described by DeLuca et al. (4) appears to be another rhythmic input to the MN pool that is unrelated to short-term synchrony (40). In general, a change in corticomuscular coherence with the motor task is attributed to a changing descending motor command. The present study suggests that processes at both spinal and muscular levels might influence coherence spectra as well. Therefore, these coherence studies should carefully take electrode placement into account.

In conclusion, the model simulation of the SEMG interference pattern correctly predicted the experimental findings in the trapezius muscle and the bipolar SEMG of the biceps muscle. The effect of synchrony on the normalized change of MF was found to depend on electrode position and montage. The unexpected pattern of monopolar MF in the biceps muscle could be explained by activity from the co-contracting brachioradialis muscle. The results confirm and stress the importance of the influence of synchronization of MN firing on the SEMG signal during fatigue.

We thank Joleen Blok for help with the volume conduction model. We also thank Hans-Christoph Scholle and Nikolaus-Peter Schumann for comments on a draft of the manuscript and Marcie Matthews for linguistic correction.

This research was supported by Deutsche Forschungsgemeinschaft (DFG Innovationskolleg Bewegungssysteme, Projekt A2).

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