Possible mechanisms of muscle cramp from temporal and spatial surface EMG characteristics

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Roeleveld, K., B. G. M. Van Engelen, and D. F. Stegeman. Possible mechanisms of muscle cramp from temporal and spatial surface EMG characteristics. J Appl Physiol 88: 1698–1706, 2000.—In this study, the initiation and development of muscle cramp are investigated. For this, we used a 64-channel surface electromyogram (EMG) to study the triceps surae muscle during both cramp and maximal voluntary contraction (MVC) in four cramp-prone subjects and during cramp only in another four cramp-prone subjects. The results show that cramp presents itself as a contraction of a slowly moving fraction of muscle fibers, indicating that either the spatial arrangement of the motoneurons and muscle fibers is highly related or that cramp spreads at a level close to the muscle. Spectral analyses of the EMG and peak-triggered average potentials show the presence of extremely short potentials during cramp compared with during MVC. These results can also be interpreted in two ways. Either the motoneurons fire with enlarged synchronization during MVC compared with cramp, or smaller units than motor units are active, indicating that cramp is initiated close to or even at the muscle fiber level. Further research is needed to draw final conclusions.

MUSCLE CRAMP IS A SUDDEN, involuntary painful muscle contraction, accompanied by a palpable knotting of the muscle. Cramp is self-limiting within minutes and can often be relieved by passive stretch or massage of the muscle (16, 39). According to a survey of sport academy students, the prevalence of ordinary cramp can be as high as 95% (32). According to a more recent study, one-third of the Dutch adult population has at least one muscle cramp a year and 2% suffer from muscle cramp (18, 24, 29).

Although the origin of muscle cramp is generally considered to be of a neurogenic nature (e.g., Ref. 25), there are still two different views about its precise origin. The first theory proposes that cramp is the result of abnormal excitation of the terminal branches of motor axons (2, 7, 23), whereas the second theory states that cramp may result from some form of hyperexcitability or bistability of motoneurons (22, 32, 38). Each theory fails to explain all observations and to provide conclusive evidence. For example, the fact that cramp can be elicited by electrical stimulation of the peripheral nerve, even after proximal nerve block (2), is not in agreement with the motoneuron theory, whereas the observation of whole motor unit potentials (MUPs) during cramp (38) is in correspondence with that theory only. In addition, we think that even an origin at the level of the muscle fiber cannot be excluded. First, the supposed neurogenic origin is only based on observations that cramp may occur in individuals with no obvious pathophysiology and that cramp cannot be induced after the administration of curare (26). Second, single muscle fibers are capable of manifesting repetitive discharges independently (6). The normal insertional activity recorded during needle electromyogram (EMG) investigations (9) is another indication that muscle fibers under normal physiological conditions are not that far from presenting independent activity.

Ross and Thomas (38) concluded that cramp elicits whole motor unit activity. Such a result would imply an origination of cramp at the motoneuron level. They came to this conclusion from needle EMG recordings with electrodes having very small recording areas. These recordings did not show any differences between the shape of the action potentials during cramp and that during maximal voluntary contraction (MVC) (32, 38). Because of the small recording electrode used in these studies, it can be argued that potentials from single fibers or a very small number of single fibers were recorded (11). Therefore, we think that from these results it is difficult to conclude on a motor unit level, as a large part of the motor unit is not “seen.” Another methodological disadvantage of using a needle electrode for studying muscle cramp is the local and unpredictable character of the cramp (7, 23, 32, 38).

We aimed to study how cramp is distributed and how it spreads along the muscle and whether indeed whole motor units are active during cramp. A technique by which activation of superimposed patterns of whole motor units can be studied is surface EMG. During normal motor unit activation, like in voluntary contrac-
tion, the distant view of surface EMG guarantees a view of the whole motor unit (35, 36). Spectral analysis can be used to obtain information about the firing process and the shape of the average MUPs (17, 21, 33, 46). Changes in muscle fiber membrane properties are also known to show up in the surface EMG (47). Furthermore, the noninvasiveness of surface EMG allows a topographical view, which facilitates the analysis of that part of the muscle that is acting most strongly in a cramp, and allows the study of spread and propagation of the cramp. In the present study, 64-channel surface EMG obtained during muscle cramp is analyzed in time and frequency domain and compared with results obtained during MVC of the same muscle.

**METHODS**

Subjects. Eight healthy adult (age 47 ± 14 yr) cramp-prone subjects (3 men, 5 women) participated in this experiment after giving informed consent. The subjects were selected from a pool of 104 subjects who previously participated in a study concerning the efficacy of hydroquinine in preventing frequent muscle cramp (20). All selected subjects were able to voluntarily induce cramp in the triceps surae, considered themselves as healthy, did not suffer from a known neuromuscular disease at the moment of selection, and did not use quinine- or hydroquinine-containing products. Although we intended to compare the cramp characteristics with those of MVCs, only four of the selected subjects could generate an MVC without having a muscle cramp almost instantaneously. The Committee on Experiments in Humans of the Faculty of Medicine at the University of Nijmegen approved the experimental protocol.

Electrodes. Sixty-four Beckman biopotential electrodes (Sensormedics), with diameters of 7 mm, were situated as an eight-by-eight matrix in a square-shaped rubber sheet with center-to-center distances of 2 cm (both row- and column-wise). Before the electrodes were positioned, the skin surface was rubbed with Skinpure (Nihon Kohden) and cleaned with alcohol, and all electrodes were filled with electrode paste (Elefix; Nihon Kohden). The holder with the 64 surface electrodes was placed over the lower leg such that the triceps surae were completely covered with electrodes (see Fig. 1). Recordings were made in a unipolar montage. A common reference electrode was placed on the contralateral knee, and a ground electrode was placed on the ipsilateral ankle.

Data acquisition. The myoelectric signals from the skin surface were amplified (Neurotop, Nihon Kohden; input impedance of 100 MΩ) over a frequency range of 5–800 Hz with a gain of 1,000. The EMG signals and a stage marker were analog-to-digital converted (DT2839 and DT2896, Data Translation; 12 bits, maximal total sample frequency of 224 kHz) and continuously stored on the hard disk of a computer with a sample frequency of 2 kHz/channel.

Experimental protocol. After the surface electrode array was placed over the triceps surae of the left leg or over the leg that most frequently cramped for the subject, the EMG signals were inspected visually. The subjects were then asked to perform an MVC of at least 20 s, with feedback of delivered force. Each subject lay on their belly on a bench with knee bent at ~120 degrees, ankle positioned at 90 degrees, and foot placed in a force transducer. When a cramp appeared during the MVC measurement, the MVC measurement was repeated after ~10-min rest. After one to three MVC trials, the position of the subject’s leg was changed into a preferable position for generating muscle cramp with the knee bent at 160 degrees and the foot completely plantar flexed. In this position, no external force was delivered. The subject was then asked to generate a muscle cramp in the calf muscle and was instructed to report immediately when and where he or she felt a muscle cramp, to stop the voluntary contraction, and to let the cramp develop until it spontaneously disappeared. Subjective information from the subject on the time envelope of the muscle cramp (voluntary contraction, cramp, termination, disappearing cramp) was added to the EMG signals by the examiner using a stagemarker. Five to ten minutes after each trial of cramp generation (successful or nonsuccessful), a new trial was performed. The cramp trials were stopped after 1 h or sooner on indication of the subject. Thereafter, the leg was replaced into the original position, and the MVC trial was repeated.

Parameterization. Data processing was performed with software written in Matlab version 5.2. After visual inspection of the data, the root-mean-square (RMS) amplitude of all 64 EMG channels was calculated in blocks of 2.048 s (4,096 data points). Then, with the information from the stage marker, the average RMS amplitude during cramp or MVC was determined for the 64 channels. The percentage of electrodes having average values higher than 60% of the maximally averaged RMS in the recording was taken to represent the spatial size (S) of the cramp or MVC. A relative threshold was chosen so that even small-amplitude contractions had a final size of at least the domain of one electrode. The threshold value of 60% gives the largest variation in size parameter between the different contractions.

The site where the electrode showed the highest average RMS amplitude was considered to be the focus of the cramp or MVC. The power spectrum was calculated from the signals recorded by this electrode at two time segments of again 2.048 s (fast Fourier transform algorithm) 1) at the beginning of the voluntary contraction (V1) and at the onset of cramp (C1) and 2) at the time of maximum amplitude during MVC (V2) and at the time of maximum amplitude during cramp (C2). The 10th and 50th percentiles of the power spectrum distribution (P10 and P50) were calculated. P50 is also known as the median frequency and is mainly influenced by the shape of the MUPs (33). P10 is also affected by the firing characteristics (central nervous drive; Ref. 21).

The individual peaks in the EMG of voluntarily contracting healthy subjects can represent activity of a single active motor unit or the summed activity of more than one unit. For needle EMG recordings, it has beensettled that, even for high contraction levels, peak amplitudes are only marginally larger than the peak amplitudes of the largest MUPs (31). This indicates that signal averaging with peak occurrence as
the time trigger would provide an estimate of an average MUP. For the surface EMG, this procedure will most probably give such an estimate for superficial and/or large motor units.

The power spectrum of a voluntarily generated EMG arises as the frequency power distribution of the average MUP multiplied by the power spectrum of the firing process (17, 21, 33). Although a MUP estimate from peak averaging is theoretically biased, as will be discussed, it will provide an impression of the MUP and EMG spectrum with the exclusion of the firing process statistics. First, a trigger level was arbitrarily set to 60% of the fifth largest peak in the EMG. After the triggers were detected, an average MUP estimate was obtained by averaging around these triggers with a window of 50 ms (25 ms pre- and 25 ms posttrigger time), elevating the MUP estimate from the background surface EMG. To stress that these MUP estimates do not represent unbiased MUP estimates and during cramp may not even be a reflection of potentials from normal motor units, they are referred to as MUPs. Also, from these MUPs, the spectral parameters P10 and P50 were calculated with the same spectral resolution as the EMG signals. In addition, the peak-to-peak amplitudes and the RMS value of the MUPs were obtained.

Statistics. The data are presented as means ± SD. Statistical tests were applied with SPSS for windows software, release 6.1 (1989–1994). The data were analyzed twice. First, to use all obtained data, a one-way ANOVA model was used with subject and contraction type (cramp or MVC) as factors. Second, paired t-tests were used to compare MVC and cramp contractions of the four subjects (subjects 2, 4, 7, and 8) with both MVC and cramp recordings. In three (subjects 2, 4, and 7) of these four subjects, the cramp recording with the same leg position as the MVC recording was used for this purpose. The fourth subject (subject 8) had only one cramp contraction with the leg completely extended. For each of these four subjects, the MVC recording with the V2 value occurring closest to the cramp recording was chosen. The significance level was set at P < 0.05.

RESULTS

In total, 38 muscle cramp events and 8 MVCs were recorded in the triceps surae of cramp-prone subjects. Four subjects (subjects 1, 3, 5, and 6) were not able to perform an MVC test without developing a muscle cramp. For the development of a muscle cramp, maximal contraction was often not a prerequisite. A specific leg position could already trigger a cramp. Sometimes, the cramp or the MVC trial failed because of a muscle cramp in the foot, the hamstrings, or the contralateral leg.

Spatiotemporal development of cramp. Figure 2 shows an example of the development of a cramp in terms of a change in RMS amplitude distribution over time. First, the subject contracted the muscle voluntarily (5th s)
and then tried to relax the muscle, but a cramp developed in a small area (7th s). The cramp spread over a larger region, and its focus changed position (9th to 29th s) and then decreased in size and intensity until it disappeared (31st to 51st s). Such a rather slow change in position (a few centimeters per second) over the muscle was observed in ~50% of the cramps; in the other cramps, the position did not change. The direction of spreading was without preference. In Table 1, the individual and group averages of the muscle cramp parameters are shown together with the group averages of the MVCs. The spatial size parameter shows that, as in the example shown in Fig. 2, cramp often involves a part of the muscle that is smaller than that involved in a voluntary contraction. One-way ANOVA analysis of the whole data set and paired t-tests of the selected MVC and cramp contraction of four subjects showed that this difference in spatial size was significant. The subject’s sensation of the (shifting) location of the muscle cramp corresponded to sites with maximal EMG amplitudes.

Figure 3 shows recordings of six muscle cramps obtained in three different subjects (subjects 5 (row I), 6 (row II), and 3 (row III), each show two typical cramps) visualized in two ways. Figure 3, A and C, shows the amplitude of the focus of the muscle cramp as a function of time (a single channel as a function of time). Figure 3, B and D, shows the spatial distribution over the muscle of RMS at the moment of maximal cramp (all channels at a single time instant). Figure 3 shows that the location of the cramp differs considerably between subjects but also within subjects. Three of eight subjects did not have one muscle cramp covering the same region of the muscle as a previous one. The others had on average two cramps in the same region. When similarly localized muscle cramps were observed in the same subject, they were always immediately consecutive.

The change in time of EMG amplitude in the transition from MVC to muscle cramp within subjects appears more reproducible within than between subjects. In some subjects, the development of the muscle cramp was accompanied by a sudden increase in RMS amplitude to sometimes extremely high values (higher than during MVC) in a small part of the muscle (Fig. 3, rows I and II). Such high amplitudes slowly returned to a lower level or slowly changed position to another part of the muscle. The cramp of other subjects did not show an increase in RMS (Fig. 3, row III), which means that a rising cramp after the voluntary contraction hardly changes the EMG amplitude but induces a spatial reduction of the EMG activity to a part of the muscle.

Although the maximal RMS amplitude is (insignificantly) higher during MVC recordings, the highest RMS amplitudes were recorded during cramp (range of cramp = 139–1,091 µV, range of MVC = 410–774 µV). In subjects 1, 2, and 3, the duration of the muscle cramp often did not exceed 10 s. In subject 1, this occurred because the subject terminated the cramp because of extreme pain. In subjects 2 and 3, the cramp was short-term self-limiting.

Time and frequency analysis in focus of activity. Figure 4 shows four typical examples of one-channel unipolar EMG and its power spectrum and the average MUP with its power spectrum. Rows I and III in Fig. 4 show MVC recordings from subjects 4 and 2, and rows II and IV show cramp recordings from the same subjects. In Fig. 4A, it is obvious that the prominent peaks have smaller durations and appear more frequently during the cramp than during MVC. When the EMG power spectra of cramp and MVC were compared, cramp was shown to have less power at low frequencies and more power at high frequencies. The differences were quantified with the parameters P10 and P50 (Table 2).

One-way ANOVA analysis of the whole data set showed that the P10 and P50 during the beginning and the maximum of the MVC (V1 and V2) were significantly lower than those during C1 and C2. Paired t-tests of four subjects with MVC and cramp contractions showed similar results. The P50 during V1 and V2 is significantly lower than during C1 and C2, respectively (95% confidence interval of the difference between the P50 during V1 and C1 is between −61 and −3 Hz; the 95% confidence interval of the difference between the P50 during V2 and C2 is between −36 and −10 Hz). The P10 during V1 and V2 was not significantly lower than during C1 and C2, respectively (95% confidence interval of the difference between the P10 during V1 and C1 is between −42 and +3 Hz; the 95% confidence interval of the difference between the P10 during V2 and C2 is between −38 and +6 Hz).

Average MUPs were determined to estimate the spectral content of the EMG after exclusion of the firing statistics. The substantially shorter duration of a cramp MUP compared with an MVC MUP in the same subject (e.g., Fig. 4C, row III, vs. Fig. 4C, row IV) is evident. The differences are also summarized for the population in Tables 2 and 3. Only the P50 of the cramp MUPS differed significantly from the corresponding value in the raw EMG (95% confidence interval of the differences of C1 (EMG − MUP) = between +3 and +12 Hz, of C2 (EMG − MUP) = between +4 and +13 Hz).

Table 1. Individual muscle cramp characteristics and group averages of cramp and MVC

<table>
<thead>
<tr>
<th>Subject</th>
<th>Number of Recordings</th>
<th>Spatial Size, %</th>
<th>Duration, s</th>
<th>RMS, µV</th>
<th>P50, Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4</td>
<td>41 ± 30</td>
<td>11 ± 9</td>
<td>447 ± 91</td>
<td>103 ± 31</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>50 ± 33</td>
<td>10 ± 4</td>
<td>640 ± 23</td>
<td>96 ± 27</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>73 ± 33</td>
<td>9 ± 8</td>
<td>380 ± 127</td>
<td>108 ± 31</td>
</tr>
<tr>
<td>4</td>
<td>7</td>
<td>52 ± 19</td>
<td>31 ± 17</td>
<td>494 ± 63</td>
<td>112 ± 13</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>44 ± 38</td>
<td>52 ± 25</td>
<td>463 ± 182</td>
<td>106 ± 18</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>23 ± 17</td>
<td>53 ± 18</td>
<td>783 ± 172</td>
<td>116 ± 22</td>
</tr>
<tr>
<td>7</td>
<td>4</td>
<td>26 ± 41</td>
<td>49 ± 23</td>
<td>473 ± 252</td>
<td>97 ± 23</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>41 ± 20</td>
<td>27 ± 22</td>
<td>506 ± 223</td>
<td>103 ± 19*</td>
</tr>
<tr>
<td>Mean cramp</td>
<td>38</td>
<td>48 ± 33*</td>
<td>27 ± 22</td>
<td>506 ± 223</td>
<td>103 ± 19*</td>
</tr>
<tr>
<td>Mean MVC</td>
<td>8</td>
<td>81 ± 21</td>
<td>31 ± 30</td>
<td>551 ± 240</td>
<td>71 ± 20</td>
</tr>
</tbody>
</table>

Values are means ± SD. Mean maximal voluntary contraction (MVC) values were from 4 subjects (subjects 2, 4, 7, and 8). Root-mean-square (RMS) and 50th percentile of power spectrum distribution (P50) values were obtained during maximal cramp (C2) and maximal voluntary contraction (V2). *ANOVA analysis on the whole data set and paired t-tests on the selected MVC and cramp contraction of 4 subjects showed significant differences between the cramp and MVC parameters.
Similar to the spectral EMG differences, one-way ANOVA analysis of the MUP parameters showed that P10 and P50 were significantly lower during V1 and V2 than during C1 and C2, respectively. Paired t-tests on four subjects with MVC and cramp contractions showed similar results. The P50 during V1 and V2 was significantly lower than during C1 and C2, respectively (95% confidence interval of the difference between the P50 during V1 and C1 is between -2.51 and -1.4 Hz; the 95% confidence interval of the difference between the P50 during V2 and C2 is between -2.35 and -2.3 Hz). The P10 during V1 and V2 was not significantly lower than during C1 and C2, respectively (95% confidence interval of the difference between the P10 during V1 and C1 is between -2.43 and -1.1 Hz; the 95% confidence interval of the difference between the P10 during V2 and C2 is between -2.38 and -1.6 Hz).

The peak-to-peak and RMS amplitudes are shown in Table 3. Neither the ANOVA analysis on the whole data set nor the t-tests showed any significant difference between the amplitudes recorded during cramp and MVC.

Because the leg position was different in most MVC and cramp trials, a systematic effect caused by differences in muscle fiber length and muscle fiber orientation could have appeared that might show up in the EMG results (8, 30). In five subjects, cramp was induced and recorded during some MVC trials as well as during some cramp trials. Paired t-tests on the average values of these five subjects did not reveal any significant effect of the leg position on any of the parameters investigated. For example, the 95% confidence intervals of the P10 and P50 values obtained from the interference EMG at maximal RMS recorded during cramp in the MVC trials minus the ones recorded during the cramp trials are between -8.3 and 3.4 Hz and between -7.5 and 2.8 Hz, respectively. Thus the difference in leg position between the MVC and cramp contractions did not cause the higher spectral values of the EMG during cramp.

To ensure that the cramp-prone subjects had normal EMG during MVC, two MVC measurements were obtained in two healthy subjects without muscle cramp. EMG spectra and MUP spectra of the MVC recordings

Fig. 3. EMG representation of 2 muscle cramps is visualized in two ways for 3 different subjects (rows I, II, and III). A and C: RMS value at the focus of the muscle cramp as a function of time. RMS values are obtained in blocks of 2 s. At time 0, the subjects felt a muscle cramp. B and D: RMS distribution over the skin surface at maximal cramp in time and space. Intensity of the gray scale corresponds to RMS values (ranges are plotted above each graph). Lightest blocks represent the electrodes with RMS values lower than 60% of the maximum (the complement of spatial size). dist, Distal; prox, proximal; med, medial; lat, lateral.
of the control subjects did not differ in any aspect from the EMG and MUP spectra of cramp-prone subjects during MVC.

**DISCUSSION**

Thirty-eight muscle cramps in the triceps surae of eight healthy subjects were compared with eight MVCs of the same muscle in four of these subjects. The results showed that the location of cramp in the muscle can vary considerably between and within subjects. Cramp can start at several places simultaneously and can spread out slowly over the muscle. Compared with the surface EMG measured during MVC, the power spectrum of the EMG in the cramp regions contained less low frequencies and more high frequencies. This was observed at the onset of cramp vs. submaximal voluntary contraction as well as at full cramp compared with MVC. In our view, the results provide evidence against normal motor unit activity during cramp and therefore against the spinal origin of cramp. In addition, our results are well in accordance with the concept that the action potentials during cramp are generated at the level of the terminal branches of motor axons or at the level of the muscle fibers themselves. This viewpoint will be elucidated below.

**Table 2.** P10 and P50 values at the focus of 2 different stages of cramp (initial C1 and full C2) and MVC (initial V1 and full V2)

<table>
<thead>
<tr>
<th></th>
<th>P10 EMG, Hz</th>
<th>P50 EMG, Hz</th>
<th>P10 MUP, Hz</th>
<th>P50 MUP, Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1</td>
<td>47 ± 12</td>
<td>113 ± 20</td>
<td>50 ± 15</td>
<td>106 ± 31</td>
</tr>
<tr>
<td>C2</td>
<td>46 ± 10</td>
<td>103 ± 20</td>
<td>48 ± 11</td>
<td>95 ± 29</td>
</tr>
<tr>
<td>V1</td>
<td>32 ± 5*</td>
<td>86 ± 20†</td>
<td>34 ± 6*</td>
<td>86 ± 30*</td>
</tr>
<tr>
<td>V2</td>
<td>26 ± 5*</td>
<td>71 ± 20†</td>
<td>28 ± 4*</td>
<td>66 ± 25†</td>
</tr>
</tbody>
</table>

Values are means ± SD. P10 and P50 values were obtained from continuous electromyogram (EMG) data and from an estimate of the average motor unit potential (MUP). *ANOVA showed that P10 and P50 of C1 and C2 differed significantly from those of V1 and V2, respectively. †ANOVA analysis on the whole data set (as for *) but also paired t-tests on the selected MVC and cramp contraction of 4 subjects showed significant differences between the cramp and MVC parameters.

**Table 3.** MUP RMS and PP values for the 2 different stages of cramp (C1 and C2) and MVC (V1 and V2)

<table>
<thead>
<tr>
<th></th>
<th>MUP RMS, µV</th>
<th>MUP PP, mV</th>
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<tbody>
<tr>
<td>C1</td>
<td>203 ± 132</td>
<td>1.31 ± 0.84</td>
</tr>
<tr>
<td>C2</td>
<td>248 ± 119</td>
<td>1.55 ± 0.76</td>
</tr>
<tr>
<td>V1</td>
<td>214 ± 76</td>
<td>1.28 ± 0.44</td>
</tr>
<tr>
<td>V2</td>
<td>305 ± 114</td>
<td>1.77 ± 0.56</td>
</tr>
</tbody>
</table>

Values are means ± SD. PP, peak to peak.
Spatiotemporal development of cramp. The amplitude of the EMG signals, expressed in the RMS value, was distributed more equally over the muscle during MVC than during cramp (Fig. 2). During MVC, the complete muscle group is active, whereas often only a part of the muscle is involved in cramp. This local character of muscle cramp was previously reported (7, 23, 32, 38), but the 64-channel recordings allowed a precise study of the distribution and especially the propagation of many cramp episodes. In an individual subject, a cramp could occur in any of the outer boundaries or centrally in the muscle. Sometimes the cramp was even multifocal (Figs. 2 and 3). The cramp could slowly change its region, but it never jumped from one part of the muscle to another. Apparently, a cramp spreads through activation of neighboring (groups of) muscle fibers. Consequently, the hypothesis of the spinal origin of muscle cramp can only hold when the motoneurons that innervate neighboring muscle fiber populations are also neighbors. It is known that the most distal muscles are supplied by motor nuclei located in the dorsolateral part of the ventral horn, whereas more proximal muscles are innervated by motor nuclei in the ventrolateral part of the ventral horn, with a more or less continuous change in between (somatotopic spinal motor unit pools; Refs. 37, 48, 50). This indicates that a strong topographical correlation between motoneurons and the motor unit muscle fibers cannot be completely excluded. Nevertheless, a generation in the closely packed motoneurons in longitudinal and cross-sectional level and the large innervation ratio of motor units in the calf muscles are at odds with the focal character of cramps, often limited to a small part of one muscle.

Time and frequency analysis in focus of activity. The EMG power spectrum can be considered as the spectrum of the average MUP multiplied by the spectrum of the average process of central innervation (21, 33). The power spectrum of a regular and stationary process of motoneuron firing displays almost no power up to frequencies equal to the average firing rate, with a large peak at this average firing rate and smaller peaks at the first and higher harmonics of this frequency. The steep peaks flatten with increasing firing rate irregularity (12, 46). A possible explanation for part of the higher frequency content of the cramp EMG could thus be higher firing rates of the contributing motor units. Ross and Thomas (38) reported higher firing rates in the first 20 s of cramp (~23 pulses per second (pps)) with incidentally very high firing rates (up to 80 pps) compared with those recorded during MVC (~12 pps). However, such a difference of, on average, 10 pps cannot cause the 20-Hz difference in P10 and the 30-Hz difference in P50 between the EMG during MVC and cramp, especially not since a change in firing rate or firing stationarity mainly affects the low-frequency region (P10) and hardly affects the median frequency (P50; Ref. 12). Moreover, during cramp, the firings are supposed to be less stationary and more irregular than during MVC (38), which can even slightly reduce the median frequency (12). In addition, the spectrum of the average MUP recorded during cramp and MVC changed similarly to the frequency contents of the EMG data. Therefore, the spectral difference must mainly be attributed to a difference between the active building blocks of the signal, being action potentials with short vs. longer duration.

The average MUP was obtained by peak-triggered averaging. It is possible that the peaks were not caused by single motor units but by two or more approximately simultaneously active ones. Nandedkar et al. (31) showed in a simulation study that for needle EMG such combined action potentials are statistically improbable. Furthermore, such combined action potentials would bias the MUP estimate and generate peaks with a longer duration than single MUPs. Then, the average MUP during MVC should have been the result of more summated single MUPs than the average MUP during cramp. Although the spatial size parameter indicates that the total number of active units is larger during MVC than during cramp, similar RMS at a specific electrode indicates that the volume of active tissue as observed by a single electrode was similar during the two contraction types. In addition, there was no difference in the peak-to-peak amplitudes of the average MUPs. Therefore, the fact that the RMS and peak-to-peak amplitudes do not differ between cramp and MVC, in combination with the shorter but equally sized MUPs and the increased firing rate during cramp, indicates a higher number of total generated MUPs during cramp and thus a higher chance of summation for cramp instead. In short, we cannot think of a way to get shorter average MUPs with similar peak-to-peak amplitudes when the underlying “real” MUPs are not shorter in cramp.

The shape of a MUP and its spectrum are determined by the shape and/or spectrum of the single-fiber action potentials and the temporal dispersion between them (27) and, therefore, by the propagation velocity of the action potentials, by the organization of the innervation zone, and by the number of fibers in the motor unit (10, 27, 40, 43). In principle, surface EMG recordings can be used to estimate the propagation velocity (e.g., Refs. 1 and 28). Unfortunately, the architecture of the medial gastrocnemius or, more specifically, the length of its muscle fibers and the angle between these fibers and the skin surface prevent the direct detection of the propagation velocity from the present data. The possible causes of short MUPs and their implications for the origination of muscle cramp are discussed below.

Both an increased propagation velocity and a decreased number of fibers in the motor unit could have caused the relatively short MUPs during cramp. Generally, small motor units (few fibers) have slow-type muscle fibers (low propagation velocity) and large motor units have fast-type muscle fibers. Because the MUP shape is more dependent on the propagation velocity than on the size of the motor units, large, fast motor units have shorter MUPs than small, slow motor units (51, 14). Therefore, if we consider that whole motor units are active during cramp and that during MVC both type 1 and type 2 motor units are active, the
increased median frequency by 30 Hz can only be explained by a selective or exclusive recruitment of fast motor units during cramp or by a sudden change in muscle fiber membrane properties. Because fast muscle fibers fatigue rapidly, this hypothesis also provides an explanation for the self-limiting character of cramp. Furthermore, if we consider an origination of cramp at the level of nerve termination or muscle fibers, this fits with the major involvement of fast-type motoneurons having larger axons and thicker muscle fibers with a lower excitation threshold. Interestingly, there seems to be a type 2 fiber predominance in subjects with muscle cramp (45). One observation is not in agreement with this hypothesis: visual inspection of the shape of single-fiber action potentials during cramp and during MVC, as measured by Norris et al. (32) and by Ross and Thomas (38), showed no obvious differences, whereas it is known that single-fiber action potentials with a high propagation velocity are of shorter duration and larger amplitude (34, 49) than those with a lower propagation velocity.

The above leads to the discussion of whether MUPs with less than normal numbers of single-fiber potentials with less temporal variation are active during cramp. A high degree of motor unit synchronization during voluntary contraction and a more desynchronized motor unit activity during cramp would be an explanation. This seems to be unlikely because, first, motor units are supposed to synchronize with relevant influence on the EMG characteristics only with high contraction levels (5), whereas we observed similar differences between the voluntary and involuntary contraction at low (C1, V1) and maximal (C2, V2) contraction levels. Second, the significant but rather low level of synchronization between pairs of motor units in terms of extra firings within a certain time frame (e.g., Refs. 3 and 13) also indicates that synchronization will not play an important role in the normal surface EMG characteristics of unfatigued muscle.

Excluding synchronization as an option, a submotor unit or single-fiber level of organization of cramp is left as being in complete and elegant agreement with our data and with observations of others made during cramp. First, such a level of origination is basically not compatible with a spinal origin. Although in healthy muscles microstimulation of one of the axon’s branches causes a response of all fibers in the motor unit through dromic and antidromic spread to all nerve terminals (15, 44), periodic bursts of repetitive potentials recorded in several neuromuscular disorders have been ascribed to fractional activation of a motor unit (4). A terminal nerve branch origin can explain both the spreading through neighboring (groups of) muscle fibers and shorter than normal MUP shapes and thus should be considered. Only in muscles with chronic denervation have such high-frequency discharges of a fraction of a motor unit been proven to exist (42). Such activation would involve hyperexcitable twigs involved in collateral innervation, possibly presenting an unstable polarization and could be secondary to terminal multifocal conduction blocks. Although the subjects used in our study didn’t suffer from a neuromuscular disease and were considered healthy, a muscle in cramp can certainly not be considered as being in a normal situation. Therefore, the above-depicted repeated activation of fractions of motor units is a plausible cause of muscle cramp. An alternative explanation on the basis of our data is the muscle activation at a single fiber level, thus by an ephaptic connection between neighboring muscle fibers. Such a peripheral cause of cramp would also provide a reason why cramps are more easily generated in a shortened position, since an increased sodium concentration around the endplates in shortened muscle fibers increases the rise of action potentials and reduces the depolarization needed to reach the threshold for action potential generation (41). The MUPs at cramp can very well be interpreted as interference single-fiber activity. More precise analysis, for instance by combining our topographical technique with in-dwelling electrodes, should shed light on the origin of muscle cramp.

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