Recurrence quantification analysis of surface electromyographic signal: Sensitivity to potentiation and neuromuscular fatigue

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1. Introduction

Nonlinearity is the hallmark of complex dynamical systems (Elbert et al., 1994). The assessment of nonlinear dynamics in physiological signals may be an important key for quantifying and eventually understanding systems evolving in time (Webber and Zbilut, 1994; Goldberger et al., 2002). More specifically, it has been shown that the electromyographic (EMG) signal displays nonlinear features (Filligoi and Felici, 1999; Ikegawa et al., 2000; Lei et al., 2001). Nonlinear techniques have gained attention in this domain (Felici et al., 2001). Unfortunately, to our knowledge, little attention has been given to identifying the physiological factors implicated in the muscle fatigue to which RQA is sensitive.

This study aimed to assess the capacity of recurrence quantification analysis (RQA) to detect potentiation and to determine the fatigue components to which RQA is sensitive. Fifteen men were divided into two groups [8 endurance-trained athletes (END) and 7 power-trained athletes (POW)]. They performed a 10-min intermittent (5 s contraction, 5 s rest) knee extension exercise at 50% of their maximal voluntary isometric contraction. Muscular fatigue and potentiation were evaluated with neurostimulation technique. Mechanical (peak torque, Pt) and electrophysiological (M-wave) responses following electrical stimulation of the femoral nerve were measured at rest and every 10 s throughout exercise. Vastus lateralis muscle activity (root mean square, RMS) was recorded during each contraction, and RMS was normalized to M-wave area (RMS/M). During contraction, muscle activity was analyzed with RQA to obtain the percentage of determinism (%Det). At the beginning of exercise, a significant Pt increase (+52%, P < 0.001) was observed in both groups, indicating potentiation. At this time, %Det remained constant in both groups, indicating that RQA did not detect potentiation. Thereafter, Pt decreased in POW from 5 min 30 s of exercise (−30%, P < 0.001), reflecting impairment in excitation–contraction coupling, and %Det increased from 3 min 30 s (P < 0.01). In END, Pt remained high and %Det was unchanged. These two results indicated that RQA detected the peripheral component of fatigue. Conversely, RQA did not detect central adaptation to fatigue since %Det remained constant when a significant increase in RMS/M (P < 0.01) appeared in END.
Given the capacity of RQA to detect early muscle changes (Webber et al., 1995), it is relevant to raise the question of its capacity to detect potentiation during repetitive muscle contractions.

During submaximal muscle exercise, potentiation may occur concurrently with fatigue. The most common definition of fatigue is an exercise-induced reduction in the ability of the muscle to produce force or power, whether or not the task can be sustained (Barry and Enoka, 2006). The development of fatigue involves several components that can interact: motor command, axonal conduction, sarcolemma excitability and, more generally, modulations of spinal and muscle properties. The fatigue model classically used by many authors dissociates central and peripheral factors, which are defined as being on either side of the neuromuscular junction (Bigland-Ritchie et al., 1986; Fitts, 1994; Gandevia, 2001; Taylor and Gandevia, 2008). To our knowledge, no studies have sought to detect fatigue components during exercise with a dedicated sensitive EMG analysis method, such as RQA.

Therefore, the main aim of this study was to assess the capacity of RQA to detect potentiation and fatigue. Our hypothesis was that RQA would detect any muscle state changes, especially potentiation and fatigue, during repeated submaximal contractions. As a secondary outcome, we sought to determine the fatigue components to which RQA was sensitive, given that neuromuscular fatigue is a plurifactorial phenomenon.

2. Methods

2.1. Subjects

Two groups of subjects volunteered to participate in the study: a group of 8 endurance-trained men (END: distance runners and triathletes, age 26.9 ± 1.2 years) and a group of 7 power-trained men (POW: weightlifters and rugby union players with back and forward positions, which require explosive force qualities, age 23.7 ± 1.2 years). All subjects had been training regularly for more than 5 years and participated in competitions at the national or international level. The study procedures complied with the Declaration of Helsinki on human experimentation and were approved by the local Ethics Committee. Each subject gave written informed consent before beginning the tests.

The choice of these two groups was based on the work of Hamada et al. (2003), who showed that the time courses of potentiation and fatigue clearly differ according to training background. Consequently, it was assumed that this difference would be an advantage in tests of RQA sensitivity toward potentiation and fatigue manifestations.

2.2. Experimental protocol

The subjects visited the laboratory on two occasions (familiarization session and testing session) separated by 24 h. In the preliminary session, a full explanation of the experimental protocol and recommendations were given to the subjects. They were familiarized with the neurostimulation procedures and practiced performing three 5-s maximal voluntary contractions (MVC) of the knee extensor muscles, with a 1-min rest interval between each trial (the highest was considered as the MVC). The optimal neurostimulation intensities were determined and individually appropriate adjustments to the knee-extension device were made.

In the testing session, subjects performed a 5-min standardized warm-up at 2 W/kg on an ergocycle followed by a 10-min repeated submaximal exercise at 50% of MVC of the knee extensors (with a duty cycle of 5 s of contraction and 5 s of rest). During the exercise, single twitches were evoked before every submaximal contraction. These twitches were used to draw the time courses of potentiation and fatigue. Force feedback displayed on a screen allowed the subjects to control the force level. Strong verbal encouragement was given to all subjects during the voluntary contractions.

2.3. Material and data collection

2.3.1. Apparatus

Subjects were seated on a homemade padded bench with the strongest leg (right leg in all instances) fixed by several semi-rigid Velcro bands just above the malleolus and behind a load-cell transducer (DEC 60, Captels, France) fixed to the frame of a rigid chair. In order to evaluate only the knee extensor muscles, subjects were firmly belted in place around the waist, the arms were crossed on the chest, and the chest was maintained against the back of the device by an adjustable Velcro strap. The back formed a 90° angle with the seat. The knee angle was set at 80° compared with the complete extension (0°).

2.3.2. EMG activity

Surface EMG from the muscle belly of the right vastus lateralis (VL) was recorded by means of bipolar Ag/AgCl electrodes (Contrôle Graphique Medical, Brie-Comte-Robert, France) with a diameter of 9 mm and an inter-electrode distance of 20 mm. Low impedance between the two bipolar electrodes (<5 kΩ) was obtained by shaving, abrading and washing the skin with emery paper and cleaning with 70% alcohol. The optimal electrodes position was chosen when the highest amplitude of a single compound muscle action potential (M-wave) was observed. The estimated position was then marked with indelible ink to ensure that the electrodes would be placed in exactly the same position in both familiarization and testing sessions. The reference electrode was placed on the kneecap of the opposite lower limb. EMG activity was recorded continuously during exercise via a dedicated acquisition system (MP30, Biopac Systems, Inc., Santa Barbara, CA, USA). The EMG signal was amplified (1000×), band-pass filtered (30–500 Hz) and sampled at 2000 Hz. The EMG signal amplitude was quantified by the calculation of the root mean square (RMS) over a 1-s median period during each 5-s contraction of the exercise.

2.3.3. Neuromuscular function tests

Percutaneous stimuli were delivered to the femoral nerve with a monopolar cathode in the inguinal crease. The anode consisted of an adhesive rectangular electrode of 50 cm² (MédecimpeX S.A., Ecublens, Switzerland) located in the gluteal fold opposite the cathode. Square wave pulses of 200–μs at a maximal voltage of 400 V were delivered by a stimulator (Digitimer, Model DS7AH, Hertfordshire, UK).

During the familiarization session, the optimal intensity of stimulation was determined for each subject by progressively increasing the intensity (10-mA increments) until the M-wave and the corresponding torque reached a plateau. During the testing session, the stimulation intensity applied to the femoral nerve was set at 10% above the optimal intensity in order to ensure that a supramaximal stimulation intensity would be delivered (Martin et al., 2004). The intensity ranged between 80 and 130 mA, depending on the subject. From the mechanical response obtained, the peak torque (Pt) was measured. From the neurophysiological response obtained (M-wave max), the M-wave area was measured and the VL RMS activity was normalized by this one (RMS/M) in order to avoid errors of interpretation [for a review see Millet and Lepers (2004)].

2.4. Recurrence quantification analysis

RQA, as a time series analysis tool, is able to detect the deterministic structure of the underlying dynamical process of a time
series. RQA, like many nonlinear methods [see for instance Kantz and Schreiber (1997)], is based on the time delay embedding (TDE) procedure for phase space reconstruction (PSR). The phase space is the abstract geometrical space into which the state of the underlying system is represented by trajectories evolving over the course of time. The PSR and TDE techniques were numerically demonstrated by Packard et al. (1980) and mathematically proved by Takens (1981). In order to understand the technical aspect of the RQA procedure, let us consider a measured (with some sampling frequency $f_s$) time series of $N$ values:

$$x_1, x_2, x_3, \ldots, x_N$$

The first step of the procedure is to construct the time delayed vectors $y_i(d)$ by simply cutting the original time series into vectors:

$$y_i(d) = (x_i, x_{i+\tau}, x_{i+2\tau}, \ldots, x_{i+(d-1)\tau})$$

These $d$-dimensional vectors are dependent on the choice of the time delay $\tau$. A classical approach used to estimate the optimal value of $\tau$ is the average mutual information (AMI) based method (Fraser and Swinney, 1986). The main advantage of this method is that it takes into account the possible nonlinear interrelations of the time series. In this approach, the optimal $\tau$ (which minimizes the temporal correlations between the samples of the time series) is simply given by the first local minimum of the AMI function. In our study, we set $\tau = 5$, as this value was obtained for the major part of the experimental recorded time series.

The next input parameter of RQA is the so-called embedding dimension $d$. To determine this parameter, we used the false nearest neighbors method proposed by Kennel et al. (1992). For our data, we set $d = 15$. This value is consistent with those of other studies applying RQA to EMG signals (Webber et al., 1995; Fillipović and Felici, 1999; Ikegawa et al., 2000; Farina et al., 2002).

The next step of RQA is the construction of the recurrence plot (RP) introduced by Eckmann et al. (1987). The RP is basically a two-dimensional plot obtained by computing all distances between the delayed vectors $y_i(d)$ and $y_j(d)$ estimated in a normalized matrix according to the maximum distance. Then, after defining a threshold radius $\epsilon$, one can construct a binary plot (the RP), into which the point $(i, j)$ is represented by a “black” dot if the distance between $y_i(d)$ and $y_j(d)$ is smaller than $\epsilon$. In this case, the point is a recurrent one. Otherwise, the point $(i, j)$ is assigned a “white” dot.

The RQA method allows the extraction of a great deal of quantitative information from the RP [see Marwan et al. (2007) for a detailed overview on the subject]. The first quantifications were proposed by Zbilut and Webber (1992). The percentage of recurrence $\%Rec$ quantifies the density of recurrent points in the RP (Webber and Zbilut, 1994). The second important measure considers the diagonal lines and quantifies the ratio of the recurrence points forming diagonals of length $l_{\text{min}}$ to all recurrence points. This is called the percentage of determinism ($\%Det$). Uncorrelated random time series produce no diagonals or very short ones. If the underlying system is deterministic, the diagonals are much longer and there are few isolated recurrence points. $\%Det$ is more related to the predictability of the system (Thiel et al., 2004; Marwan et al., 2007).

In our study, the radius was fixed at 25% of the maximum distance so that $\%Rec$ would not be too high (<10%). This strategy is generally recommended in the literature (Riley et al., 1999). The $l_{\text{min}}$ was set to 3 points.

From a more experimental point of view, during each contraction of 5 s, we analyzed the 0.5-s central period of the EMG signal of VL (1000 samples). For each epoch, $\%Rec$ and $\%Det$ were computed. To perform RQA, we used the Cross Recurrence Plot Toolbox 5.13 (R25) developed by Marwan et al. (2002).

### 2.5. Shuffled surrogates tests

A common approach used for the validation of the results of a nonlinear analysis such as RQA is the shuffled surrogates test (Riley et al., 1999; Marwan et al., 2007). Basically, such a procedure is used to test the null hypothesis that the data are produced by a purely random uncorrelated process. The first step is to shuffle the time ordering of the recorded data. The obtained time series have the same statistical parameters as the original data but the temporal correlations are destroyed. In our study, for each EMG recorded time series, a number of such surrogates were generated. Then, the $\%Det$ of the shuffled surrogates were computed (with $d = 15$, $\tau = 5$, $l_{\text{min}} = 3$ and radius $= 35\%$ of the maximum distance such that $\%Rec$ of the shuffled surrogates were equivalent to those of the original data) and compared with $\%Det$ of the original recorded time series using a one-tailed rank-order test. To obtain a level of significance $\alpha < 0.05$, we generated $(1/\alpha) – 1 = 19$ surrogates (Kantz and Schreiber, 1997). The expected result was a systematic and dramatic drop in the $\%Det$ of the shuffled surrogates.

### 2.6. Statistical analysis

Data are presented as means ± SE. During the exercise, Pt and RMS/M were normalized to the first evoked or voluntary contraction. The values of the measured parameters (Pt, RMS/M, $\%Det$ and $\%Rec$) were averaged on three successive contractions (30-s intermittent exercise duration). The homogeneity of the variances was checked with Hartley, Cochran, Bartlett and Levene tests, which were never significant. Thus, Pt, RMS/M, $\%Det$ and $\%Rec$ were analyzed using a two-way ANOVA: a between-subject factor (group of subjects: END vs. POW) and a within-subject factor (time: one contraction at rest and 20 repetitions of 3 contractions). When the ANOVA $F$ ratio was significant, Fisher LSD post hoc tests were carried out to compare means for statistical differences. The normal distribution of the errors was checked using the normal probability plot to confirm the validity of analysis.

Correlations between variables were calculated with the Pearson correlation coefficient.

The statistical level of significance was always set at 0.05.

### 3. Results

#### 3.1. Neuromuscular function tests

As indicated in Fig. 1A, the Pt time courses differed between END and POW [$F_{20,260} = 20.3, P < 0.001$]. For 1 min of exercise, the two groups potentiated in the same way: Pt was $+52\%$ and $+53\%$ ($P < 0.001$) for END and POW, respectively. Thereafter, Pt in END was still increasing at 1 min 30 s ($+56\%$) and remained high throughout the entire exercise duration, whereas Pt in POW decreased over time (from $+48\%$ at 1 min 30 s to $−30\%$ at the end of exercise) and was significantly below the baseline value starting from 5 min 30 s ($P < 0.001$).

The VL RMS/M ratio represented in Fig. 1B increased markedly in POW from 3 min of exercise until the end of exercise ($+32$ and $+49\%$, $P < 0.001$). This ratio increased less and later in END from 7 min (between $+7$ and $+16\%$, $P < 0.001$).

#### 3.2. Shuffled surrogates tests

With our EMG data, the values of $\%Det$ obtained for the shuffled surrogates fell close to 0%. The average $\%Rec$ over all surrogates was...
Fig. 1. Evolution of the mean values (±SE) during the 10-min exercise for the two groups of subjects. ***P< 0.001, **P< 0.01, significantly different from the first value.
(A) Peak torque (Pt), (B) VL RMS/M ratio, 100% corresponds to the first value of the exercise and (C) percentage of determinism (%Det).

3.79 ± 0.4% in END and 4.46 ± 0.55% in POW. %Det was 0.68 ± 0.21% in END and 0.89 ± 0.29% in POW. Indeed, random shuffling destroyed all temporal structures revealed by the %Det parameter of the original recorded signals. Fig. 2 shows the RPs obtained from a recorded sequence and from one of its shuffled surrogates.

3.3. Recurrence quantification analysis

In Fig. 3, four recurrence plots obtained during the first and last contractions of the exercise in END and POW are shown. %Rec remained constant throughout exercise in both END (2.86 ± 0.31%) and POW (3.27 ± 0.33%). The evolution of %Det is reported in Fig. 1C. The %Det time courses differed between END and POW [F(20,260) = 2.02, P< 0.01]. %Det increased significantly in POW from 3 min 30 s of exercise to the end of exercise, whereas it remained unchanged in END (P between 0.13 and 0.98). The mean values of %Det were 80.54 ± 2.55% and 87.68 ± 2.64% during the first and last contractions of exercise, in POW, and 81.78 ± 4.28% and 83.92 ± 2.79% in END, respectively.
3.4. Correlation between %Det and neuromuscular variables

In the POW group, a negative correlation coefficient between \( \text{Pt} \) and %Det (Fig. 4) was most often significant from the fourth minute of exercise (\( r \) from \(-0.77\) to \(-0.94\); \( P < 0.04 - 0.001 \), respectively). In contrast, the correlation in END was never significant (\( r \) from \(0.08\) to \(0.36\)). The correlations between other variables remained inconsistent.

4. Discussion

The aim of this study was to assess the capacity of RQA to detect potentiation and to determine the fatigue components to which RQA is sensitive during a repeated submaximal isometric exercise. Our results showed that %Det (i) was unchanged during the increase in \( \text{Pt} \) in either group; (ii) increased only when \( \text{Pt} \) decreased, and (iii) was unchanged despite an increased central command in END (as reflected by an increase in RMS/M).

4.1. Methodological considerations

To verify the robustness of the %Det parameter in detecting dynamical structures in the EMG signals, surrogate data testing was applied to all data recorded during the 10-min intermittent exercise (Fig. 2). Shuffling the temporal series produced a strong reduction in %Det. Thus we can reject the hypothesis of an uncor-

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Fig. 3. Typical recurrence plots obtained and the associated VL EMG signals in one power-trained athlete (POW) and one endurance-trained athlete (END) during the first (A) and the last (B) contractions of the 10-min exercise at 50% of MVC.

Fig. 4. Change in the Pearson correlation coefficient between peak torque (Pt) and percentage of determinism (%Det) during the 10-min exercise. *Correlation coefficient under the statistical level of significance.
related stochastic underlying process. In other words, this result confirmed that the \%Det parameter of the original EMG time series reflected a certain (not purely random) temporal structure (Riley et al., 1999).

4.2. Detection of muscle state changes

The Pt time courses (Fig. 1A) indicated that at the beginning of exercise in POW, and throughout exercise duration in END, an elevated potentiation occurred (Hamada et al., 2000), characterized by increased torque development for the same electrical stimulation. During this phase, \%Det did not change in either group (Fig. 1C), showing that the RQA method did not detect potentiation. The potentiation of torque production is classically explained by the increased sensitivity of skeletal muscle fibers to calcium driven by the phosphorylation of myosin regulatory light chains (Moore and Stull, 1984; Sweeney and Stull, 1986; Metzger et al., 1989; Houston and Grange, 1991). The absence of \%Det modification during the Pt increase phase showed that the sensitivity of RQA during muscle state change, as described by Webber et al. (1995), does not reflect muscle potentiation.

In a second phase observed in POW, Pt decreased from 5 min 30 s to the end of exercise (Fig. 1A). The submaximal intermittent exercise produced manifest fatigue in this group. In parallel to the Pt drop and that this detection of fatigue occurred earlier than with the neurostimulation method (time difference of about 2 min, Fig. 1A vs. Fig. 1C). In fact, our results showed that, in terms of differential sensitivity, the subtypes of the muscle's internal dynamics were followed more closely by the nonlinear RQA variable. This is consistent with the conclusion of Webber et al. (1995), who showed that RQA is highly sensitive to muscle state changes. Given that Pt reflects the net balance between potentiation and fatigue (Rankin et al., 1988; Rassier and Macintosh, 2000) and that \%Det did not detect potentiation (present study), it was logical to find that \%Det detected fatigue earlier. In END, \%Det remained constant while Pt was above the baseline value. Thus, in this group we obtained a confirmation that \%Det remains unchanged during potentiation. The difference in the Pt time courses between the two groups thus allowed us to confirm that \%Det increases only if potentiation vanishes during exercise. The increase in \%Det is classically attributed to EMG self-organization into more periodic waves, a process which may be caused by an increase in the probability that motor units will discharge in unity (Webber et al., 1995). This mechanism can be consistent with greater motor unit synchronization, which is in agreement with previous studies (Farina et al., 2002; Del Santo et al., 2006). Liu et al. (2004) reported similar results during dynamic exercise and indicated that the increase in \%Det was correlated with muscle fatigue, even though the underlying physiological mechanisms are still not completely understood, particularly the implication of the different fatigue components. Indeed, motor unit synchronization, which is presented in the literature as a potential mechanism underlying the \%Det increase, can involve either purely central pathways (central command) or an interaction between central and peripheral components. As an example, the recurrent inhibition of Renshaw cells, known to influence motor unit synchronization (Mattei et al., 2003; Del Santo et al., 2006; Uchiyama and Windhorst, 2007), can be affected by descending central command per se and/or by sensorial muscle afferences during contractions that induce fatigue (Windhorst, 1996; Gandevia, 2001).

4.3. RQA and fatigue components

The specific time courses of Pt and RMS/M yielded important information about RQA sensitivity to fatigue components. Similar to our Pt results in the POW group, Behm and St-Pierre (1997) found a reduction in Pt during a prolonged submaximal exercise and presented this result as an impairment in excitation–contraction coupling. As seen previously, the reduction in Pt in POW was associated with an increase in \%Det (Fig. 4, P < 0.05), whereas Pt remained high in END with unchanged \%Det. These results were in line with the finding that \%Det detects the peripheral component of fatigue. The significance of these findings is that RQA detected one or several mechanisms involved in peripheral fatigue, i.e., from the neuromuscular junction to excitation–contraction coupling. Allen and Westerblad (2001) suggested that impaired excitation–contraction coupling could be caused by a decrease in calcium release from the sarcoplasmic reticulum and the sensitivity of contractile proteins to calcium. Above we have indicated that RQA did not detect the mechanisms linked with potentiation, i.e., release of and sensitivity to calcium. Thus, our results support the hypothesis that \%Det could detect an alteration in the depolarization capacity of sarcotubule. Indeed, Fowles et al. (2002) indicated that neuromuscular fatigue can be associated with sodium–potassium–ATPase activity. West et al. (1996) found a strong relationship between Pt fall and femoral venous plasma potassium, suggesting that the increased extracellular potassium may be exerting its effect in the T-tubular region, leading to a hyperpolarization state and thus an inhibition of the triggering excitation–contraction coupling mechanisms. Potassium pumps can affect the ionic state of the sarcotubule and this is consistent with EMG signal alterations (Shushakov et al., 2007). Meanwhile, our study cannot rule out the association of other potential mechanisms, such as the activity-dependent hyperpolarization of motor axons (Vagg et al., 1998). This would cause conduction failure in action potentials, thereby producing changes in muscle output, even though the increase in RMS/M indicates increased central activity, as discussed further on.

Another factor of the peripheral component of fatigue can be proposed: the action potential conduction velocity, although this was not measured in the present study. According to Farina et al. (2002) and Fattorini et al. (2005), the conduction velocity could influence the evolution of \%Det, but unfortunately these observations were made on the basis of a simulation mathematical model and, to our knowledge, direct measurements of physiological conduction velocity confronted with RQA results are yet unavailable. In POW, the increase in RMS/M from 3 min of exercise (Fig. 1B) can be attributed to an increase in motor unit recruitment and/or an increase in discharge frequency to maintain a constant force level (Duchateau et al., 2002). The RMS normalization to the M-wave area enabled us to exclude peripheral changes from the interpretation of the data and provided an estimate of central command (Pasquet et al., 2000; Millet and Lepers, 2004). Our result presented in Fig. 1B indicates a marked and early increase in the central command of POW in order to maintain task performance despite minimized excitation–contraction coupling properties (i.e., decrease in Pt). Noakes et al. (2005) interpreted this strategy of adaptation as the action of a central ([brain] neural control that regulates performance “in anticipation”. This was supported by the RMS/M ratio, which increased before Pt returned to its baseline value (4 min 30 s). In END, the increase in central command characterized by the decrease in RMS/M was observed at the end of exercise, with Pt values that always remained above the baseline value and no modification in \%Det. In summary, the Pt and RMS/M time courses in POW indicated both an alteration in excitation–contraction coupling and an increase in central command, associated with an increase in \%Det. In this group, the sensitivity of RQA to peripheral or central fatigue
components could not be identified. In contrast, a persistent potentiation and an increase in central command were observed at the end of exercise in END, while %Det remained unchanged. Thus, a link between %Det and RMS/M changes can be ruled out. Overall, our results clearly indicated that RQA does not detect an increase in central command.

In conclusion, as expected, the two selected populations allowed us to distinguish which phenomena could be detected by RQA during the submaximal intermittent exercise. The comparisons between END and POW yielded strong evidence that RQA does not detect potentiation or neural drive increase and that it is a discriminating tool to assess peripheral fatigue. In addition to the already described advantages of this method, this work further defines and reinforces its interest.

References


