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# Clinical Implications of Muscle-Tendon & -Force Interplay: Surface Electromyography Recordings of *m. vastus lateralis* in Renal Failure Patients Undergoing Dialysis and of *m. gastrocnemius* in Individuals with Achilles Tendon Damage

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## 1. Introduction

The Surface Electromyography (sEMG) technique offers a safe, quick, pain-free, non-invasive and repeatable means of assessing the important physiological processes that cause muscles to generate force and produce movement, yet it has many limitations that must be understood, considered, and removed where possible (Hermens et al., 1999). Thus, it has been said of electromyography that;

*"EMG is a tempting muse .. it is too easy to use and consequently too easy to abuse"*  
(De Luca CJ 1993 – Wartenweiler Memorial Lecture).

An sEMG signal comprises what is otherwise termed a compound muscle action potential (CMAP) being the sum of any number of motor unit action potentials within a given recording area. However, just such an sEMG signal, which provides an insight into the level of activity of a given skeletal muscle, can be altered/affected in many ways by what have been defined as causative, intermediate and deterministic factors (De Luca, 1997). The causative factors include such parameters as the configuration of the recording electrodes (size, distance apart etc.), skin thickness and subcutaneous tissue composition and depth, blood flow, muscle fibre diameter and orientation as well as the number of active motor units within the muscle of choice and their firing rate one with another, that is to say their synchronization (De Luca, 1997). Added to these parameters are the intermediate factors which include the choice of using a differential electrode filter, the risk of cross-talk from adjacent muscles or underlying tissue, the occurrence of spatial filtering as a consequence of fibre depth, the volume of the muscle that is detectable, and the conduction velocity of muscle fibres. Finally, there are the deterministic factors which comprise the number of

active motor units within a muscle as well as their detectability, their recruitment and stability, plus the action potentials resulting from such active motor units and their amplitude and duration. These then, when combined, give rise to an sEMG or compound muscle action potential which has a given amplitude and any number of spectral variables. It is the analysis of such sEMG signal data and its interpretation in terms of muscle force (RMS conversion), the activation of a muscle (On *versus* Off), any signs of fatigue and hints relating to the biochemical processes in a muscle that make this such a tempting, yet complex field to study (see Fig. 1 & 2).

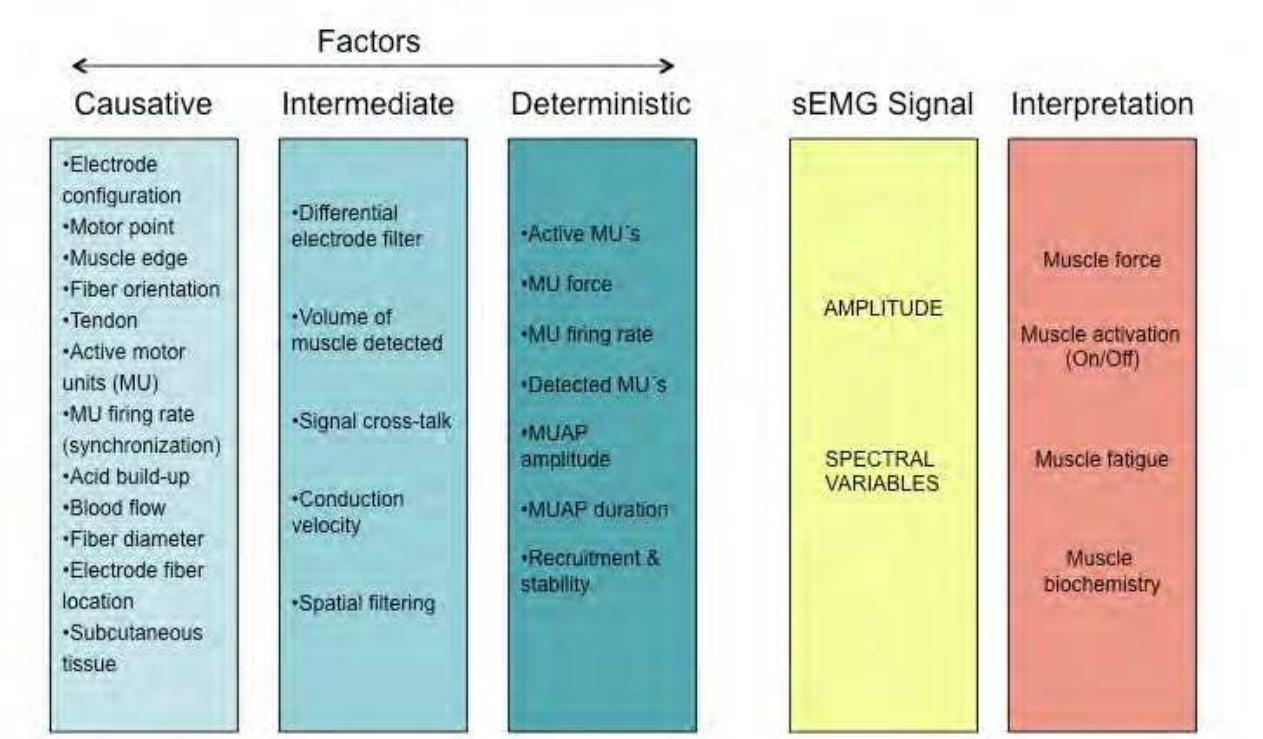


Fig. 1. The factors “causative”, “intermediate” and “deterministic” and their effect on both sEMG signal amplitude and spectral variables, and the interpretation one can make of a recorded sEMG signal (after De Luca, 1997).

In spite of the large number of variables that influence the sEMG signal (De Luca,1997; Farina et al., 2004; Hogrel, 2005), studies on correlations between intrinsic muscle parameters and recorded signals are becoming more numerous (Gerdle et al., 1997; Karlsson & Gerdle, 2001; Kupa et al.,1995; Larsson et al., 2002 & 2006; Tygesen & Harrison, 2005). In rats, EMG parameters have been shown to be a useful tool to predict muscle excitability (Harrison & Flatman, 1999), and in sheep the technique has been used to follow postnatal muscle development (Tygesen & Harrison, 2005). sEMG recordings have also been related to the morphological state of muscles, for example i) muscle fibre composition (identified using MHC isoforms or mATPase activity)in humans (Gerdle et al., 1997; Larsson et al., 2002 & 2006) and in rats (Kupa et al., 1995) and, ii) muscle fibre diameter in humans (Gerdle et al., 1997; Larsson et al., 2006) and in rats (Kupa et al., 1995). With the non-invasiveness of sEMG recordings comes the possibility, for clinicians and physiologists alike, to determine whether structural and functional changes have occurred in muscles over a period of time.

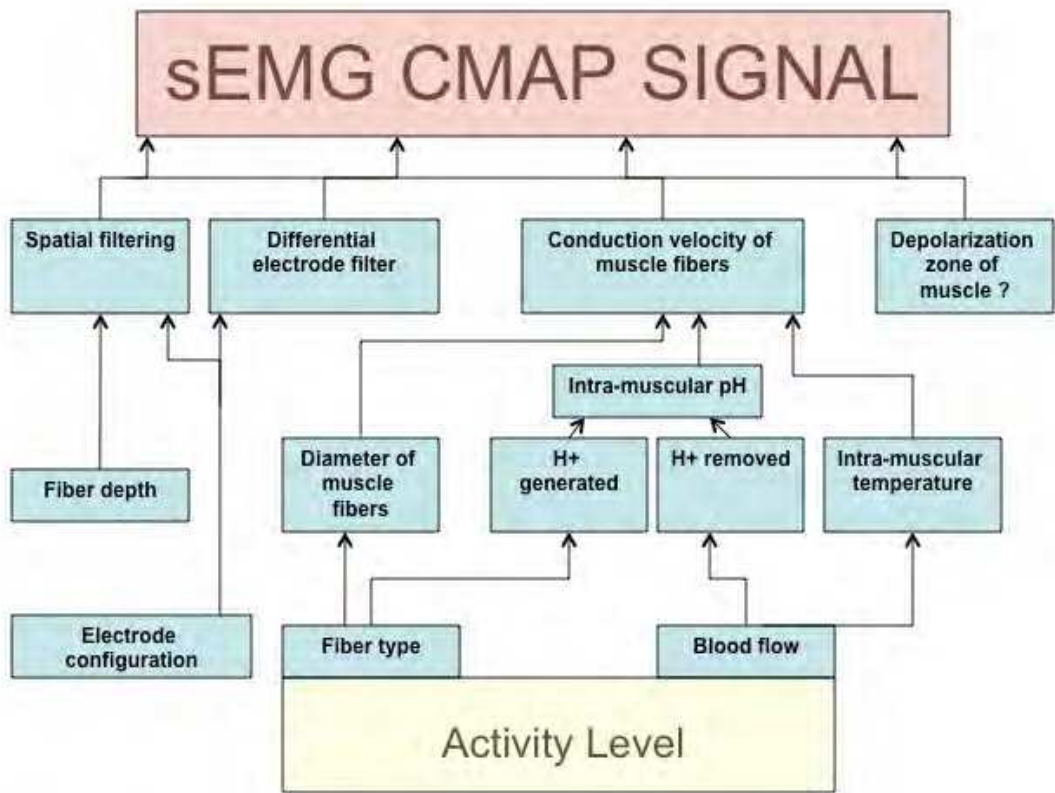


Fig. 2. Factors affecting the sEMG signal during a period of sustained contraction. In the case of “conduction velocity”, “intramuscular pH” and “H<sup>+</sup> generated” a clear association between changes in these parameters over time during a period of sustained contraction has been established. It is less clear, however, as to whether and with what degree “depolarization zone of muscle” is changed during a period of sustained contraction (after De Luca, 1997).

Recorded parameters such as signal Area and Corrected Peak relate to muscle force production, and over time they can be used to give an idea of an individual’s fatigue resistance, along with an RMS analysis of the recorded signal, whereas Leading Slope and Trailing Slope are more closely related to the speed of contraction and relaxation in a given muscle, providing information about an individual’s physical capabilities, as well as the fibre-type composition of specific muscles (De Luca, 1997).

It is relevant to measure sEMG prior to and after strength training. An increase in muscle strength after a period of strength training can be due to an improvement in neuro-muscular communication and/or an increase in the cross-sectional area of muscle fibres. In cases where no response to strength training is observed, the limiting factor may be due to affected neuro-muscular function. Indeed, CKD are associated with neuropathies (Krishnan et al., 2005), which could well affect the sEMG signal and any response to strength training. Thus the sEMG serves as a control/explanatory factor together with muscle strength measurements and CSA fibre measurements when looking for an indicator of strength training success in individuals.

The sEMG of a person with good muscle function would therefore be characterized by a signal with the following characteristics; relatively high frequency, large corrected peak, relatively stable RMS – at least initially, and fast leading and trailing slopes.

## 1.1 Understanding & interpreting sEMG signals

### 1.1.1 sEMG parameters

sEMG signals can be characterized either by temporal analysis (e.g. peak amplitude, root mean square or average rectified values), by spectral analysis (e.g. mean frequency, median frequency), or by propagation analysis (e.g. muscle fibre conduction velocity).

Amplitude estimations are used frequently in sEMG research. The amplitude of a voluntary signal is stochastic (random) and can therefore be reasonably represented by a Gaussian distribution function. The amplitude of a sEMG signal is highly influenced by the distance and the conductivity of the tissue between the active fibres as well as the recording electrodes, their properties and placement. The root mean square, that is to say the square root of the average power of the sEMG signal for a given period of time, is a technique that allows for rectification of the raw “bipolar” signal, converting it to a “monopolar” amplitude that is easier to present and interpret (Hermens et al., 1999). The average rectified value takes the rectified signal one step further and represents the area under the rectified sEMG signal over a period of time. It is generally thought that the root mean square is more appropriate when assessing voluntary contractions as it represents the signal power, unlike the average rectified value, which has no specific physical meaning (De Luca, 1997).

### 1.1.2 sEMG issues

As motor units are recruited and potentially increase their firing rate during sustained muscle activation, increasing numbers of motor unit action potentials (MUAP's) may contribute to an sEMG signal at any one point in time. It would be logical then to predict that the magnitude of the sEMG signal should increase almost linearly with activation rate. However, the sEMG signal underestimates the actual activation signal sent from the spinal cord to a particular muscle since increasing MUAP overlap and signal cancellation typically occurs, and this reduces the sEMG signal. This loss of sEMG signal has been shown to arise from the linear summation of overlapping positive and negative phases of motor unit potentials which serve to cancel one another out and thereby reduce the amplitude of the signal (Day & Hullinger, 2001; Keenan et al., 2005; Keenan et al., 2006).

In general terms, the sEMG parameters “area” and “amplitude” relate to the force of contraction, yet no simple equation exists to describe their relation. Indeed, the fact that the amplitude increases as muscle force increases only serves to provide a qualitative indicator. Quantitatively, exactly how much the force varies between two tasks cannot at present be answered with any certainty. One should be very cautious then when using the sEMG signal as an absolute measure of the force developed by a muscle as there are occasions when this relation can become non-linear (De Luca, 1997; Turker, 1993). Apart from the fact that the amplitude is highly influenced by both extrinsic and intrinsic parameters, two other factors have an enormous effect on the relation between force and amplitude. The first is that of the number of motor units detected by the recording electrodes is almost always less than the number of active motor units in an active muscle. The second is the issue of relative placement of active motor units with regard to the recording electrodes, such that if a newly recruited motor unit is located in close proximity to a recording electrode, that motor unit will contribute more than an average unit of energy to the recorded signal. Conversely, if a newly recruited motor unit is located at a distance from the recording electrode, the force in that muscle will most likely increase whilst the amplitude of the sEMG signal will not alter dramatically (De Luca, 1997).



Spectral analysis of the sEMG signal has been used to study muscle fatigue (Komi & Tesch, 1979; Linssen et al., 1991; Milnerbrown & Miller, 1986; Moritani et al., 1982), and changes in motor unit recruitment (Bernardi et al., 1996; Bernardi et al., 1999; Solomonow et al., 1990). Spectral analyses have also been used to estimate the activation of type I and type II muscle fibres during a contraction (Gerdle et al., 1988; Gerdle et al., 1991; Tesch et al., 1983), as well as to predict the actual muscle fibre type composition (Kupa et al., 1995). Interpretation of the most commonly used spectral descriptors (mean frequency and median frequency) is facilitated by the use of the power spectral density function. The mean frequency has a smaller estimation of variance than the median frequency, yet the median frequency is less sensitive to noise and more sensitive to fatigue (Hermens et al., 1999; Merletti et al., 1992). Thus these different characteristics should be considered when analyzing an sEMG signal. For example, under conditions of a high signal:noise ratio the mean frequency is preferable due to the low variance, but under conditions where the signal:noise ratio is low, one should consider using the median frequency because of its relative insensitivity to noise (Hermens et al., 1999).

In propagation analysis, the muscle fibre conduction velocity is measured using two electrodes with the same alignment as the muscle fibres. By measuring the inter-electrode distance, the muscle fibre conduction velocity can be calculated as the ratio between the distance and the time delay between the two recorded signals. In most cases the underlying tissue poses very little local variation, so in terms of both detected signals the filtering effects can be assumed to be similar. This gives the muscle fibre conduction velocity parameter an advantage compared to temporal and spectral analysis, where the filtering effect of underlying tissue may distort the travelling signal (Hermens et al., 1999; Hogrel, 2005). The muscle fibre conduction velocity is to some degree proportional to the muscle fibre diameter. Thus in general muscles with large diameter fibres, such as those belonging to higher threshold motor units, will possess a greater average muscle fibre conduction velocity, which will in turn shift the frequency spectrum towards the high frequency range (De Luca, 1997).

To illustrate use of sEMG in a clinical setting, we will give an account of two chosen studies which together will show where sEMG has its strength and which considerations have to be made when using sEMG in these settings.

## 2. Uremic study

### 2.1 Introduction

Patients with end-stage renal disease (ESRD; uremia) have limited physical fitness, which can lead to subsequent problems, namely cardiac dysfunction and depression (Gutman et al., 1981; Painter, 1988; Kouidi et al., 1997; Shalom et al., 1984), as well as muscle abnormalities, which severely affect their daily life, whether it be work- or recreation-related (Nakao et al., 1982). Yet, whilst muscle weakness is a common complaint amongst dialysis patients, it remains an unexplained phenomenon.

Muscle atrophy with uremia has been shown to be fibre-specific, in that the disease is mainly associated with a loss of type II fibres and predominantly the type IIB fibres, which are fast-twitch and glycolytic in nature (Fahal et al., 1997). Atrophy of type I (slow-twitch, oxidative fibres) also occurs in some patients (Molsted et al., 2007; Moore et al., 1993). Yet despite these changes, exercise training has been shown to significantly alleviate the loss of physical capacity that occurs in dialysis patients with end-stage renal disease (Shalom et al., 1984; Painter, 1988; Eidemak et al., 1997; Kouidi et al., 1998; Molsted et al., 2004).

Whilst these patients suffer an array of medical problems that plagues them and the quality of their life (QoL), they are interested in receiving any form of help that will improve their condition and help them to lead a more normal life. It is for this reason that studies have been attempted to assess the causes behind the observed muscle atrophy and physical weakness that they incur. It should be mentioned though that these studies are not without problems. Patients arriving for a period of dialysis are usually very weak and can become easily confused, and after their dialysis most cannot wait to get out into the World and use the time and energy they have had temporarily restored upon them. This necessitates that any experimental design must be simple and easy to understand, it should not be painful, and most of all it must be relatively quick – time being of the essence after a period of dialysis. Thus it has been our experience that whilst studies of nerve conduction have not been accepted by patients with end-stage renal disease, being seen as both time consuming and painful, our sEMG recordings have been well tolerated.

## 2.2 Patients

Patients were recruited from Rigshospitalet, Denmark. Inclusion criteria were; ages above 18 years, undergoing haemodialysis for a minimum of three months, and being able to climb stairs. Exclusion criteria were inability to participate in the intervention due to physical health, blindness, lower limb amputation, diabetic retinopathy, severe cognitive reduction, and treatment with the anti-coagulation drug Fondaparinux. All patients gave their informed consent and the protocol was approved by the local ethical committee (H-D-2008-124).

## 2.3 Training programme

The included patients participated in a training programme consisting of heavy load resistance training three times a week for a period of 16 weeks. The exercise sessions varied in time from the last dialysis, since some patients were dialyzed three times a week, others only twice a week.

The exercise began with 5 minutes of warm-up. Three exercises were performed at each exercise session: leg press, leg extension and leg curl. During the intervention period the load was increased and repetition maximum (RM) decreased from 15 to 6 RM. An individual patient's progress during the interventions was adjusted according to changes of 1-6 RM.

## 2.4 sEMG recordings, measurements and analyses

The study used both a single and a double differential electrode configuration, with electrodes (N-00-S & R-00-S; Blue Sensor R, Medicotest A/S, Ølstykke, Denmark) configured as described previously (Harrison et al., 2006). sEMG recordings were taken *via* an ML 131 amplifier connected to a PowerLab 4/25T A/D converter (AD Instruments, Chalgrove, Oxfordshire, UK) with a further connection to a Mac PowerBook Air with Chart v. 5.5.6 Software, Peak Parameters, and Spike Histogram extensions. Input impedance was 200 M $\Omega$  differential, and a high- and a low-pass filter of 3 Hz and 500 Hz, respectively, were used. Sampling speed was set to 40,000 per second.

Recordings, which were taken from the vastus lateralis of the left leg, followed the guidelines laid out in the *European Recommendations for Surface ElectroMyoGraphy* as detailed by the SENIAM project (Hermens et al., 1999). Differential recordings of sEMG signals were made *via* surface electrodes from the *vastus lateralis*, as described in detail previously. Information of any expected electromyography performance results were not divulged, and

patients were not allowed to follow their sEMG recordings on the computer screen in real time. The recorded sEMG signal was assessed as described previously (Harrison et al., 2006) in terms of signal frequency (Hz) and peak-to-peak amplitude (V), using Chart analysis software (AD Instruments, Chalgrove, Oxfordshire, UK).

2.5 Statistics

Data distribution was tested using a Q-Q plot. Due to the discovery that the data was not normally distributed, statistical analysis of any effects was performed using non-parametric tests. A Wilcoxon Signed Ranks Test was applied to measure significant differences between pre-training (Baseline) and post-training (Re-test) values. Data are presented as means plus and minus the standard error of the means (SEM). All tests were two-tailed and significance was deemed to be at  $P \leq 0.05$ .

2.6 Results

A clear and significant improvement in the amplitude of the recorded sEMG signal was noted in the uremic patients when values taken pre-training were compared with those obtained post-training (see Fig. 3).

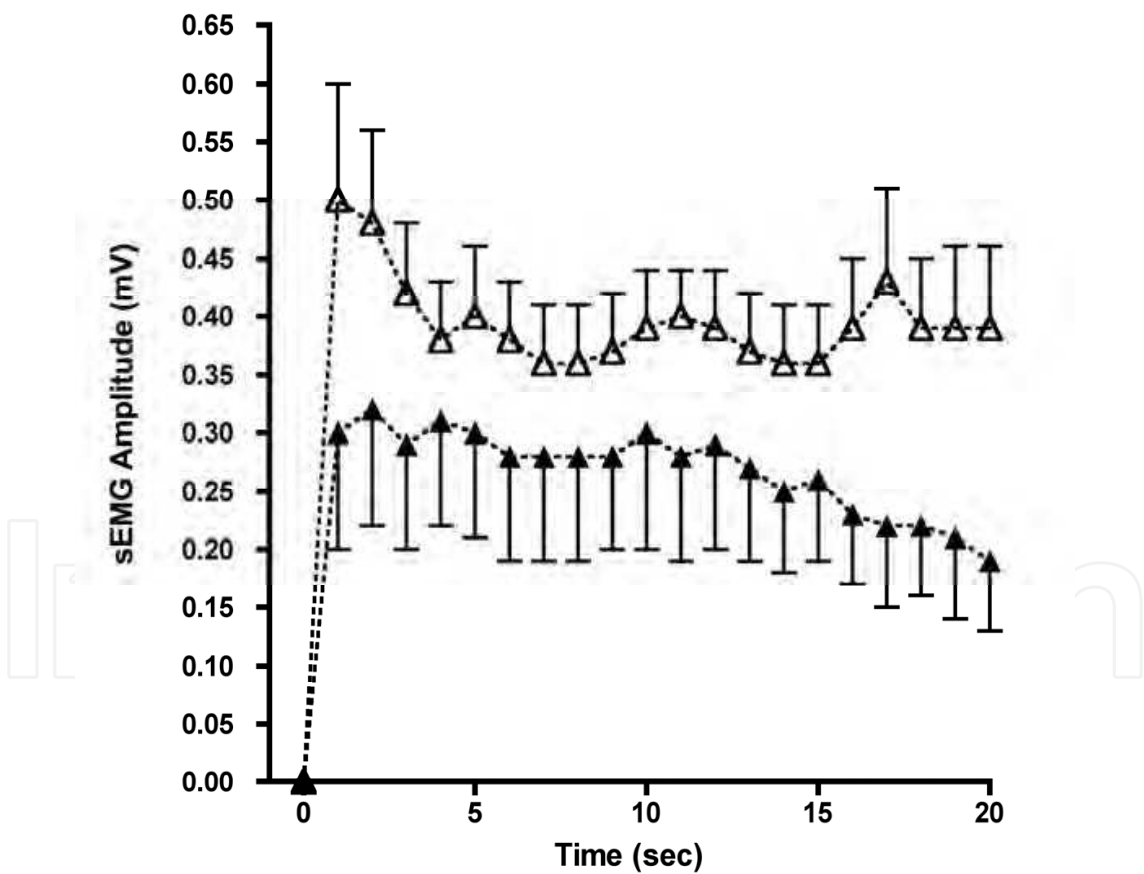


Fig. 3. The recorded sEMG signal from *m. Vastus lateralis* of uremic patients taken prior to (▲) and after a 16 week programme of strength training (△). Recordings, which represent a 20 second period of sustained leg lift, were taken at a sampling rate of 40,000 data samples per second, using an impedance of 200 MΩ and a high- and low-pass filter of 3 Hz and 500 Hz, respectively. Values represent the Mean ± SEM of 8 patients.



In contrast, to the sEMG amplitude values, measurements of the sEMG signal frequency from these uremic patients were not significantly altered by a 16 week period of strength training (see Fig. 4). Indeed, if anything one gets the impression, at least initially, that a lower firing frequency is employed by these patients during a period of sustained leg extension and contraction of *m. Vastus lateralis*.

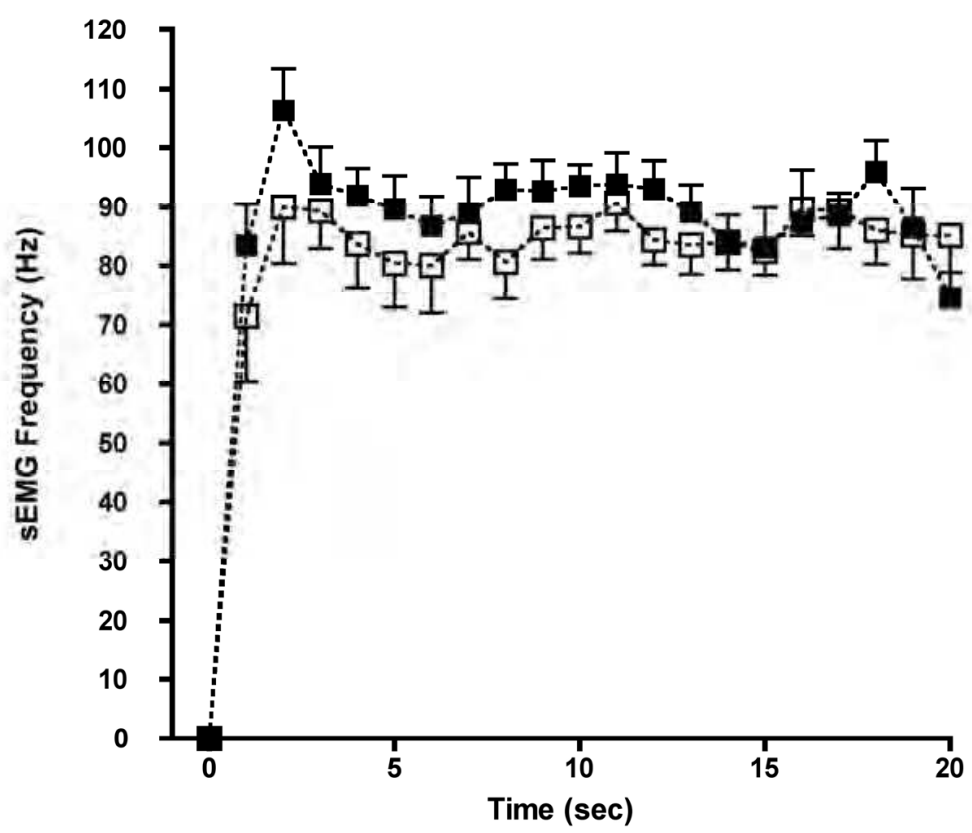


Fig. 4. The recorded sEMG signal from *m. Vastus lateralis* of uremic patients taken prior to (■) and after a 16 week programme of strength training (□). Recordings, which represent a 20 second period of sustained leg lift, were taken at a sampling rate of 40,000 data samples per second, using an impedance of 200 MΩ and a high- and low-pass filter of 3 Hz and 500 Hz, respectively. Values represent the Mean ± SEM of 8 patients.

Our investigation of muscle performance in the uremic patients also included a sEMG recording of *m. Vastus lateralis* taken during a period of dynamic contraction (see Fig. 5).

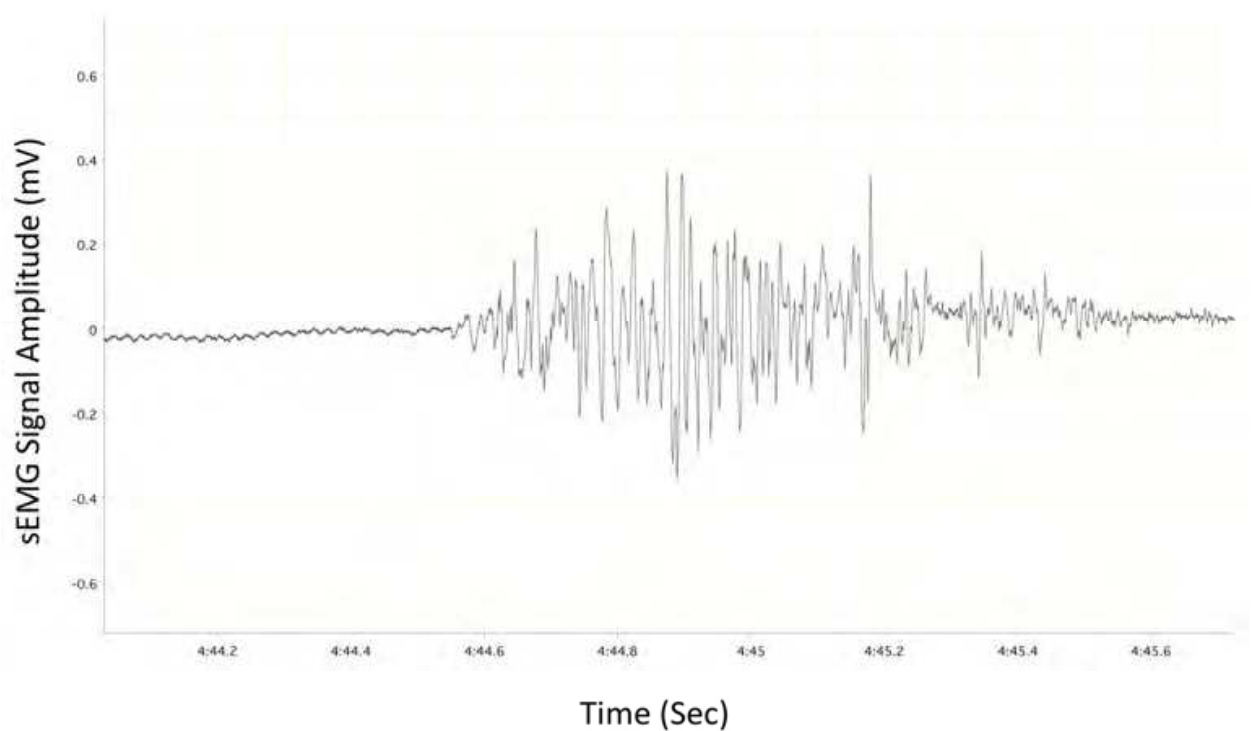


Fig. 5. The results of a dynamic muscle strength test involving a knee extension with 1 RM. This figure illustrates a sEMG recording taken from *m. Vastus lateralis* at a recording speed of 40,000 data samples per second. Note the burst of power as the leg is extended. This sEMG signal was analyzed in terms of its peak-to-peak amplitude (mV) and its inherent frequency (Hz).

Both the dynamic and the isometric sEMG signal were analyzed in terms of their inherent frequency and their amplitude. Recordings taken after a period of 16 weeks of strength training "Re-test" were then compared with "Baseline" pre-training levels, and the results are shown below in Table 1.

<i>Dynamic 1RM 1. Sec</i>			
<i>Parameter</i>	<i>Baseline</i>	<i>Re-test</i>	<i>P</i>
Frequency (Hz)	71 ± 11	71 ± 6	NS
Amplitude (mV)*	309 ± 81	571 ± 71	0.017
<i>Dynamic 1RM 1. sec/kilo</i>			
Frequency/kilo (Hz/Kg)	1.3 ± 0.2	0.9 ± 0.1	NS (0.093)
Amplitude/kilo (mV/Kg)*	6.2 ± 2.4	7.2 ± 1.1	NS
<i>Isometric 50% 1RM 20 sec</i>			
<i>Parameter</i>	<i>Baseline</i>	<i>Re-test</i>	<i>P</i>
Frequency Peak (Hz)	107 ± 6	102 ± 5	NS
Amplitude Peak (mV)*	350 ± 104	568 ± 86	NS
Frequency Mean (Hz)	89 ± 5	85 ± 5	NS
Amplitude Mean (mV)*	273 ± 83	396 ± 54	NS
<i>Isometric 50% 1RM 20 sec/kilo</i>			
Frequency Peak/kilo (Hz/Kg)	3.7 ± 0.5	2.6 ± 0.4	0.017
Amplitude Peak/kilo (mV/Kg)*	14.8 ± 6.6	14.2 ± 2.3	NS
Frequency Mean/kilo (Hz/Kg)	3.0 ± 0.3	2.2 ± 0.3	0.017
Amplitude Mean/kilo (mV/Kg)*	11.6 ± 5.2	10.1 ± 1.7	NS

Table 1. The response of *m Vastus lateralis* to a period of 16 weeks of strength exercise training in uremic patients. Data are presented as uncorrected values *versus* values corrected for the weight lifted (per kilo) by the patients. Note that sEMG correction for individual variation in terms of the weight lifted has a huge effect on the results, highlighting the fact that signal analysis can affect the interpretation of the recordings. Values represent the Mean ± SEM of n=8 haemodialysis patients. \* indicates x 1000.

2.7 Discussion

The first large-scale studies of the physical capacity of patients with chronic renal failure were performed at the end of the 1970’s as reviewed by Laville & Fouque (1995). It was noted that 50% of patients requiring dialysis had stopped their professional activity as a result of chronic renal failure, attributed to coronary (15% of patients), cardiovascular (23%), and bone or muscle (24%) related conditions (Evans et al., 1985). In a recent study involving patients on haemodialysis, *in vivo* measurements of muscle function were made using sEMG (Harrison et al., 2006). The sEMG frequency recorded prior to dialysis was generally found to be abnormal, when compared with a normal range of 2<sup>nd</sup> dorsal interosseous muscles of the hand and on the thigh *m. Vastus lateralis*. This study also revealed a clear benefit of haemodialysis in terms of sEMG frequency of the 2<sup>nd</sup> dorsal interosseous muscle (Harrison et al., 2006). Moreover, these authors found that the *in vivo* analysis of sEMG changes with a session of haemodialysis seemed to be limited to relatively

fast-twitch muscles, a finding that was supported by a study of isolated rat *extensor digitorum longus* muscle, in which changes in the uremic environment were shown to exert a rapid loss of contractile force (Harrison et al., 2006).

In the present study, the combined graphs and table show that a period of exercise training results in an improved sEMG amplitude with very little change to the signal frequency. This finding, which indicates that a period of exercise training in these patients results in the ability to lift more weight (kilos), may be due to enhanced spatial summation (Henneman et al., 1965) - that is to say an increased and more synchronized recruitment of motor units. However, a considerable degree of individual variation is masked by this data set, and upon correction of the sEMG amplitude and frequency data for the weight in kilos used under training, a different interpretation is arrived at. Suddenly, signal analysis designed to remove an element of individual variation from the data now reveals that a period of exercise training induces an increase in sEMG signal frequency, and not an alteration in sEMG amplitude. It has to be said though, that this form of signal analysis reveals a finding that is more consistent with what is known to occur during the initial period following commencement of exercise training. What is observed is an improvement in neuro-muscular recruitment and activation "coordination" that typically precedes fibre and vascular changes "hypertrophy". Indeed, this conclusion is supported by the fact that looking at the histology of the fibres, we have not found any significant changes in fibre, cross-sectional area with the 16 week period of strength training in biopsies taken from *m. Vastus lateralis* in these patients (data not shown).

Of course recordings of sEMG from muscles of such patients are not without problem, and the interpretation of such data should not be made without careful consideration. In Figures 1 & 2 we identified a number of factors that can affect sEMG recordings, and in terms of uremic patients perhaps the most important would be as follows;

**Fig. 1:** **Causative** – active motor units, motor unit firing rate (synchronization) & fibre diameter

**Intermediate** – volume of muscle detected (fibre diameter) & conduction velocity

**Deterministic** – active motor units, detected motor units, motor unit action potential amplitude, motor unit action potential duration, recruitment & stability

**Fig. 2:** Diameter of muscle fibres, conduction velocity of muscle fibres & blood flow

In uremic patients one finds that ion imbalances and the build up of toxic compounds, confounded by other health issues and the effects of medication, affect neuro-muscular recruitment and activation. The effect is that neural conduction velocity is impaired, that fewer motor units tend to be active, and when active they often reveal action potentials that are of long duration and consequently of small amplitude. More long-term changes with uremia also include an atrophy of muscle fibres and a reduction in vascularisation, which will affect causative factors as well as intermediate factors, and furthermore reduce the blood flow to the contracting muscle fibres.

With strength training, it seems that a natural loss of synchronization and recruitment of motor units as the uremia progresses can be reversed. As mentioned earlier, we have not seen any changes in fibre size, and it therefore seems that the improved sEMG signal, which is supported by an improved physical function determined with a sit-to-stand test (data not shown), is due not to an increase in the number or size of active fibres, but rather a better control of those already working fibres.

### 3. Tendinopathy study

#### 3.1 Introduction

The Achilles tendon is one of the most frequently injured tendons of the body due to trauma and overuse – very often in young or middle-aged, physically active subjects (Alfredson et al., 2000; Cook 2009). This arises, most likely, from muscle force in the gastrocnemius being trained to develop an extremely rapid and high level of force without being compensated by equally rapid changes in the Achilles tendon (Olesen et al., 2006; Holm et al., 2009). Although adaptation of tendons has been demonstrated (Langberg et al., 2007; Couppé et al., 2009), this adaption is slower than the development of a stronger muscle, and the tendon does not possess the dimensions necessary to accommodate such rapid increases in force production, and the result will very often be damage to the tendon structure or partial/complete rupture (Hess, 2010).

A number of systemic diseases are also known to be associated with general defects in matrix metabolism and structure that compromise tendon strength and elasticity, moreover the term ‘spontaneous tendon rupture’ is being used on a more regular basis in both the sports- and work-environments (Järvinen et al., 2001). Indeed, despite the fact that the Achilles tendon is the strongest tendon in the human body, there are increasing numbers of cases of overload of the Achilles tendon, both associated with sports as well as to work-related situations. It is estimated that the most common healthcare problem in Denmark is muscular-skeletal, accounting for approximately 15 % of all diseases (Ekholm et al., 2006). In spite of the extent of the problem not much is at present known about the etiology and pathogenesis of chronic tendon pain. The cause of the lack of knowledge within the area is primarily due to several limiting factors. First, that the early development of tendinopathy is unsymptomatic (Fredberg & Stengaard-Pedersen, 2008). Second, the establishment of a human tendinopathy model is deemed unethical. A few animal studies have been performed using an overuse protocol developed by Soslowsky and colleagues (Scott et al., 2007; Perry et al., 2005; Soslowsky et al., 2002), where rats ran with a velocity of 17m/minute, 5 days/week, 1 hour/day, either uphill or downhill for a period of between 2-16 weeks. In such experiments, a decreased collagen fiber organization and increased numbers of cell nuclei were observed (Soslowsky et al., 1996; Glazebrook et al., 2008). Yet, exactly how the tendon manages the active tension transfer from muscle to bone and if this tension transfer is affected by tendinopathy remains unclear. Thus, a form of assessment or a technique that could detect potentially damaging changes in a tendon very early on, and in so doing prevent muscle dysfunction, would be of great benefit.

It is with this point in mind, that we have investigated Achilles tendon parameters in combination with the contractile profile of *m. Gastrocnemius* in a number of healthy sports subjects, as a way of increasing the current knowledge of the ways in which tendon adapts to the mechanical loading associated with running/jogging. Our approach has been to better understand the active participation of tendons in the tension transfer from muscle to bone, and in order to achieve this goal we have returned to the topic of recording artefacts to see if they might not be used to some advantage.

#### 3.2 Subjects

Healthy subjects were recruited from [www.forsøgsperson.dk](http://www.forsøgsperson.dk). Inclusion criteria were; no current tendon injury issues, regular training and ability to complete 60 minutes running on a treadmill. In total 8 subjects were examined (5 males; 3 females). The average age was 31



years (24-40) and average weight of was 74 kg (54-85). All had a BMI between 19 and 25, i.e. normal weight. All participants gave their informed consent and the protocol was approved by the Copenhagen and Frederiksberg Municipalities ethical committee (H-2-2010-121).

### 3.3 sEMG recordings, measurements and analyses

The study used both a single and a double differential electrode configuration, with electrodes (N-00-S & R-00-S; Blue Sensor R, Medicotest A/S, Ølstykke, Denmark) configured as described previously (Harrison et al., 2006). sEMG recordings were taken *via* an ML 131 amplifier connected to a PowerLab 4/25T A/D converter (AD Instruments, Chalgrove, Oxfordshire, UK) with a further connection to a Mac PowerBook Air with Chart v. 5.5.6 Software, Peak Parameters and Spike Histogram extensions. Input impedance was 200 M $\Omega$  differential, and a high- and a low-pass filter of 3 Hz and 500 Hz, respectively, were used. Sampling speed was set to 40,000 per second.

Recordings were taken from the vastus lateralis of the left leg, following the guidelines laid out in the *European Recommendations for Surface ElectroMyoGraphy* as detailed by the SENIAM project (Hermens et al., 1999). Differential recordings of sEMG signals were made *via* surface electrodes from the *vastus lateralis*, as described in detail previously. Information of any expected electromyography performance results were not divulged, and subjects were not allowed to follow their sEMG recordings on the computer screen in real time. The recordings were carried out during 60 minutes treadmill running at a velocity of around 10 km/hour. The recorded sEMG signal was assessed as described previously (Harrison et al., 2006) in terms of signal frequency (Hz) and peak-to-peak amplitude (V), using Chart analysis software (AD Instruments, Chalgrove, Oxfordshire, UK).

### 3.4 Statistics

Data are presented as means plus and minus the standard error of the means (SEM). All tests were two-tailed and significance was deemed to be at  $P \leq 0.05$ .

### 3.5 Results

The results, (see Fig. 5 & 6), show that for lateral and medial *m.Gastrocnemius*, there is a pattern of sEMG signal parameter change over a 60 minute run/jog. After a few minutes of adjustment to a steady running/jogging rhythm, the sEMG mean amplitude becomes fairly stable, only showing signs of fatigue during the last ten minutes. It is likely that there is a fibre type distribution difference between the lateral and medial heads of *m.Gastrocnemius* as the lateral head increases slightly in sEMG frequency, while the amplitude decreases as the run/jog proceeds, whilst the medial head shows the reverse trend.

Of more importance from our perspective is the measurement of the sEMG to tendon artefact peak ( $\Delta mSec$ ), which remains very stable and uniform throughout the entire run/jog (see Fig. 6 & 7).

### 3.6 Discussion

In a recent paper written by De Luca and colleagues (2010) the authors address the issue of inevitable noise contamination of sEMG signals taken from muscles. Such noise originates from the skin-electrode interface, the electronics used to amplify the recorded signal, and a number of external sources (see Figs. 1 & 2). De Luca and colleagues focused on the low-frequency part of the recorded sEMG spectrum and proposed a number of approaches that can be taken to refine the recorded signal in a clinical setting (De Luca et al., 2010).

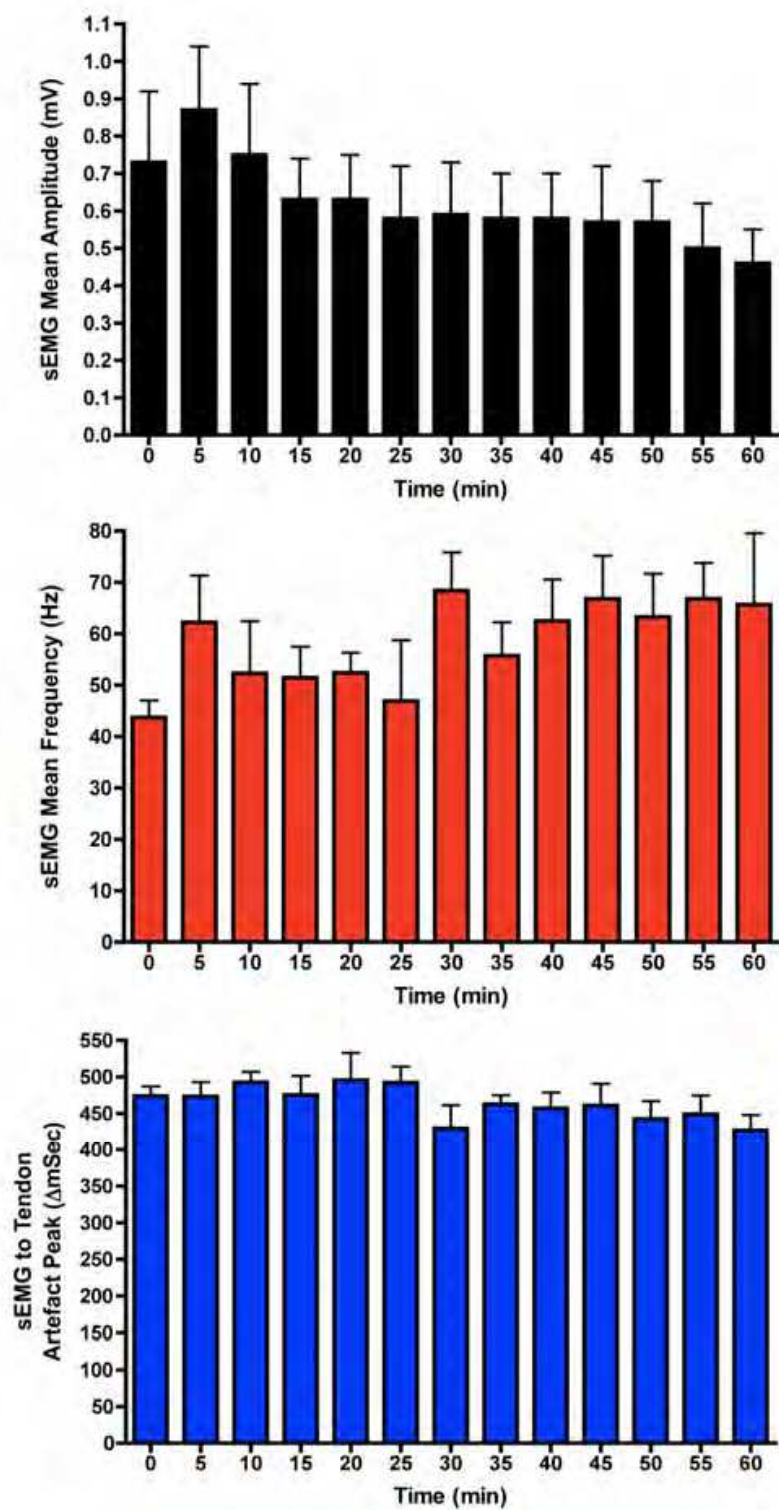


Fig. 6. The mean recorded sEMG signal from *m. Gastrocnemius* (Lateral Head) of healthy subjects – Upper Panel: Amplitude (mV), Middle Panel: Frequency (Hz) and Lower Panel: sEMG to Tendon Artefact Peak (ΔmSec). Recordings, which represent 5 minute interval means of a 60 minute run/jog at 11-13 km/hr, were taken at a sampling rate of 40,000 data samples per second, using an impedance of 200 MΩ and a high and low pass filter of 3 Hz and 500 Hz, respectively. Values represent the Mean ± SEM of 8 subjects.

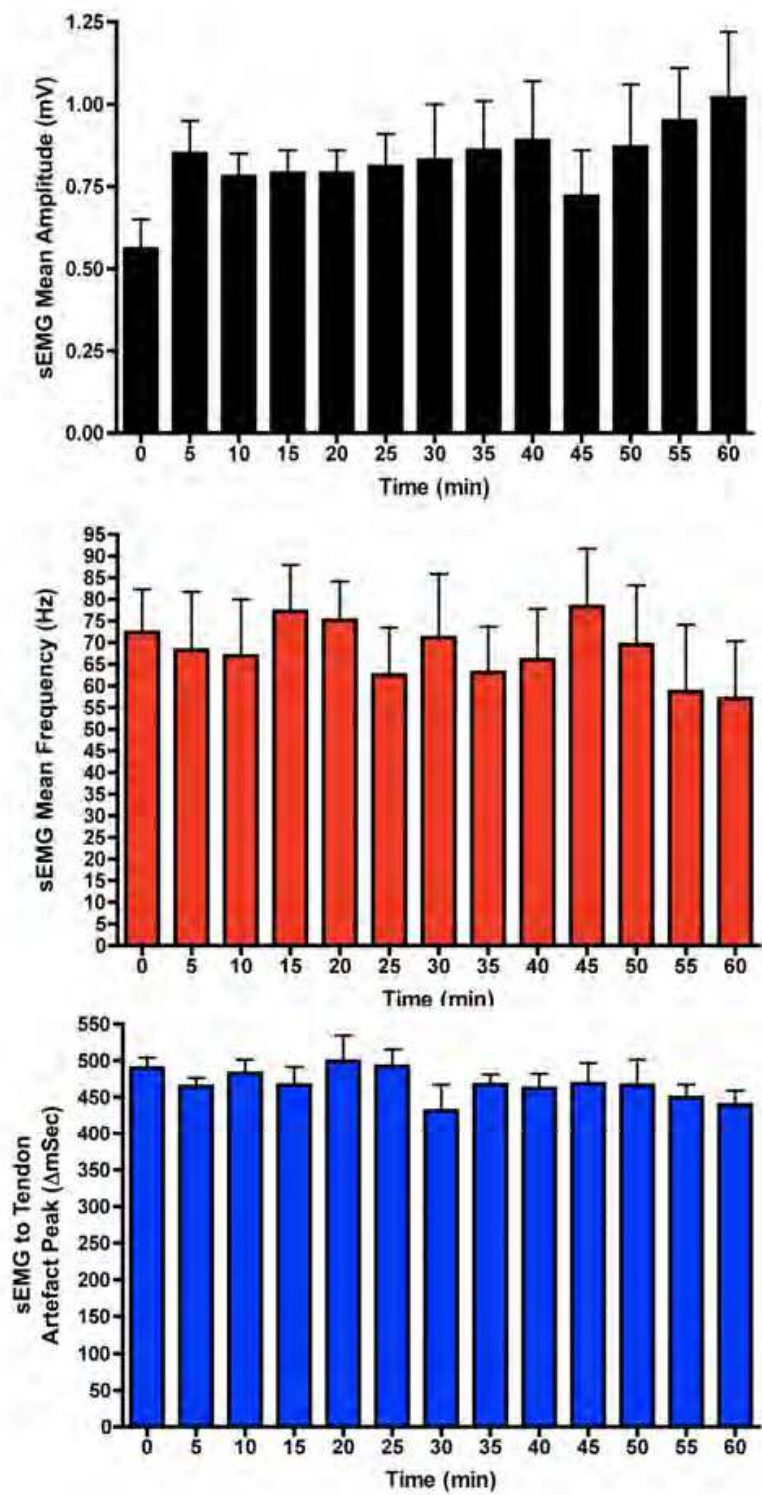


Fig. 7. The mean recorded sEMG signal from *m. Gastrocnemius* (Medial Head) of healthy subjects – Upper Panel: Amplitude (mV), Middle Panel: Frequency (Hz) and Lower Panel: sEMG to Tendon Artefact Peak (ΔmSec). Recordings, which represent 5 minute interval means of a 60 minute run/jog at 11-13 km/hr, were taken at a sampling rate of 40,000 data samples per second, using an impedance of 200 MΩ and a high and low pass filter of 3 Hz and 500 Hz, respectively. Values represent the Mean ± SEM of 8 subjects.

However, a good working knowledge of the artefacts one can encounter when recording a sEMG signal in a clinical setting can also provide a recording opportunity, which researchers may wish to exploit rather than eradicate. Typically, one finds that movement artefacts represent a large proportion of the recorded signal at low spectrum frequencies (60% or more) (De Luca et al., 2010). Here, one may envisage that just such a movement artefact could be of importance and use when assessing muscle:tendon interactions. Indeed, our results, obtained with healthy controls, show that just such a recorded movement artefact taken from the Achilles tendon of individuals whilst running/jogging can be used successfully in connection with a sEMG signal recording from both heads of *m. Gastrocnemius*.

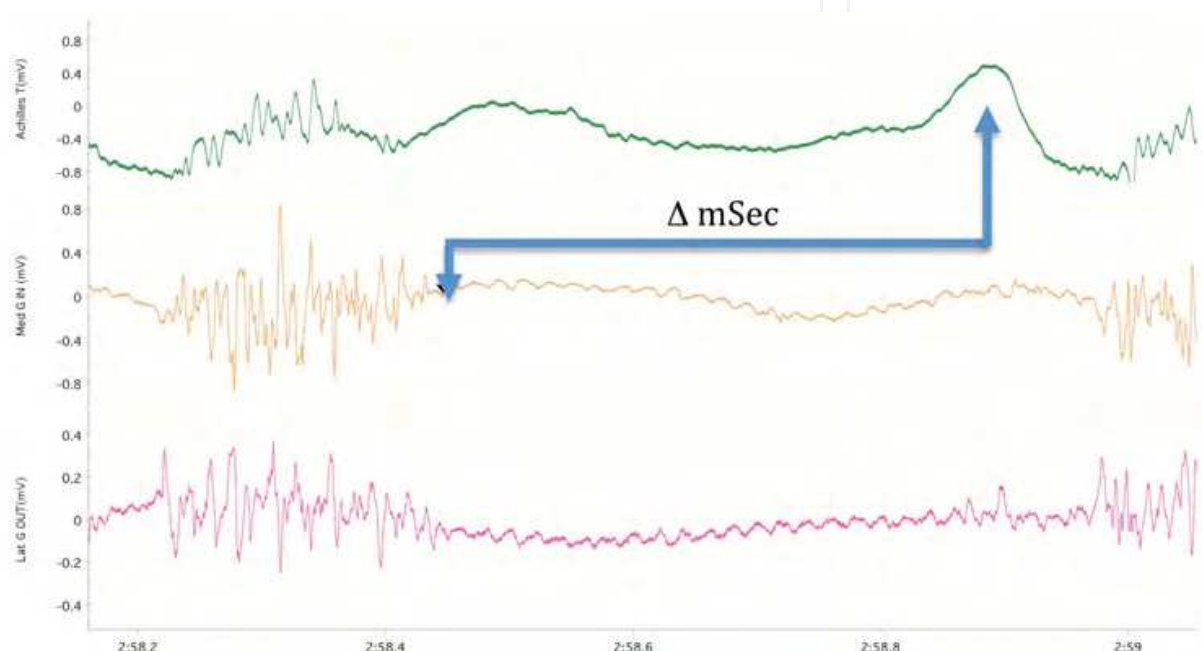


Fig. 8. The results of a 10 km/hr jog on Achilles tendon (upper panel) and both the medial head (middle panel) and lateral head (lower panel) of *m. Gastrocnemius*. The sEMG recording was from the right leg and at a recording speed of 40,000 per second per channel. Note, that after the synchronized bursts of power in the medial and lateral heads of Gastrocnemius a rise in the Achilles tendon signal to a maximum follows, after a delay of some 430 mSec, and this is again followed by a rapid fall back to resting levels. This deflection in the Achilles tendon trace is, however, merely a recording artefact representing the movement of the tendon under tension relative to the electrodes on the skin. The sEMG signal was analyzed in terms of its peak-to-peak amplitude (mV) and its inherent frequency (Hz).

The Achilles tendon is one of the most frequently injured tendons of the body due to trauma and overuse – very often in young or middle-aged, physically active subjects (Alfredson et al., 2000; Cook 2009). However, a recent study has shown that many inactive and often obese people develop tendinopathy (Gaida et al., 2008). Furthermore, one could easily imagine that tendinopathy in such individuals will result in these patients becoming even more inactive and therefore even more overweight. The question that then arises is how does muscle force affect the tendon tissue, when the tissue is overloaded either chronically as with obesity, or very frequently as with elite athletes. Muscle force in the gastrocnemius can be trained to develop an extremely rapid and high level of force, which may not always



be met by equally rapid changes in the Achilles tendon (Olesen et al., 2006; Holm et al., 2009). Although adaptation of tendons has been demonstrated (Langberg et al., 2007; Couppé et al., 2009) the tendon does not possess the dimensions necessary to accommodate such rapid increases in force production, with the result that damage, often to the tendon structure or partial/complete rupture, occurs. Thus, an understanding of the active participation of tendons in the tension transfer from muscle to bone may lead to more optimal and effective treatment of tendinopathy – perhaps through further studies of the sEMG to tendon artefact peak.

Recent *in vivo* studies utilizing ultrasound imaging have shown that the human Achilles tendon undergoes a pattern of rapid lengthening and shortening during the stance phase of running (Lichtwark et al., 2007). Such a change in the tendon has a clear benefit in terms of the return of elastic energy stored in the Achilles tendon to the muscle, making the muscle:tendon unit largely free of metabolic costs. Another advantage of this change is that the Achilles tendon enables *m. Gastrocnemius* fascicles to remain nearly isometric for a large part of each stance (Ishikawa et al., 2007). Combined these changes greatly enhance the efficiency of this muscle:tendon interface.

Creep is defined as the lengthening of an elastic structure held under constant tension, or in other words an increasing lengthening per contraction cycle (Ker et al., 2000). To date, some scientists believe that tendon is capable of creep during periods of constant loading (Ker et al., 2000; Maganaris et al., 2002). However, increased lengthening for a given force as explained by creep would necessitate a reduction in tendon stiffness. Moreover, just such a creep, should it exist in human tendon, must most likely be detectable during the course of a run. In just such an experiment Farris and colleagues (2011) recently documented that the loading experienced during a single bout of running had no effect on the stiffness of the Achilles tendon, and that its properties remained stable throughout the period of activity – a finding that argues strongly against the existence of creep in loaded human tendons.

Looking at our results (see Fig. 6 & 7) for the sEMG to tendon artefact peak, we find that throughout a 60 minute run/jog there are no signs of a change in the time interval between the onset of muscle contraction with the initiation of the sEMG signal in both the lateral and medial heads of *m. Gastrocnemius* in healthy subjects. This confirms the findings of Farris et al., (2011), lending weight to the fact that Achilles tendon properties remain stable throughout a period of activity. Keeping this in mind, the tendon artefact would seem to be a useful tool, rather than something that should be filtered out of the recording, enabling the assessment of tendon inflammation and/or damage at an early stage, thus avoiding muscle dysfunction.

Indeed, the sEMG to tendon artefact peak has been found to be very consistent between individuals in the healthy subject group. The delta mSec value indicates the elasticity of the tendon as it interacts with the contracting muscle, absorbing the muscle force and transferring it gradually and smoothly over to the bone and the ankle joint. This should not be confused with creep, which is a change in tendon length once loaded. Rather the recorded artefact illustrates the development of tension/loading within a healthy tendon as muscle contraction proceeds and tendon loading approaches a maximum. Consequently, if an Achilles tendon is damaged and seriously inflamed, one would expect it to have less elasticity and a lower maximum load upon muscle contraction, which would be observed as a much lower delta value. Preliminary data with a few subjects with tendon injuries supports this idea (data not shown).

As stated earlier, recordings of sEMG from leg muscles of healthy subjects, as well as from injured individuals with tendinopathy, are not without problem, and the interpretation of



such data should not be made without careful consideration. In Figures 1 & 2 we identified a number of factors that can affect sEMG recordings, and in terms of running/jogging and tendinopathy perhaps the most important would be as follows;

Fig. 1: **Causative** – motor point, tendon, active motor units, motor unit firing rate, acid build-up & blood flow

**Intermediate** – conduction velocity

**Deterministic** – active motor units, motor unit firing rate, detected motor units, motor unit action potential amplitude, motor unit action potential duration, recruitment & stability

Fig. 2: Intra-muscular temperature, conduction velocity of muscle fibres & blood flow

In the individuals examined in this section, without doubt the biggest issues to be addressed in terms of a stable sEMG recording are those of lead movement and the release of sweat, which affects electrode adhesion and stable localization. We have sought to reduce lead movement and signal noise arising from the recording cables swinging and moving during running/jogging by asking participants to wear net stockings which serve to hold the cables in place close to the leg and thereby prevent lead movement noise. The second issue of sweat accumulation under the recording electrodes as individuals begin to warm up during the 60 minute run/jog is not so easily solved. We have chosen to use a self-adhesive and elastic tape to ensure that the recording electrodes do not move appreciably, and have cooled the environment in which the run/jog takes place. Nevertheless, in some instances these precautions are not adequate and a degree of sEMG signal noise is unavoidable.

Among the other confounding factors affecting the sEMG signal are parameters closely associated with the level and duration of the exercise itself. As metabolites are used by contracting fibres there is an inevitable acidification close to the muscle membrane which has the advantage of initially improving blood flow through the displacement of  $K^+$ , which serves to induce vasodilatation. However, it is known that a build up of  $H^+$  ions close to muscles results in a loss of excitability. Thus a period of exercise over time will affect such parameters as the number of active motor units, the blood flow, and the intra-muscular temperature – the later having an impact on the conduction velocity of muscle fibres.

#### 4. General discussion

As already stated at the start of this manuscript, surface electromyography as a technique offers a safe, quick, pain-free, non-invasive and repeatable way of assessing the physiological processes which cause muscles to generate force and produce movement. It is for this reason that it must be seen as “*a tempting muse*”. As to the critique that “*it is too easy to use and consequently too easy to abuse*” this is surely unfair. It seems to us that if sEMG is being abused as a technique then it is because those using the technique do not fully understand the factors comprising the recorded signal and, as a consequence, do not take the time to assess their results in a critical fashion. One would rarely make a diagnosis of an illness based on just one symptom – the patient has a fever so it must be malaria – and in just the same way, sEMG should be used as a technique alongside other forms of assessing physical function e.g. circulation, muscle strength, Quality of Life etc.

The strength of ones scientific judgement lies surely with a detailed knowledge of any particular technique. In this way, a working understanding of the sEMG signal, its low and high frequency spectrum, and the causes of noise and artefacts, can only serve to reduce the

risk of abuse of this technique. Used alongside other assessment tools for judgment of an individual's health, sEMG will support and help towards validation of a diagnosis, and hopefully give directions towards the optimal form of treatment. In the case of tendinopathy, the described tendon artefact suddenly changes from being an artefact – which is something often considered as disturbance – to becoming the reliable measure for tendon condition.

One should not, however, ignore the fact that surface electromyography has a number of limitations that must be understood and corrected for, if meaningful measurements are to be obtained (De Luca, 1993; Hermens et al., 1999, Seniam 1999). In a memorial lecture at the International Society for Biomechanics in 1993, Carlo J. De Luca clearly outlined the inherent problems associated with surface electromyography and provided recommendations for electrode configuration, placement, signal sampling and recording (De Luca, 1993). Later a complete set of guide lines were given by the SENIAM initiative (Hermens et al., 1999). In the examples given here, these guidelines were followed closely in both studies.

Besides the technical aspects of ensuring an accurate sEMG signal recording from a muscle, one should also consider the biological aspects that affect signal parameters, most of which are beyond the recorders control. Here one should pay particular attention to factors that might cause muscle fatigue/weakness as a result of a progressive illness (e.g. uremia) or as the consequence of a period of exercise (e.g. running/jogging).

The aetiology of muscle fatigue is a particularly important question, as the losses in force, velocity and power that define fatigue often lead to serious limitations in muscle and whole body performance (Fitts, 1994). Fatigue results from the effects of multiple factors, which makes unequivocal identification of causative agents a difficult task. It is, however, known that force production is inhibited by a build-up in both  $P_i$  and  $H^+$ , two products that are known to change with a period of intense exercise with  $P_i$  increasing from 5 to 30 or 40 mM, and intracellular pH declining from 7.0 to 6.2 (Thompson & Fitts 1992; Fitts, 1994). Moreover, such changes not only affect force production in fast-twitch muscles, they also reduce cross-bridge interactions due to a changed ionic environment (Metzger & Moss, 1990), leading to a fatigue-induced drop in tension. In uremic patients, a clear reduction in phosphate is often noted after dialysis (48% reduction  $P < 0.01$ ) from a value of 1.85 mmol/l (normal adult range of 0.8-1.5 mmol/l). It is also known that alterations in sarcolemma function induce muscle fatigue by preventing cell activation (Sjøgaard, 1990). Indeed, it has been shown that exposure of muscles to a high extracellular  $K^+$  concentration gives rise to depolarization of muscle fibres and results in a loss of contractility (Fitts, 1994), particularly when this is associated with a simultaneous reduction in extracellular  $Na^+$  concentration (Nielsen & Overgaard, 1996).

In an earlier study by the authors investigating recorded interference EMG signals in the hand muscle *2nd dorsal interosseous* of uremic patients under a period of voluntary contraction, it was shown that a significant increase in the deterministic factor “mean signal frequency” occurred when one compared post- to pre-dialysis values (Harrison et al., 2002). These findings, which indicate an inhibitory effect of the uremic state prior to dialysis on the number of recordable events from this particular muscle, are unlikely to be affected by such problems as detection volume of the recording electrodes or by cross-talk from adjacent muscles, as the size and position of this muscle would exclude such issues. It therefore seems realistic to assume, that a haemodialysis session removes some form of inhibition at the level of; 1) the motor nerve e.g.  $K^+$  or so called “middle molecules” with a molecular weight range of 500-2000 Daltons (Bostock et al., 2004), 2) the motor endplate, and 3) the muscle fibre, or any combination of these three. Moreover, the aforementioned study found that one of the largest changes in plasma values in patients undergoing dialysis was that of inorganic phosphate.

The exact mechanism of  $P_i$  on force production remains to be elucidated but one might speculate that the increased myoplasmic  $P_i$  may decrease force production by direct action on cross-bridge function, or change the distances in the filament lattice due to different ion binding to the filaments (Naylor et al., 1985; Bartels et al., 1985), alternatively increased  $P_i$  may inhibit the ATP driven sarcoplasmic  $Ca^{2+}$  uptake, such that less  $Ca^{2+}$  would be available for release, leading to a decline in force (Duke & Steele, 2000).

In the case of studies involving uremic patients this change in distribution of ions over the cell membrane between dialyses may very well affect the measured sEMG, showing most effect of training right after dialysis. Since each patient is assessed against themselves, this should not affect the overall effect of training, although the true maximal effect may be higher than that seen here.

Likewise, the changes seen in the runners/joggers over a 60 minute exercise period may very well be due to changes in ion balance over the muscle cell membrane. However, whilst this is a valid point with regard to muscle measurements, it has no importance in terms of the use of bipolar recording electrodes to assess tendon function, since this constitutes an artefact, which is independent of a change in frequency and/or amplitude.

## 5. Conclusions

What can be safely concluded from these results are that direct sEMG recordings can be used as part of an assessment package for weak patient groups with musculo-skeletal problems due to the following two facts. First, that sEMG is quick and painless, making the method well tolerated by weaker patient groups like the uremic patients. Although the method at present is not very specific, it may still be preferential if a clear procedure for measurements and interpretation of data can be set up. The areas to consider are the noise issues, cross-talk between fibres and muscle selection, electrode placement etc. Second, that sEMG is cheap and easily available in a clinical setting, as long as clear guidelines about measurements, data handling and interpretation for the selected setting are available.

Moreover, sEMG represents a trustworthy and reliable means of gaining an insight into muscle function/dysfunction, particularly when used in conjunction with other diagnostic tools.

In the case of uremic patients, sEMG correlates well with such forms of functional assessment as the “stand chair test” and the “Quality of Life Questionnaire – KDQOL-SF-36” (Heaf et al., 2010), and during periods of exercise training there is a significant effect on sEMG frequency and amplitude, indicating a clear improvement in coordination.

As for tendinopathy, it can be concluded from the present results that the application of non-invasive surface electromyography on both heads of *m. Gastrocnemius* during a period of jogging on a treadmill not only shows the initial phase of muscle synchronization as an athlete warms up, and the relative contributions made by the medial and lateral heads of this muscle, it also gives an insight into Achilles tendon activity when combined with a differential recording above the point of insertion to the calcaneus. Furthermore, used with understanding and alongside other diagnostic tools, this technique has the potential to extend our understanding of the active participation of tendons in the tension transfer from muscle to bone.

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## 7. References

- Alfredson, H.; Ljung, B.O.; Thorsen, K. & Lorentzon, R. (2000) In vivo investigation of ECRB tendons with microdialysis technique – no signs of inflammation but high amounts of glutamate in tennis elbow. *Acta Orthopædica Scandinavica*, Vol. 71, pp. 475-479.
- Allison, G.T. & Purdam, C. (2009). Eccentric loading for Achilles tendinopathy – strengthening or stretching? *British Journal of Sports Medicine*, Vol. 43, pp. 276-279.
- Bartels, E.M. & Elliott, G.F. (1985). Donnan potentials from the A- and I-bands of glycerinated and chemically-skinned muscles, relaxed and rigor. *Biophysical journal*, Vol. 48(1), pp. 61-76.
- Bernardi, M.; Solomonow, M.; Nguyen, G.; Smith, A & Baratta, R. (1996). Motor unit recruitment strategy changes with skill acquisition. *European Journal of Applied Physiology and Occupational Physiology*, Vol. 74, pp 52-59.
- Bernardi, M.; Felici, F.; Marchetti, M.; Montellanico, F.; Piacentini, M.F. & Solomonow, M. (1999). Force generation performance and motor unit recruitment strategy in muscles of contralateral limbs. *Journal of Electromyography and Kinesiology*, Vol. 9, pp. 121-130.
- Cook, J. (2009). In search of the tendon holy grail: predictable clinical outcomes. *British Journal of Sports Medicine*, Vol. 43, p. 235.
- Couppé, C.; Hansen, P.; Kongsgaard, M.; Kovanen, V.; Suetta, C.; Aagaard, P.; Kjaer, M. & Magnusson, S.P. (2009) Mechanical properties and collagen cross- linking of the patellar tendon in old and young men. *Journal of Applied Physiology*, Vol. 107, pp. 880-886.
- Day, S.J. & Hullinger, M. (2001). Experimental simulation of cat electromyogram: Evidence for algebraic summation of motor-unit action-potential trains. *Journal of Neurophysiology*, Vol. 86, pp. 2144-2158.
- De Luca, C.J. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics*, Vol. 13, pp. 135-163.
- De Luca, C.J.; Gilmore, L.D.; Kuznetsov, M. & Roy, S.H. (2010). Filtering the surface EMG signal: Movement artifact and baseline noise contamination. *Journal of Biomechanics*, Vol. 43, pp. 1573-1579.
- Ekholm, O.; Kjølner, M.; Davidsen, M.; Hesse, U.; Eriksen, L.; Christensen, A.I. & Grønbaek, M. (2006). Sundhed og sygelighed i Danmark 2005 & udviklingen siden 1987. København: Statens Institut for Folkesundhed.
- Farina, D.; Merletti, R.; Enoka, R.M. (2004). The extraction of neural strategies from the surface EMG. *Journal of Applied Physiology*, Vol. 96, pp. 1486-1495.
- Farris, D.J.; Trewartha, G. & McGuigan, M.P. (2011). The effects of a 30-min run on the mechanics of the human Achilles tendon. *European Journal of Applied Physiology*. DOI: 10.1007/s00421-011-2019-8.
- Fredberg, U. & Stengaard-Pedersen, K. (2008). Chronic tendinopathy: tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. *Scandinavian Journal of Medicine and Science in Sports*, Vol. 18(1), pp. 3-15.
- Gaida, J.E.; Cook, J.L. & Bass, S.L. (2008). Adiposity and tendinopathy. *Disability and Rehabilitation*, Vol. 30(20-22), pp. 1555-1562.

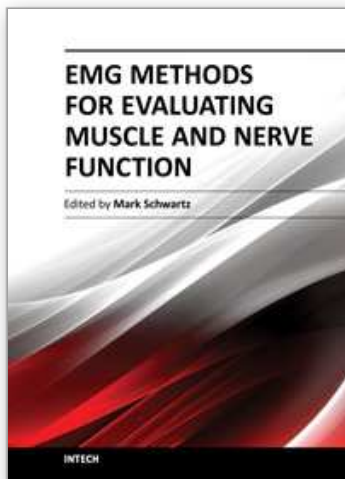


- Gerdle, B.; Karlsson, S.; Crenshaw, A.G. & Friden, J. (1997). The relationships between EMG and muscle morphology throughout sustained static knee extension at two submaximal force levels. *Acta Physiologica Scandinavica*, Vol. 160:, pp. 341–351.
- Gerdle, B.; Wretling, M.L. & Henriksson-Larsen, K. (1988). Do the fibre-type proportion and the angular velocity influence the mean power frequency of the electromyogram. *Acta Physiologica Scandinavica* Vol. 134, pp. 341-346.
- Gerdle, B.; Henriksson-Larsen, K., Lorentzon, R. & Wretling, M.L. (1991). Dependence of the mean power frequency of the electromyogram on muscle force and fibre type. *Acta Physiologica Scandinavica*, Vol. 142, pp 457-465.
- Glazebrook, M.A.; Wright, J.R. Jr; Langman, M.; Stanish, W.D. & Lee, J.M. (2008). Histological analysis of Achilles tendons in an overuse rat model. *Journal of Orthopedic Research* Vol. 26, pp. 840-846.
- Harrison, A.P. & Flatman JA. (1999). Measurement of force and both surface and deep M wave properties in isolated rat soleus muscles. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, Vol. 277, pp. R1646–R1653.
- Harrison, A.P.; Flatman, J.A.; Nielsen, A.H.; Eidemak, I.; Mølsted, S. & Unmack, M.A. (2002). Integrated surface electromyography (EMG) analysis in m. vastus lateralis and m. 2. dorsal interosseous in uremic patients. *Journal of Muscle Research and Cell Motility*, Vol. 23, p. 29.
- Harrison, A.P.; Nielsen, A.H.; Eidemak, I.; Molsted, S. & Bartels, E.M. (2006). The uremic environment and muscle dysfunction in man and rat. *Nephron Physiology* , Vol. 103(1), pp. 33-42.
- Heaf, J.G.; Mølsted, S.; Harrison, A.P.; Eiken, P.A.; Prescott, L. & Eidemak, I. (2010). Vitamin D, Surface Electromyography and Physical Function in Uraemic Patients. *Nephron*, Vol. 115, pp. c244-c250.
- Henneman, E.; Somjen, G. & Carpenter, D.O. (1965). Functional significance of cell size in spinal motor neurons. *Electroencephalography and Clinical Neurophysiology*, Vol. 19, pp. 533-580.
- Hermens, H.J.; Freriks, B.; Merletti, R.; Stegeman, D.; Blok, J.;, Rau, J.; Disselhorstklug, C. & Hâgg, G. (1999). SENIAM 8 - european recommendations for surface electromyography. *Roessingh Research and Development b.v.*, Enschede, The Netherlands, 1999.
- Hess, G.W. (2010). Achilles tendon rupture: a review of etiology, population, anatomy, risk factors, and injury prevention. *Foot Ankle Speciality*. Vol. 3(1), pp. 29-32.
- Hogrel, J.Y. (2005). Clinical applications of surface electromyography in neuromuscular disorders. *Clinical Neurophysiology*, Vol. 35, pp. 59-71.
- Holm, L.; van Hall, G.; Rose, A.J.; Miller, B.F.; Doessing, S.; Richter, E.A. & Kjaer, M. (2009). Contraction intensity and feeding affect collagen and myofibrillar protein synthesis rates differently in human skeletal muscle. *American Journal of Physiology Endocrinology and Metabolism*, Vol. 298, pp. E257-E269.
- Ishikawa, M.; Pakaslahti, J. & Komi, P.V. (2007). Medial gastrocnemius muscle behavior during human running and walking. *Gait and Posture*, Vol. 25, pp. 380-384.
- Järvinen, T.A.; Kannus, P.; Paavola, M.; Järvinen, T.L.; Józsa, L. & Järvinen, M. (2001). Achilles tendon injuries. *Current Opinion in Rheumatology*, Vol. 13(2), pp. 150-155.
- Karlsson, S. & Gerdle, B. (2001). Mean frequency and signal amplitude of the surface EMG of the quadriceps muscles increase with increasing torque – a study using the continuous wavelet transform. *Journal of Electromyography and Kinesiology*, Vol. 11, pp. 131-140.



- Keenan, K.G.; Farina, D.; Maluf, K.S.; Merletti, R. & Enoka, R.M.(2005). Influence of amplitude cancellation on the simulated surface electromyogram. *Journal of Applied Physiology*, Vol. 98, pp. 120-131.
- Keenan, K.G.; Farina, D.; Merletti, R. & Enoka, R.M. (2006). Amplitude cancellation reduces the size of motor unit potentials averaged from the surface EMG. *Journal of Applied Physiology*, Vol. 100, pp. 1928-1937.
- Ker, R.F.; Wang, X.T. & Pike, A.V.L. (2000). Fatigue quality of mammalian tendons. *Journal of Experimental Biology*, Vol. 203, pp. 1317-1327.
- Komi, P.V. & Tesch, P. (1979). EMG frequency-spectrum, muscle structure, and fatigue during dynamic contractions in man. *European Journal of Applied Physiology and Occupational Physiology*, Vol. 42, pp. 41-50.
- Krishnan, A.V.; Phoon, R.K.S.; Pussell, B.A.; Charlesworth, J.A.; Bostock, H. & Kiernan, M.C. (2005). Altered motor nerve excitability in end-stage kidney disease. *Brain*, Vol. 128, pp. 2164-2174
- Kupa, E.J.; Roy, S.H.; Kandarian, S.C. & Deluca, C.J. (1995). Effects of muscle-fiber type and size on EMG median frequency and conduction-velocity. *Journal of Applied Physiology*, Vol. 79, pp. 23-32.
- Langberg, H.; Ellingsgaard, H.; Madsen, T.; Jansson, J.; Magnusson, S.P.; Aagaard, P. & Kjaer, M. (2007). Eccentric rehabilitation exercise increases peritendinous type I collagen synthesis in humans with Achilles tendinosis. *Scandinavian Journal of Medicine and Science in Sports*, Vol. 17, pp.61-66.
- Larsson, B.; Andersen, J.L.; Kadi, F.; Bjork, J. & Gerdle, B. (2002). Myosin heavy chain isoforms influence surface EMG parameters: a study of the trapezius muscle in cleaners with and without myalgia and in healthy teachers. *European Journal of Applied Physiology*, Vol. 87, pp. 481-488.
- Larsson, B.; Kadi, F.; Lindvall, B. & Gerdle, B. (2006). Surface electromyography and peak torque of repetitive maximum isokinetic plantar flexions in relation to aspects of muscle morphology. *Journal of Electromyography and Kinesiology*, Vol. 16, pp. 281-290.
- Lichtwark, G.A.; Bougoulais, K. & Wilson, A.M. (2007). Muscle fascicle and series elastic element length changes along the length of the human gastrocnemius during walking and running. *Journal of Biomechanics*, Vol. 40, pp. 157-164.
- Linssen, W.H.J.P.; Stegeman, D.F.; Joosten, E.M.G.; Binkhorst, R.A.; Merks, M.J.H.; Terlaak, H.J. & Notermans, S.L.H. (1991). Fatigue in type-I fiber predominance – a muscle force and surface EMG study on the relative role of type-I and type-II muscle-fibers. *Muscle & Nerve*, Vol. 14, pp. 829-837.
- Maffulli, N. & Longo, U.G. (2008). How do eccentric exercises work in tendinopathy? An Editorial. *Rheumatology*, Vol. 47, pp. 1444-1445.
- Maganaris, C.N. & Paul, J.P. (2002). Tensile properties of the in vivo human gastrocnemius tendon. *Journal of Biomechanics*, Vol. 35, pp. 1639-1646.
- Merletti, R.; Knaflitz, M. & De Luca, C.J. (1992). Electrically evoked myoelectric signals. *Critical Reviews in Biomedical Engineering*, Vol. 19, pp. 293-340.
- Meyer, A.; Tumilty, S. & Baxter, G.D. (2009). Eccentric exercise protocols for chronic non-insertional Achilles tendinopathy: how much is enough? A Review. *Scandinavian Journal of Medicine and Science in Sports*, Vol. 19, pp. 609-615.
- Milnerbrown, H.S. & Miller, R.G. (1986). Muscle membrane excitation and impulse propagation velocity are reduced during muscle fatigue. *Muscle & Nerve*, Vol. 9, pp. 367-374.

- Molsted, S.; Eidemak, I.; Sorensen, H.T. & Kristensen, J.H. (2004). Five months of physical exercise in hemodialysis patients: Effects on aerobic capacity, physical function and self-rated health. *Nephron*, Vol. 96(3), pp. c76-c82.
- Molsted, S.; Eidemak, I.; Sorensen, H.T.; Kristensen, J.H.; Harrison, A.P. & Andersen, J.L. (2007). Myosin heavy-chain isoform distribution, fibre-type composition and fibre size in skeletal muscle of patients on haemodialysis. *Scandinavian Journal of Urology and Nephrology*, Vol. 41(6), pp. 539-545.
- Moritani, T.; Nagata, A. & Muro, M. (1982). Electro-myographic manifestations of muscular electromyography. *Medicine and Science in Sports and Exercise*, Vol. 14, pp. 198-202.
- Morrissey, D.; Roskilly, A.; Twycross-Lewis, R.; Isinkaye, T.; Screen, H.; Woledge, R. & Bader, D. (2011). The effect of eccentric and concentric calf muscle training on Achilles tendon stiffness. *Clinical Rehabilitation*, Vol. 25, pp. 238-247.
- Naylor, G.R.S.; Bartels, E.M.; Bridgman, T.D. & Elliott, G.F. (1985). Donnan potential changes in rabbit psoas muscle in rigor. *Biophysical Journal*, Vol. 48(1), pp. 47-60.
- Olesen, J.L.; Heinemeier, K.M.; Langberg, H.; Magnusson, S.P.; Kjaer, M. & Flyvbjerg, A. (2006). Expression, content, and localization of insulin-like growth factor I in human achilles tendon. *Connective Tissue Research*, Vol. 47, pp. 200-206.
- Perry, S.M.; McIlhenny, S.E.; Hoffman, M.C. & Soslowsky, L.J. (2005). Inflammatory and angiogenic mRNA levels are altered in a supraspinatus tendon overuse model. *Journal of Shoulder and Elbow Surgery*, Vol. 14(1 Suppl S), pp. 79S-83S.
- Scott, A.; Cook, J.L.; Hart, D.A.; Walker, D.C.; Duronio, V. & Khan, K.M. (2007). Tenocyte responses to mechanical loading in vivo: a role for local insulin-like growth factor 1 signaling in early tendinosis in rats. *Arthritis and Rheumatology*, Vol. 56, pp. 871-881.
- Simpson, M.R. & Howard, T.M. (2009). Tendinopathies of the Foot and Ankle. *American Family Physician*, Vol. 80, pp. 1107-1114.
- Solomonow, M.; Baten, C.; Smit, J.; Baratta, R.; Hermens, H.; Dambrosia, R. & Shoji, H. (1990). Electromyogram power spectra frequencies associated with motor unit recruitment strategies. *Journal of Applied Physiology*, Vol. 68, pp. 1177-1185.
- Soslowsky, L.J.; Carpenter, J.E.; DeBano, C.M.; Banerji, I. & Moalli, M.R. (1996). Development and use of an animal model for investigations on rotator cuff disease. *Journal of Shoulder Elbow Surgery*, Vol. 5, pp. 383-392.
- Soslowsky, L.J.; Thomopoulos, S.; Esmail, A.; Flanagan, C.L.; Iannotti, J.P.; Williamson, J.D. 3<sup>rd</sup> & Carpenter, J.E. (2002). Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. *Annals of Biomedical Engineering*, Vol. 30, pp. 1057-1063.
- Tesch, P.A.; Komi, P.V.; Jacobs, I.; Karlsson, J. & Viitasalo, J.T. (1983). Influence of lactate accumulation on EMG frequency-spectrum during repeated concentric contractions. *Acta Physiologica Scandinavica*, Vol. 119, pp. 61-67.
- Turker, K.S. (1993). Electromyography – some methodological problems and issues. *Physical Therapy*, Vol. 73, pp. 698-710.
- Tygesen, M.P. & Harrison, A.P. (2005). Nutritional restriction in utero programs postnatal muscle development in lambs. *Animal Science Journal*, Vol. 76, pp. 261-271.
- Van der Worp, H.; van Ark, M.; Roerink, S.; Pepping, G.-J.; van den Akker-Scheek, I. & Zwerver, J. (2011). Risk factors for patellar tendinopathy: a systematic review of the literature. *British Journal of Sports Medicine*, Vol. 45, pp. 446-452.



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This first of two volumes on EMG (Electromyography) covers a wide range of subjects, from Principles and Methods, Signal Processing, Diagnostics, Evoked Potentials, to EMG in combination with other technologies and New Frontiers in Research and Technology. The authors vary in their approach to their subjects, from reviews of the field, to experimental studies with exciting new findings. The authors review the literature related to the use of surface electromyography (SEMG) parameters for measuring muscle function and fatigue to the limitations of different analysis and processing techniques. The final section on new frontiers in research and technology describes new applications where electromyography is employed as a means for humans to control electromechanical systems, water surface electromyography, scanning electromyography, EMG measures in orthodontic appliances, and in the ophthalmological field. These original approaches to the use of EMG measurement provide a bridge to the second volume on clinical applications of EMG.

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