The Application of sEMG in Aging: A Mini Review

Gennaro Boccia^{a, b} Davide Dardanello^a Valeria Rosso^a Luisa Pizzigalli^a
Alberto Rainoldi^a

^aMotor Science Research Center, School of Exercise and Sport Sciences, SUISM, Department of Medical Sciences, University of Turin, Torino, and ^bCeRiSM Research Center 'Sport, Mountain, and Health', University of Verona, Rovereto, Italy

Key Words

Innovation · Surface electromyography · Elderly neuromuscular assessment

Abstract

The aim of this mini-review is to describe the potential application of surface electromyography (sEMG) techniques in aging studies. Aging is characterized by multiple changes of the musculoskeletal system physiology and function. This paper will examine some of the innovative methods used to monitor age-related alterations of the neuromuscular system from sEMG signals. A description of critical assumptions which underlie some of these approaches is emphasized. The first part focuses on the evolution of the recording techniques and describes some methodological issues. The second part focuses on how to use the following techniques to characterize aging: amplitude and spectral sEMG signal analysis, muscle fiber conduction velocity estimation, and myoelectric fatigue assessment. The last part describes a number of advanced sEMG approaches which seem promising in the geriatric population to estimate motor unit number, size, recruitment thresholds, and firing rates.

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Introduction

During daily activities, a distribution of electric potentials is available over the skin surface. They are produced by the membrane depolarization which allows muscle contraction, and are related to the pool of recruited motor units (MUs) as a consequence of the requested force output. To record such surface electromyography (sEMG) signals is quite easy, but potentially misleading [1] if the procedure is not properly followed. The recent European project SENIAM (Surface EMG for a Non-Invasive Assessment of Muscles) answered to such a possible drawback providing a number of parameters and procedures to properly record sEMG signals [2, 3]. This short review aims to briefly inform the reader of the latest developments in this field and to provide possible applications of interest in aging.

Evolution of Recording Techniques

Bipolar configuration is an easy and fast way to record sEMG signals. In the last decade, however, a lot of different devices have been designed based on the awareness that a greater number of electrodes results in more points of view of traveling action potentials. In fact, the redun-

Fig. 1. Differential recording from a healthy biceps brachii contracting at about 50% of the MVC. A linear electrode array of 16 electrodes with an interelectrode distance of 1 cm is applied in the direction of the fibers. A large amount of information can be obtained from the visual analysis of the signals as reported in many publications [6]. The location of the IZ is evident under the electrode 7 as the channel with lower amplitude and the point of inversion of the propagation of the detected action potentials. The attenuation of the signal at the extremes (channels 14 and 15) of the array indicates the reaching of the tendon zones (the so called 'end-of-fiber effects'). The propagation of MUAPs is evident from the IZ to the tendons. The two sections of the signal array showing potential propagation allow estimation of CV based on the temporal delay separating potentials detected at different spatial locations.

dancy in spatial recording facilitates increases the available techniques for the analysis of these signals. Linear electrode arrays allow us to localize innervation zones (IZs) and tendon terminations in a noninvasive way [3], estimate muscle fiber conduction velocity (CV) with a narrower error with respect to the traditional double differential montage, recognize different MU action potentials (MUAPs) on the bases of their shape and CV [4], and properly record sEMG signals both in static and dynamic conditions (fig. 1) [5]. In the last years, this evolution moved to bidimensional electrodes, i.e. matrixes of pins which, with different shapes and technological solutions, allow the recording of sEMG signals from up to hundreds of electrodes and to transform them into colored maps showing the level of activity in different muscle areas in real time [6]. This technique, by using multiple closely spaced electrodes overlying an area of the skin, provides spatial distribution of the electrical activity over the muscle. It thus opens new possibilities to study muscle characteristics and places this tool in the category of the imaging techniques which are transforming the amount and quality of information currently available.

Where to Place the Electrodes

The physical region where the motor neuron terminations and muscle fibers systems connect through the neuromuscular junctions (or end plates) is called the IZ

[7]. From there, action potentials travel in opposite directions toward the tendon terminations and generate propagating potentials over the skin (fig. 1). The technique of the multichannel montage has provided the necessary redundancy to highlight such an anatomical area which was now clearly identified as the *worst site* to place a pair of recording electrodes [3]. Moreover, during dynamic contractions, the relative movement of the muscle with respect to the skin (i.e. the electrode system) determines a strong alteration of the signal when the IZ shifts under the electrode pair [8]. Hence, electrodes must be placed between the IZ and tendon terminations to assure the recording of meaningful signals. Since, for the moment, high-density EMG recording techniques are available almost exclusively in research laboratories, practitioners can refer to papers and books to obtain guidelines to find the best electrode placement on the most superficial muscles [2, 3]. A further critical topic related to electrode positioning is the reciprocal orientation between fiber direction and electrodes. Traditional bipolar configuration needs electrodes to be placed parallel to muscle fiber and such a position can be carefully obtained by checking for the highest EMG signal amplitude. Otherwise, innovative electrode designs have been proposed; bipolar concentric electrode, for instance, can be used without having to worry about muscle fiber direction due to their intrinsic isotropy [7].

sEMG Amplitude

It is well known that the larger the requested force output, the greater the amplitude of the sEMG signal [1]. The most common descriptors of sEMG signal amplitude are the average rectified value and the root mean square value. The sEMG amplitude can be detected with a single pair of electrodes in bipolar configuration. It provides an overall estimation of muscle activation since it is related to the number of MUs recruited and their discharge frequency [4]. However, the relation between force exerted and sEMG amplitude is not necessarily linear since a number of confounding factors influence the raw sEMG signal recorded over the skin, e.g. the amplitude cancellation, which is the cancellation of positive and negative phases of MUAPs, and the distribution of active MUs within the muscle, among others [9].

Maximal muscle strength, power, and rate of force development decrease with aging. The absolute value of sEMG amplitude during maximal voluntary contraction (MVC) shows the same trend, being lower in elderly compared with young and middle-aged adults [10]. These differences are related to lower MU recruitment and discharge rates, but the roles played by these two features cannot be distinguished with bipolar sEMG [9]. Moreover, the comparison between young and elderly subjects using the absolute values of sEMG amplitude should be applied with caution. The higher subcutaneous adipose tissue usually detected in elderly compared with young can affect the measurements with misleading findings [9, 10]. The thickness of subcutaneous fat layers should be measured with ultrasound imaging. Only when these measures are the same between populations is it possible to compare the absolute values of sEMG amplitude estimates. Otherwise only relative variations can be meaningfully detected. For example normalizing the sEMG amplitude obtained during submaximal contractions with respect to the amplitude obtained during an MVC can provide an estimate of the relative muscle activation. With due caution and in particular experimental setups, this percentage value can be used to compare different subjects and populations [2].

The decreasing agonist muscle activation cannot be the sole cause for the lower torque production in elderly. Some authors [11] have observed a relative increase of sEMG amplitude of antagonist muscles during isometric and dynamic contractions in older adults. This phenomenon, which is referred to as cocontraction, may serve to protect and stabilize the joints during forceful and balance tasks. The increase of coactivation with aging could

be an additional explanation for the age-related decline in net force production [10].

A number of studies have documented that strength training elicits significant changes in muscle strength in elderly individuals, even in subjects over 90 years of age. The maximal strength gains is accompanied by significant increases in sEMG amplitude of the agonist muscles, recorded in both isometric and dynamic actions, suggesting an increase in the magnitude of voluntary neuromuscular activity [12]. Moreover, elderly individuals showing elevated coactivation before training, typically decrease the antagonist activation following strength training [12].

The decrease of neuromuscular power and rate of force development with age have been shown to have severe consequences, such as increasing the risk of fall and disability, and decreasing autonomy maintenance [13]. In fact, it has been demonstrated that in elderly people functional performance is more related to neuromuscular power than to maximal muscle strength. Unfortunately, the decrease in muscle power with age is bigger than the decrease in maximal strength, and declines are more pronounced from 70 to 90 years [14]. There are many fundamental motor tasks which have reported this kind of decrease, such as concentric contractions, explosive isometric contractions, and jumps. This is due to a decrease in maximal discharge frequency of MUs and in the percentage of MUs which exhibit doublets (i.e. a double discharge at intervals <5 ms) [15]. It is well documented that maximal muscle power increases after strength training in elderly subjects [16]. These training adaptations produce an increase in sEMG amplitude [11]. In response to progressive resistance training, both young and elderly subjects exhibit increased levels of magnitude and onset-rate sEMG in the initial 200 ms of rapid contractions [17]. These modifications represent an increase in rapid MU firing, an earlier recruitment of MUs, and probably also an augmented MU synchronization.

Frequency Spectrum

The power spectrum of EMG signal is in the frequency range of 10–400 Hz. During sustained fatiguing contractions the power spectrum changes as a sign of myoelectric manifestations of muscle fatigue. Many variables have been defined and discussed in the literature with the aim to quantify this change [18], but the most widely used are the mean frequency and the median frequency. They are related to a number of physiological factors including the CV of the MUAP along the fibers [9]. The

frequency content of the signal is generally considered to be related to the recruitment of different MU types on the base of the following scheme: the faster the recruited MUs, the higher the mean/median frequency of the recorded signal. Thus, mean/median frequency time course during fatiguing contractions should show a steeper decrease if the muscle is characterized by a higher proportion of fast fibers (type II) than slow fibers (type I). The rate of decrease of power spectrum parameters has been observed to be greater in young people than in elderly people during submaximal isometric contractions in many experimental setups [19]. This observation supports the bioptic evidence according to which elderly muscles undergo a shift toward type I muscle fibers. Nevertheless, a recent debate described a number of confounding factors (fiber length, depth of MU within the volume conductor, volume conductor low-pass filtering effect, etc.) which prevent claiming that spectral properties of the sEMG signal can provide information about MU recruitment and muscle fiber type [20]. In conclusion, when only bipolar configuration is available, spectral variables can be used to provide an overall index of muscle fatigue. Nevertheless, when multichannel sEMG is available, muscle fiber CV should be preferred to spectral variables.

Muscle Fiber CV

CV seems to be the most affordable candidate to relate, under a physiological framework, the modifications in EMG signals with the recruited MU pool and with histochemical characteristics of the muscle. sEMG registrations with linear array provide the possibility of estimating propagation velocity of MUAP traveling along the muscle fibers [21]. CV increases gradually when faster and larger MUs are recruited, such as when intensity muscle contraction increases [22], and it is positively related to the fiber diameter [23]. When monitoring CV throughout a wide range of contraction levels (e.g. from 20 to 80% of MVC) the increment of CV is higher in younger subjects and lower in elderly subjects. This is in agreement with bioptic studies reporting a decrease in muscle fiber size in elderly subjects when compared with young people [24]. This reduction in muscle fiber size has been shown to be fiber-type specific with the size of type II fibers decreasing by 10–40% in comparison to unchanged type I fiber size [25].

Interestingly, in a recent study investigating active elderly, CV values were found lower in vastus lateralis (VL)

than in vastus medialis obliquus (VMO) muscles during isometric contraction at 70% of MVC (unpubl. results). It is possible to argue that the decrease of muscle fiber size was more relevant in the VL than in VMO muscles. Since a preferential greater decrease of type II fiber size has been found [25], such a discrepancy between muscles could be related to their different fiber-type composition in origin. Indeed, histological studies in adults have demonstrated a significant higher proportion of type II fibers in VL muscles in comparison to VMO [26]; therefore, the decrease of muscle fiber size with age affected more the VL than the VMO since VL is the muscle with the higher proportion of type II fibers. This finding supports the hypothesis that sarcopenic effects address muscles with intensities related to their fiber-type composition and could be different even within single muscle groups, such as quadriceps. This is an important finding since allowing the investigations conducted on VL, such as bioptic assessment, could lead to overestimating the aging effects with respect to the whole quadriceps muscle.

Myoelectric Manifestations of Fatigue

Neuromuscular fatigue and fatigability are important factors in age-related phenomena. Muscle fatigue is defined as the ‘muscle’s inability to maintain an expected force’ [27]. Based on this definition, many mechanical protocols have been proposed to investigate muscle fatigue. For example, the time to task failure in a prolonged submaximal isometric contraction could offer an index of fatigability: old adults exhibit a longer time to task failure than young adults when performing a submaximal isometric contraction [28]. This counterintuitive finding is known as the ‘fatigue paradox’: old adults seems to be more fatigue resistant than young adults. Many features of elderly neuromuscular system can be attributed to this process: selective atrophy of type II fibers, slowing in the contractile properties, lower MU firing rates, and greater reliance on oxidative metabolism [28].

It is well known that changes in sEMG variables anticipate mechanical muscle fatigue from the beginning of the contraction [21]. Therefore, modifications of sEMG highlight neuromuscular fatigue before mechanical failure: this allows the estimation of indices of fatigue avoiding discomfort and danger possibly related with exhaustive efforts. We referred to myoelectric manifestations of fatigue as all the changes in sEMG variables occur during sustained muscle contractions. Since myoelectric fatigue is a multifactorial process, the ‘fatigue plot’ diagram has

Fig. 2. Representative examples of two fatigue plot diagrams: COPD patients (**a**) and healthy subjects (**b**). Time courses of average rectified value (estimates of signal amplitude: grey circles), mean spectrum power frequency (black circles), and CV (muscle fiber CV – white circles) are represented for each epoch (0.5-sec-

ond length) of the isometric contraction at 70% of MVC. Each variable is normalized with respect to its initial value, and the slope of the regression line represents an index of myoelectric fatigue. In this example, the higher myoelectric fatigue seen in COPD corresponds to higher mechanical fatigue leading to earlier task failure.

been adopted to describe the time course of sEMG variables as a whole [21]. In a fatigue plot, all myoelectric variables (mean frequency, average rectified value, and CV) are normalized with respect to their initial value. In such a way, any variation of an sEMG variable is expressed in terms of percentage change with respect to the beginning of the contraction. The time course of each variable can be interpolated with a regression line, the slope of which represents an index of fatigue.

Myoelectric manifestations of fatigue are due to changes in muscle fiber membrane excitability and MUAP propagation, and are related to alterations of muscle metabolic conditions and failure of excitation-contraction coupling (i.e. muscle activation). The changes are believed to be related to a decrease in muscle pH and are reflected by decrements of CV and spectral variables and increments of amplitude variables. The relationship between myoelectric fatigue and muscle fiber constitution has been widely investigated [5]. In general, a greater proportion of type II muscle fibers has been proposed to generate greater myoelectric manifestations of fatigue, which were measured as a greater decrease of CV and spectral variables over time. In other words, the rate of change of CV over time has been proposed to be associated with the type of fiber composition: the greater the decrease of CV over time, the higher the contribution of type II fiber in a given muscle [29].

Based on the above findings, monitoring CV during sustained isometric contraction has been proposed as a tool for noninvasive characterization of muscle fiber composition [5]. For example, elderly people (67–86 years) showed lower decrements of CV with respect to young subjects during 30 s of biceps brachii isometric contractions at 40 and 60% of MVC [30]. This is an evidence of loss of type II fiber and shift towards type I fiber in elderly muscles. Conversely, chronic obstructive pulmonary disease (COPD) patients (62–73 years) showed a higher decrease of CV with respect to matched-age healthy subjects in quadriceps isometric contractions at 70% of MVC (fig. 2) (unpubl. results). This is in agreement with the already reported loss of type I fiber and shift towards type II fiber in lower limb muscles of COPD patients observed in histological specimens. These findings lead to speculate that a protocol based on CV monitoring during fatiguing contraction is a promising tool to investigate muscle fiber composition. It is able to differentiate the effects of aging and chronic pathology in populations with well-differentiated muscle fiber composition, such as those involved in reported studies. However, the sensitivity of such a tool has yet to be determined.

When well-trained elderly people (60–85 years) were compared to well-trained young people (20–40 years) the rate of change of CV during sustained isometric contraction was not found to be different between groups [31].

Indeed, prolonged physical training has been demonstrated to specifically counterbalance the loss or hypotrophy of type II muscle fibers usually seen in sedentary elderly [32]. Hence, elderly people counterbalanced the loss of type II fibers with physical training and showed a similar muscle fiber composition as young subjects.

MU Number and Size Estimation

Determining the number of axons innervating a muscle group is important since the loss of MUs is related to the muscle weakness commonly observed in elderly. Over the past four decades, some noninvasive electrophysiological techniques have been developed to provide an accurate estimation of the number of innervating axons. In 1971, the first MU number estimation technique was proposed [33], allowing the estimation of the actual number of MUs in a muscle through the sEMG signals evoked by many motor nerve stimulations at gradually increasing intensity. However, since many MU number estimation techniques are time consuming and invasive with physical discomfort for participants, the MU number index (MUNIX) technique was proposed in 2004 (for details regarding mathematical model, see [34]). The MUNIX technique is fast (5 min) and simple. It requires the elicitation of a maximal compound action potential evoked by supramaximal nerve stimulation and the recording of sEMG at different grades of voluntary contractions (at 5 levels). MUNIX is not a physiologic count of MUs, but it allows estimating a parameter related to the number of MUs in a muscle (contrary to MU number estimation which aims to estimate the actual number of MUs). Furthermore, the MU size index (MUSIX) is also calculated. MUSIX is an index of the average size of the MUs and is based on the ratio between maximal compound action potential amplitude and the MUNIX value. It should be stressed that MUNIX and MUSIX are indices of the number and size of MUs, and are not absolute values.

The MUNIX technique can help to determine whether age-related differences in muscle strength are directly related to the number of MUs. Kaya et al. [35] used MUNIX to compare older and young adults in a pinch-grip strength test to estimate differences in abductor pollicis brevis muscle strength. The MUNIX estimate was related to pinch-grip strength among the elderly population (but not in young adults), suggesting that weakness in the elderly may be related to fewer MUs, among other factors. As a compensation for this progressive death of motor neurons in the elderly, the partial reinnervation of some

denervated muscle fibers lead to the formation of very large MUs (with more uniform muscle fiber composition). The loss of MUs accompanied by an increase in size of the remaining MUs has been detected in sarcopenic patients [36], showing a low MUNIX and high MUSIX relationship. These findings could provide the basis for further specific diagnostic tools of sarcopenic patients.

sEMG Decomposition: Matrixes of Electrodes

Nowadays, intramuscular EMG is the gold standard tool for MU analysis. However, in the last two decades, high-density electrodes have allowed a big increase in research activity in sEMG technology and signal processing, which in turn have allowed noninvasive evaluation of MU behavior [7]. A number of studies have shown how bidimensional maps of muscle activation could be used to reveal MU firing patterns [6, 37]. The recording of sEMG in multiple locations over a muscle (spatial sampling) provides the redundancy useful to recognize the shape of action potentials of different MUs within the EMG signal. One of the most promising and challenging algorithms was developed for the decomposition of the sEMG, and is called correlation kernel compensation [38]. The correlation kernel compensation method is fully automatic and is suitable for identification of MU firing patterns in isometric contractions at different levels of force (from 5 to 70% of MVC) [39]. This technique has been tested on several muscles, both in simulation and in experimental conditions, and has been proven to provide MU rate codes with good accuracy [6, 39]. In particular, a recent study has used the correlation kernel compensation method to compare the MU firing behavior of a group of healthy elderly and a group of patients affected by type 2 diabetes mellitus. In this work, the MU rate code is evaluated in the VL muscle during isometric knee extension at low force levels using high-density sEMG decomposition. The main findings of this study are that type 2 diabetes mellitus patients showed, with respect to healthy control elderly, higher variability of MU firing and a lower MU firing rate during sustained contraction [40].

Despite a number of promising scenarios of MU analysis based on high-density sEMG, this technique still suffers from limitations. High-density sEMG analysis identifies only a small proportion of the active MUs on the surface of the muscle, the application of bidimensional matrixes of electrodes is sometimes not elementary, and the quantity of data recorded is large and sometimes

needs data compression procedure. An additional limitation is that the MU analysis of high-density sEMG is still limited to isometric contractions [6, 7]. For these reasons, the use of sEMG for the analysis of the MU firing rate is not yet considered appropriate in the clinical setting.

Conclusions

This brief review examined some of the methods used for monitoring age-related alterations in the neuromuscular system from sEMG signals. The topics herein addressed comprise methods that are currently used extensively in this field, with an emphasis on the critical assumptions underlying some of these approaches. Moreover, advanced processing methods seem promising for the analysis of MU control properties, such as recruitment thresholds and firing rates.

The age-related loss in neuromuscular function comprise loss of MUs, reduction in muscle fiber number and size, changes in the maximal MU firing rate, agonist muscle activation, antagonist muscle coactivation, and force steadiness. As a consequence, mechanical muscle performance is impaired with concurrent decreases in maximal muscle strength, power, rate of force development, and exercise tolerance in the elderly. However, elderly individuals have demonstrated substantial adaptive plasticity in both skeletal muscles and neuromuscular system in response to strength training, which to a large extent can compensate for the age-related declines in muscle size and neuronal function.

Disclosure Statement

The authors declare that they have no conflicts of interest.

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