Analysis of Dynamic Voluntary Muscle Contractions in Parkinson’s Disease


Abstract—A novel method for discrimination of dynamic muscle contractions between patients with Parkinson’s disease (PD) and healthy controls on the basis of surface electromyography (EMG) and acceleration measurements is presented. In this method, dynamic EMG and acceleration measurements are analyzed using nonlinear methods and wavelets. Ten parameters capturing Parkinson’s disease (PD) characteristic features in the measured signals are extracted. Each parameter is computed as time-varying, and for elbow flexion and extension movements separately. For discrimination between subjects, the dimensionality of the feature vectors formed from these parameters is reduced using a principal component approach. The cluster analysis of the low-dimensional feature vectors is then performed for flexion and extension movements separately. The EMG and acceleration data measured from 49 patients with PD and 59 healthy controls are used for analysis. According to clustering results, the method could discriminate 80% of patient extension movements from 87% of control extension movements, and 73% of patient flexion movements from 82% of control flexion movements. The results show that dynamic EMG and acceleration measurements can be informative for assessing neuromuscular dysfunction in PD, and furthermore, they may help in the objective clinical assessment of the disease.

Index Terms—Discrimination, dynamic contractions, nonlinear methods, Parkinson’s disease (PD), surface electromyography (EMG).

I. INTRODUCTION

SURFACE electromyography (EMG) and movement measurements can be used to assess the function of human neuromuscular system. This assessment can be done by studying static (constant force and posture) and/or dynamic (varying force and posture) muscle contractions. Both kinds of studies provide different information about motor unit (MU) recruitment strategies of muscles. The most common methods to analyze static EMG are based on amplitude and Fourier-based spectral analysis, and they are used to measure the level of muscle activity and fatigue. However, the analysis of dynamic EMG can be problematic since much less is known about appropriate methods for analysis and relations between signal features and underlying physiological mechanisms [1].

In Parkinson’s disease (PD), the function of neuromuscular system is disturbed. This is due to a dopaminergic neuronal loss in the substantia nigra, which is part of the basal ganglia of the brain [2]. Simultaneous measurements of EMG and magnetoencephalographic (MEG) signals have shown that basal ganglia have a specific effect on the temporal organization of motor cortical activity during muscle contractions [3]. This leads to abnormalities observed in skeletal muscles: involuntary oscillatory movements (tremor), slowness of movements (bradykinesia), muscle rigidity and postural instability [2], and furthermore, changes in EMG and kinematic measurements [4]. The severity of PD symptoms is typically assessed using standardized rating scales such as the Unified PD Rating Scale (UPDRS). This clinical test and PD diagnosis, in general, are based on physician’s subjective observations. It is well known that PD diagnosis can be problematic (diagnostic accuracy 70% in the early stages [2]), and there is a lack of objective methods for clinical assessment of the disease. Therefore, it is important to examine the potential of EMG and movement measurements in helping the diagnosis and assessment of the disease.

Previously, there have been EMG and movement studies on static and dynamic contractions in PD. In static studies, EMG and acceleration measurements have been analyzed by using spectral methods [5], [6], and by applying nonlinear [6]–[8] and morphological methods [9], [10]. Spectral methods have shown a correlation between the low-frequency EMG power and the UPDRS motor score [5]. In addition, an increased low-frequency coherence between EMG and acceleration has been connected with PD [6]. Nonlinear methods have been used to analyze the increased level of MU synchronization in PD [7], and differences in the EMG signals between patients with PD and healthy subjects [8], [10]. In fact, the novel methods of EMG analysis (i.e., nonlinear methods and higher order moments) have shown to be more effective in analyzing PD-like EMG signals than traditional EMG analysis methods (i.e., amplitudes and median frequencies) [8]. Furthermore, a combination of nonlinear and morphological methods in analysis of EMG and acceleration measurements has proven to be effective...
in discriminating static muscle contractions between patients with PD and healthy controls [10]. However, to our knowledge, nonlinear methods have not been used to analyze dynamic EMG and acceleration measurements in PD.

Previous studies on dynamic muscle contractions in PD have concentrated on analyzing kinematic parameters and EMG burst characteristics (duration, number, magnitude, etc.) during rapid flexion and extension movements on a horizontal plane [11]–[14]. The EMG patterns measured during these movements are well understood in healthy young people. They are characterized by a triphasic EMG burst pattern with alternating agonist–antagonist EMG bursts [12]. It has been shown that for patients with PD, extension movements are slower than flexion movements [14]. In addition, extension EMGs contain more agonist bursts than flexion EMGs [14]. Furthermore, these short bursts of EMG activity cannot be normalized with practice or medication [11], [13]. With neurologically healthy subjects, no significant differences have been observed between flexion and extension movements [14]. In contrast to previous studies, here the agonist activity during free elbow flexion and extension movements in vertical direction are studied, and wavelets and nonlinear methods are used to analyze EMG burst characteristics. Previously, there have been two EMG studies where wavelet analysis has been used to analyze ballistic movements in PD [15], [16]. In these studies, it has been observed that the power distribution in EMG cross-correlation spectra, between pectoralis major and posterior deltoid muscles, appears to be broader for patients with PD than for healthy subjects.

The aim of the present study was to test the hypothesis that dynamic EMG and acceleration measurements can be used to discriminate between patients with PD and healthy controls. The EMG and acceleration measurements were analyzed using nonlinear methods and wavelets, and altogether ten parameters capturing PD-characteristic features in these signals were extracted for discrimination. The selected parameters were used to form high-dimensional feature vectors, the dimensionality of which was then reduced by using a principal component (PC) approach. Finally, cluster analysis of the feature vectors was performed in a low-dimensional space by using the obtained PCs. The method was tested with EMG and acceleration data measured from 49 patients with PD and 59 healthy controls.

II. MEASUREMENTS

A total of 108 subjects participated in EMG and movement measurements after giving their informed consent. The patient group consisted of 49 patients with PD whose age was (66 ± 9) years (mean ± SD), height was (169 ± 9) cm, weight was (75 ± 12) kg, duration of the disease was (7 ± 6) years, and UPDRS motor score was 27 ± 14. All patients had diagnosis of idiopathic PD, and they were taking medication (individual doses and combinations of L-dopa, dopa-decarboxylase inhibitors, dopamine agonists, B-type monoamine oxidase (MAO-B) inhibitors, or anticholinergics) for the condition. They did not have other diseases that could interfere with motor function. All patients were measured on-medication mainly due to the risk of inconvenient symptoms and tendency to fall. In addition, it has been shown previously that medication cannot restore the temporal pattern of EMG to normal [13], i.e., medication can decrease differences in the EMG patterns between patients with PD and healthy controls, but the differences (multiple agonist bursts) stay present. The control group consisted of 26 healthy older persons whose age was (61 ± 11) years, height (164 ± 8) cm, and weight (72 ± 11) kg, and 33 healthy younger persons whose age was (24 ± 8) years, height (174 ± 9) cm, and weight (66 ± 12) kg. The control subjects were recruited from generally healthy Finnish and Russian citizens. The study was approved by the human ethics committees of Kuopio University Hospital and Petrozavodsk State University.

During the measurement, all subjects (patients and healthy controls) were asked to flex and extend their both elbows vertically and freely in 2-s cycles with their palms up, i.e., 1 s was used for flexion and 1 s for extension. However, because of bradykinesia, all patients could not follow this rate of moving, which may have affected the measured EMG and acceleration signals. The forearms were moved with full range of motion, and at least four flexion–extension cycles were recorded for each subject. Additional weights were not used because it has been shown in earlier studies that PD characteristic EMG features are most visible in the signals of unloaded condition [8], [9].

Bipolar surface EMGs were registered continuously from the biceps brachii (BB) muscles using disposable Ag/AgCl electrodes (recording area 154 mm²; Medicotest, model M-00-S, Denmark). The recording electrodes were attached bilaterally over BB muscles. Inter electrode spacing was chosen large enough (3 cm from center to center) to catch information about the function of sufficient number of MUs. Reference electrodes were placed laterally 6–7 cm apart from the bipolar recording electrodes. Raw EMG signals were analogically bandpass-filtered (Butterworth, passband 1–500 Hz), amplified (differential amplifier, CMRR > 130 dB, total gain 1000, noise <1 μV), and A/D converted (14 bit).

Accelerations and inclinations of forearms were recorded from the palmar side of subject’s wrists using triaxial accelerometers (Mega Electronics, Ltd., range ±10g, 14-bit A/D converter) and biaxial inclinometers (Mega Electronics, Ltd., range ±90°, 14-bit A/D converter). All recordings were done using ME6000 biosignal monitor (Mega Electronics, Ltd.). The sampling rate of all signals was 1000 Hz.

III. ANALYSIS OF EMG AND ACCELERATION MEASUREMENTS

A. Preprocessing of EMG and Acceleration Signals

The flexion and extension phases of the movement were separated from the rest of data by visually inspecting measured inclinometer and accelerometer data. Four elbow flexion and extension movements per subject were chosen for analysis. The total acceleration, which was calculated as a resultant of the three acceleration components, was used for analysis. The inclinometer data were not used in further analysis.

The low-frequency trends were removed from EMG and acceleration signals using a smoothness priors method [17], which is basically a time-varying high-pass filter. The high-pass cutoff frequencies were 10 Hz for EMG and 2 Hz for acceleration. The
high-pass cutoff frequency was chosen as 2 Hz for acceleration signals because we were interested in the small-amplitude oscillation in hands and not in the low-frequency trend (< 2 Hz) that was caused by lifting the forearm up or laying it down.

B. Time-Varying Analysis

1) Nonlinear Analysis of EMG: In order to study the nonlinear dynamics of neuromuscular system, the EMG time series was mapped into a phase space using delay coordinates. According to Taken’s embedding theorem [18], this is done by forming embedding vectors

\[ u(t) = [x(t) \ x(t + \lambda) \ x(t + 2\lambda) \ldots \ x(t + (m - 1)\lambda)] \]  

where \( x(t) \) is the original time series, \( \lambda \) the delay parameter, and \( m \) the embedding dimension. These embedding vectors describe states of the dynamical system.

A parameter called the recurrence rate (%REC) can be used to measure the number of recurring states of a dynamical system. It is computed as a percentage of Euclidean embedding vector distances that are smaller than a chosen threshold level. The parameter was introduced into the analysis of physiological systems by Webber and Zbilut in 1994 [19]. We calculated the time-varying recurrence rate of EMG by dividing the signal into 250-ms-long overlapping epochs (overlap 75%) of the epoch length. The optimal epoch length for discrimination between patients and controls was chosen by gradually increasing and decreasing it. The parameter %REC was expected to be higher for patients with PD than for healthy controls because of the increased level of MU synchronization [7]. The cross-recurrence rate (%REC\(_{xy}\)) between two different time series \( x \) and \( y \) can be estimated by computing distances between the embedding vectors of the two time series. We calculated the time-varying %REC\(_{xy}\) between right- and left-side EMG signals. Both of these signals were scaled between −1 and 1 before analysis.

2) Wavelet Analysis of EMG: EMG signals were analyzed using wavelets (see [20] for reference), which can provide information about the frequency contents of signals. When compared with Fourier analysis, the wavelets can be more effective in analyzing nonstationary biosignals because of their ability to accurately detect time evolutions in frequency distribution [15]. In wavelet transform, the signal is decomposed into a set of basis functions, which are obtained by scaling and shifting the wavelet function \( \psi(t) \). The scale is denoted here with \( a \) and the shift with \( b \). The continuous wavelet transform of signal \( x(t) \) is defined as

\[ W_x(a, b) = \frac{1}{\sqrt{a}} \int_{-\infty}^{\infty} x(t) \psi^*(t - b) \frac{dt}{a} \]  

where * denotes the complex conjugate operator. The squared magnitude of the wavelet transform is called the scalogram

\[ P_x^W(a, b) = |W_x(a, b)W_x^*(a, b)|. \]  

If the wavelet transforms of two signals \( x(t) \) and \( y(t) \) are denoted with \( W_x(a, b) \) and \( W_y(a, b) \), the wavelet cross-scalogram is defined as

\[ P_{xy}^W(a, b) = |W_x(a, b)W_y^*(a, b)|. \]  

There are many possibilities to select the wavelet function for transform. In this study, we were interested in the wavelet power spectra, which is why the choice of the analyzing wavelet was not very critical [15]. The Morlet wavelet was used here for analysis, as in [15], [16], and [20]. All wavelet computations in this study were performed using MATLAB Wavelet Toolbox software (see [21] for reference).

We expected the power distribution in wavelet scalograms to be more spread for PD-like than for normal EMG signals based on [15]. When the scalograms were scaled to present the percentage of energy for each wavelet coefficient as a function of time, the maximum power was expected to be lower for patients with PD than for healthy subjects. In the cross-scalogram between right- and left-side EMG signals, the difference between PD-like and normal EMG signals was expected to be similar. We calculated the maximum power of all wavelet coefficients \( W_{\text{max}} \) as a function of time for both scalograms and cross-scalograms.

3) Acceleration Analysis: We computed two time-varying parameters from the measured acceleration signals by dividing the signals into 250-ms-long overlapping epochs (overlap 75%). One of them was the power of acceleration (\( P_{\text{acc}} \)) and the other was the sample entropy (SampEn). The power of acceleration was expected to be higher for patients with PD than for healthy controls because of tremor and rigidity observed in patients.

The sample entropy assesses the complexity of time series. It is obtained from the original time series by forming embedding vectors according to (1) and by calculating distances between these vectors. However, the distance is computed here as the maximum distance between the corresponding embedding vector components. Briefly, SampEn measures the negative natural logarithm of the conditional probability that two embedding vectors that are similar for \( m \) points are also similar for \( m + 1 \) points. The mathematical details about SampEn are described in [24]. SampEn was expected to be lower for patients with PD (more regular signals) than for healthy controls because of tremor and rigidity.

C. PC Approach

When one wants to discriminate between subjects on the basis of biosignal measurements, there are often many parameters that capture the essential features of the signal, and therefore, are potential for discrimination. The discrimination rate of a single parameter could be tested by using a receiver operating characteristic (ROC) curve, which is a graphical plot of true positive ratings versus false positive ratings as the discrimination threshold of the parameters is varied. Here, ROC curves were not used because often a combination of several parameters can work better in discrimination than a single parameter alone. However, there are two things that are important and useful to do before discrimination. First, it is important to transform the original possibly correlated parameters into uncorrelated parameters. Second, it is useful to reduce the dimension of the discrimination problem, i.e., to reduce the number of parameters. PC approach can be used for those two purposes.

In PC approach, the selected parameters (described in Section III-B) are first used to form a feature vector
z_j \in \mathbb{R}^N (N = 10)

\[ z_j = [\%REC_r \ \%REC_l \ \%REC_{rl} \ P_{acc,r} \ P_{acc,l} \ \ldots \ \text{SampEn}_r \ \text{SampEn}_l \ W_{max,r} \ W_{max,l} \ W_{max,rl}]^T \] (5)

where \%REC are the EMG recurrence rates, \( P_{acc} \) and SampEn are the powers and sample entropies of acceleration signals, respectively, and \( W_{max} \) are the EMG wavelet parameters. The subscript \( r \) denotes the right side and \( l \) the left side of the body, subscript \( rl \) is the cross-variable between right- and left-side signals, and superscript \( T \) denotes transpose. If we have \( M \) subjects, we can form a feature matrix \( Z \in \mathbb{R}^{N \times M} \) containing the feature vectors of individual subjects in its columns, i.e.

\[ Z = [z_1 \ z_2 \ \ldots \ z_M]. \] (6)

The next step in PC approach is to model the data matrix \( Z \) with a linear model

\[ Z = H\theta + v \] (7)

where \( H = [\phi_1 \ \phi_2 \ \ldots \ \phi_K] \in \mathbb{R}^{N \times K} \) is the model matrix, \( \theta = [\theta_1 \ \theta_2 \ \ldots \ \theta_M] \in \mathbb{R}^{K \times M} \) is the model weights, and \( v = [v_1 \ v_2 \ \ldots \ v_M] \in \mathbb{R}^{N \times M} \) is the model errors. In PC approach, the basis vectors \( \phi_k \) are selected to be the eigenvectors of data correlation matrix that can be estimated as \( R = (1/M)ZZ^T \). Since the eigenvectors are orthonormal, the least squares solution for the model weights \( \hat{\theta} \) is then of the form

\[ \hat{\theta} = (H^T H)^{-1} H^T Z = H^T Z. \] (8)

These weights are often called as PCs.

By choosing the number of basis vectors \( K \) smaller than the number of original parameters \( N \), the best \( K \)-dimensional orthogonal approximation for the data set is obtained. In other words, PC approach can be used to reduce the dimension of the feature vectors. In addition, obtained PCs are uncorrelated variables, and thus, PCs provide a way to discriminate between subjects in a low-dimensional space [22].

D. Cluster Analysis of PCs

There are different methods available for discrimination. In this study, we did not want to make presumptions on the subject groups that result from discrimination. That is why we used cluster analysis for discrimination. The best discriminating (between patients and controls) PCs were clustered into two groups using an iterative \( k \)-means algorithm, as described in [10]. The \( k \)-means algorithm was chosen for cluster analysis because of its conceptual and computational simplicity [23]. The aim of this study was not to present complex clustering tools but to show that there are differences between patients and controls in the EMG and acceleration measurements.

IV. RESULTS

A. Measured Signals

Typical EMG and acceleration signals measured from one patient with PD and one healthy control are presented in Fig. 1. The signals were measured during four elbow flexion–extension cycles. Note that acceleration signals were highpass filtered, and therefore, the accelerations caused by lifting the forearm up or laying it down are not observable in these signals. One can observe in Fig. 1 that the EMG signals of the patient are characterized by recurring bursts and the acceleration recordings of

Fig. 1. EMG and acceleration recordings of one patient with PD (left) and one healthy control (right) during four elbow flexion–extension cycles.
the patient by regular oscillations during the extension phases of the movement. These oscillations are likely due to muscle rigidity and/or tremor. To some extent, we could observe oscillations in the acceleration recordings of healthy subjects as well. However, in most of the cases, the oscillations were not similarly regular as in the recordings of patients.

B. Time-Varying Analysis

The time-varying parameters of EMG and acceleration, used to form the feature vectors in (1), were then computed for each subject during four flexion and extension movements, as described in Section III-B. The mean trends of these parameters for the patient and for the control group are presented in Fig. 2 (SD limits are not presented here for the sake of clarity). One can observe that there are differences in the means of time-varying parameters between the subject groups, and also between extension and flexion movements (significant group differences are described next).

Fig. 2 shows that the mean wavelet parameters ($W_{\text{max},r}$, $W_{\text{max},l}$, and $W_{\text{max},rl}$) are lower for patients than for controls through the four flexion–extension cycles. This indicates that the frequency distributions of EMG are broader for patients than for healthy controls. Similar spreading has been observed previously in the cross-correlation spectra between two different same-side muscles in [15]. In this study, the cross-correlation spectrum was computed between right- and left-side muscles. Fig. 2 also shows that the mean recurrence rates (%REC_r, %REC_l, and %REC_r1) are higher for the patient than for the control group. This means that patient EMGs contain more recurring patterns than the control EMGs. This is considered to be due to increased level of MU synchronization in PD [7]. For both of the subject groups, the recurrence rates of EMG are higher and the wavelet parameters are lower during the extension phases than during the flexion phases of the movement.

Furthermore, Fig. 2 shows that the mean acceleration power is higher and sample entropy is lower for the patient than for the control group through the four flexion–extension cycles. This means that the patient accelerations contain more oscillation activity, and they are more regular than the control accelerations.

The increased regularity of acceleration signals in PD has been observed previously in static measurements, and it is considered to be due to increased level of MU synchronization in PD [6]. For both of the subject groups, the power and regularity of acceleration are higher for the extension phases of the movement.

In conclusion, the differences between patients and controls occur similarly in all four flexion–extension cycles. It follows that a good discrimination can be obtained by examining the epoch averages of these parameters during flexion and extension movements (flexion and extension phases examined separately though). The mean ± SD values of normalized (to zero mean and unit SD) EMG and acceleration parameters for the

![Fig. 2. Mean values of time-varying EMG and acceleration parameters for the patient (black) and the control group (gray) during four elbow flexion (flex1, flex2, flex3, and flex4) and extension movements (ext1, ext2, ext3, and ext4).](image-url)
patient and the control group in flexion and in extension are presented in Fig. 3. The group differences in the epoch averages of all these variables were analyzed for flexion and extension phases separately. Univariate $t$-test was used to test the significance for normally distributed variables ($W_{\text{max}, f}$ in flexion, $\%\text{REC}_e$ in extension, and $P_{\text{acc}, r}$ in extension) and nonparametric Kruskal–Wallis test for other variables. For all variables, the group differences were significant ($p<0.01$).

**C. Feature Vectors and PCA**

Since the discrimination of the subject groups was done separately for flexion and extension movements, two feature vectors (one for flexion and one for extension) were formed for each subject using normalized parameter values in (5).

It was observed that the best discrimination between the subject groups is obtained by examining the first and second PCs, i.e., the weights of the first and second eigenvectors. These eigenvectors are presented in Fig. 3. The first eigenvector is the best mean square fit to the feature vectors of all subjects, and the second eigenvector is the best mean square fit to the residual of the first fit. It follows that the first PC (both for flexion and extension) describes the overall recurrence rate of EMG and the distribution of EMG frequency components. In addition, it describes the overall regularity and power of acceleration. By visually inspecting the second eigenvector in Fig. 3, we can observe what the second PC describes. Positive values of the second PC indicate higher recurrence rates, acceleration powers, and wavelet parameters than what can be described with the first eigenvector and the first PC. In addition, they indicate lower sample entropy for both sides in flexion and for right side in extension. Negative values of the second PC indicate lower recurrence rates, acceleration powers, and wavelet parameters than what can be described with the first eigenvector and the first PC. In addition, they indicate higher sample entropy for both sides in flexion and for right side in extension.

**D. Cluster Analysis and Validation**

The cluster analysis of feature vectors was done separately for the flexion and extension movements of subjects using the $k$-means algorithm, as described in [10]. The clustering results were validated using an extended version of leave-one-out method, as described in [10]. Briefly, one subject was left out each time the basis vectors and PCs were solved. The clustering of feature vectors was done for $M-1$ subjects, and after that, it was tested to which cluster the subject that was left out belongs. The correct ratings of clustering were defined by calculating the percentages of patients/controls that belong to the patient/control cluster. The results from the cluster analysis of subjects are presented in Fig. 4. According to clustering results of flexion, $73 \pm 1\%$ of patients were discriminated from $82 \pm 1\%$ of healthy controls. The corresponding ratings for extension were $80 \pm 1\%$ for patients and $87 \pm 1\%$ for healthy controls.

**V. DISCUSSION**

In PD, the dopaminergic neuronal loss in the basal ganglia of the brain affects the temporal organization of motor cortical activity during muscle contractions [3]. This leads to changes in EMG and kinematic measurements [4], i.e., tremor bursts induce changes in the EMG signal morphology and amplitude. These changes can be detected by analyzing dynamic EMG signal recorded from some selected muscles and acceleration measurement, as shown in this paper, and therefore, these analysis methods can be used for objective and quantitative assessment of PD. In this study, we analyzed elbow flexion and extension movements, and BB muscles were chosen for measurement because the measurement is easy to perform from large and well-known muscles, and the muscle action of biceps is well-defined. However, we would likely get similar results by measuring surface EMG from different muscles.

In this study, a method for discrimination of dynamic surface EMG and acceleration measurements between patients with PD and healthy controls was presented. In our previous study [10], where static EMG and acceleration measurements were analyzed, 76% of patients with PD were discriminated from 90% of healthy controls. Most of the patients (42/49) included in the current study were also included in the static study. The discrimination rates obtained in the current study for extension were similar but the discrimination rates for flexion were...
Fig. 4 Second PCs \( \hat{\theta}_j(2) \) with respect to the first PCs \( \hat{\theta}_j(1) \) for 49 patients with PD (o) and 59 healthy controls (+) in (top) flexion and (bottom) extension. The clusterings of subjects.

Weaker than in the static study. This result was expected because the differences between groups were more visible during the extension phase. In addition, the result is consistent with a previous study [14], where a greater impairment of extension compared to flexion movements were observed in PD.

In ideal situation, all patients would have formed one cluster and all controls the other cluster in this study. However, 27% of patients were incorrectly clustered in flexion and 20% in extension. Corresponding rates for healthy controls were 13% and 18%. Typical for the incorrectly clustered patients was that their UPDRS scores in tremor, rigidity, and finger tapping were low. However, all patients with low UPDRS scores were not incorrectly clustered. Comparison with the static study [10] showed that the incorrectly clustered patients were not the same in the two studies, and they were not characterized with similar symptoms. This proves, that the examination of both static and dynamic muscle contractions is important for increasing our understanding in PD. The incorrectly clustered controls, on the other hand, were characterized by age, i.e., only one younger (age < 45 years) control was incorrectly clustered in flexion and another one in extension. The acceleration measurements of incorrectly clustered controls were characterized by oscillations and EMG by recurring bursts. These findings are consistent with previous studies where older adults have been observed to demonstrate loss of acceleration complexity [25] and higher levels of EMG burst activity [26] during movement when compared with young adults. When compared to the static study [10], aging seems to have more effect on EMG and acceleration patterns during dynamic than static contractions.

Obtained clustering results for both flexion and extension showed that the first PC would have been enough for clustering the subjects into two groups (see Fig. 4). However, the second PC was included in the cluster analysis because it was interesting to see signs of subclustering of patients inside the patient cluster. The optimal set of EMG and acceleration parameters for discrimination was determined by gradually increasing and decreasing the number of parameters in the feature vectors. The chosen parameters were observed to characterize essential differences in the dynamic EMG and acceleration measurements between patients and healthy controls. In addition to analyzed kinematic features, it would have been interesting to compare the peak velocities of the forearm movement between subjects. However, this was not possible because the peak velocity was limited in our study protocol. Because the symptoms of PD are often unilateral at disease onset, it might be possible to increase the discriminative power of the presented method by choosing only the more affected side of the patient for analysis. However, the more affected side was not known for all patients in this study.

Clinical assessment of PD severity using standardized rating scales, such as UPDRS, is still the only widely used method in PD diagnosis. This method is subjective, and the diagnosis using subjective evaluation can be difficult. Comparison between the total UPDRS motor scores and the PCs of this study showed that there is a significant correlation between the first PCs and the total UPDRS motor scores in flexion (Spearman correlation, \( p < 0.01, R = -0.443 \)) but not in extension. However, this correlation was not further considered in this study for two reasons. First, the total UPDRS motor scores do not directly measure the impairment in arm muscle function that was tested in this study. Second, the UPDRS examination was not performed at the same time with the measurement for all patients, which is a problem because our previous studies have shown that the severity of PD symptoms can evolve rapidly.

The discrimination rates between patients with PD and healthy controls obtained in this study (73%/82% in flexion and 80%/87% in extension) depict the sensitivity/specifiity of the method. Even though such promising discrimination was obtained, the diagnostic value of the method should be estimated more carefully in further diagnostic studies. Considering the possible clinical use of the method, once sufficient amount of data for training the method have been collected, a new patient could be classified by using the eigenspace formed by the training data. Similarly, physician could follow disease progression or evaluate effectiveness of the treatment.
REFERENCES


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