Analysis of surface EMG signal morphology in Parkinson's disease

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Abstract. A novel approach is presented for analysis of surface electromyogram (EMG) morphology in Parkinson's disease (PD). The method is based on histogram and crossing rate (CR) analysis of EMG signal. In the method, histograms and CR values are used as high dimensional feature vectors. The dimensionality of them is then reduced using Karhunen-Loève transform (KLT). Finally, the discriminant analysis of feature vectors is performed in low dimensional eigenspace. Histograms and CR values were chosen for analysis, because Parkinsonian EMG signals typically involve patterns of EMG bursts. Traditional methods of EMG amplitude and spectral analysis are not effective in analysing impulse-like signals. The method, which was tested with EMG signals measured from 25 patients with PD and 22 healthy controls, was promising for discriminating between these two groups of subjects. The ratio of correct discrimination by augmented KLT was 86 % for the control group and 72 % for the patient group. On the basis of these results, further studies are suggested in order to evaluate the usability of this method in early stage diagnostics of PD.

Keywords: Electromyography (EMG), crossing rate, histogram, Karhunen-Loève transform, Parkinson's disease

1. Introduction

The electrical potential that is caused by the function of muscles is called electromyogram (EMG). EMG signal can be measured from muscles by using intramuscular needle electrodes or by using surface electrodes attached to the skin. The latter method is called surface EMG measurement. The traditional methods to

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analyze surface EMG signals are based on amplitude and spectral analysis. They are used mainly to measure level of muscle activation and fatigue.

Parkinson's disease (PD) is a neurodegenerative disorder that affects one percent of all people over 60 years of age in industrialized countries (De Lau and Breteler 2006). It is a progressive disorder that causes motor dysfunctions: involuntary and oscillatory movements (tremor), slowness of movements (bradykinesia), increased muscle tone (rigidity) and postural instability (Valls-Solé and Valldeoriola 2002). A common type of Parkinsonian tremors is rest tremor but also postural, kinetic and intention tremors have been reported (Milanov 2000). According to current diagnostic criteria, patients can be considered to have PD when they have two of the four symptoms of PD (tremor, bradykinesia, rigidity and postural instability). Although these dysfunctions can be assessed by electromyography, it is still used rarely in the clinical evaluation of PD. The advantage of using EMG in the assessment of PD would be its objectivity and quantitativeness to evaluate motor function. In general, neurophysiological measurements may help in the future in advancing the diagnosis and in the differential diagnosis of PD.

The connection between certain parts of brain and skeletal muscles has been previously studied by simultaneous recordings of EMG and MEG (magnetoencephalographic) signals. It has been shown that the basal ganglia have a specific effect on the temporal organization of motor cortical activity during isometric contractions (Salenius *et al* 2002). In this aspect, the dysfunction of substantia nigra and basal ganglia directly leads to abnormalities of skeletal muscles as seen in patients with PD, i.e. causes tremor, bradykinesia and rigidity.

The basic changes in the EMG signal caused by PD are increased tonic background activity and an alternating pattern of EMG bursts. In fact, in the EMG analysis of PD special attention has been paid to the analysis of these bursts by measuring their counts, magnitudes, durations and frequencies (Flament *et al* 2003, Pfann *et al* 2001, Robichaud *et al* 2002, Robichaud *et al* 2004). Analysis of these bursts (Robichaud *et al* 2004) shows that for patients with PD there are more agonist bursts during extension movements than during flexion movements. Moreover, the extension movements are slower than the flexion movements. For neurologically healthy subjects, there are no significant changes in the EMG or in speed between these movements.

Many other studies of Parkinsonian EMG have focused on the analysis of tremor. In one of these studies, four different subtypes of Parkinsonian tremors have been found (Milanov 2000). Moreover, differences between Parkinsonian and physiological tremors of healthy persons have been detected by combined measurement of EMG and acceleration (Vaillancourt and Newell 2000). In fact, distinguishing Parkinsonian tremors from other kinds of tremors can be difficult. However, it has been shown that the Parkinsonian tremor differs from the physiological tremor by being more regular. The regularity of tremor is thought to result from an increase in the synchronization of motor units (MU) (Vaillancourt and Newell 2000).

EMG may contain important information about brain and motor dysfunctions in

PD. In the future, it can be useful in the early stage diagnostics of PD. However, EMG is a spiky impulse-like waveform and the information about brain disorder is in the morphology of an impulse chain. Impulse-like signals are hard to be analysed with traditional amplitude and spectral Fourier based methods. Therefore, novel approaches such as wavelets could be applied to this problem (De Michele *et al* 2003). Another approach is presented in this study.

We have developed a new approach for discrimination of EMG waveforms of patients with PD and healthy persons. The method is based on histogram and crossing rate (CR) analysis of signals. In the method, histograms and CR values are used as high dimensional feature vectors. The dimensionality of them is then reduced using the Karhunen-Loève transform (KLT). Finally, the discriminant analysis of feature vectors is performed in low dimensional eigenspace. Histograms and CR values were chosen for analysis, because they are expected to be sensitive to impulse-like signals. Previous studies on EMG probability density have dealt with the hypothesis of Gaussian/Laplacian densities and the use of higher order statistics in the signal classification (Clancy and Hogan 1999, Nazarpour *et al* 2005). CR calculations, instead, have previously been used to detect fatigue (Inbar *et al* 1986). The number of crossings per second has usually been calculated at the threshold level of zero. However, in this study, the CRs are calculated at various threshold levels in order to form an expansion of CRs.

2. Materials and methods

2.1. Measurements

Twenty-two healthy subjects (age 38 ± 13 years, weight 75 ± 13 kg, height 174 ± 10 cm) and twenty-six patients with Parkinson's disease (age 63 ± 9 years, weight 81 ± 12 kg, height 175 ± 8 cm) participated in this study after giving their informed consent. The mean duration of PD was 9 ± 6 years and the motor score of the Unified Parkinson's Disease Rating Scale (UPDRS) was 23 ± 14. Patients were recruited by the Department of Neurology, Kuopio University Hospital (Kuopio, Finland) and the Brain Research and Rehabilitation Center Neuron (Kuopio, Finland). The general inclusion criteria for patients were that they had an earlier diagnosis of idiopathic Parkinson's disease and they were taking medication for the condition. Patients with other neurological disorders or injuries that interfere with motor function were excluded from the study. The control subjects were recruited from generally healthy Finnish citizens. This study was approved by the Human Ethics Committee of Kuopio University Hospital (Kuopio, Finland). The patients were on-medication at the time of the measurements because of three reasons. First, the off-medication state can be really unpleasant for patients and all patients can not be taken off-medication because the risk of falling increases. Second, it has been shown that the medication does not restore the temporal structure of EMG (Robichaud *et al* 2002). Therefore, the bursting behaviour of EMG stays

evident even when the patients are on-medication. And third, the ultimate aim of this study is to introduce a new approach for discrimination of EMG waveforms and not to give an overall solution for PD diagnostics.

Bipolar surface electromyography was used to quantify the activity of the biceps brachii muscle. Pairs of disposable Ag/AgCl surface electrodes (Medicotest, model M-00-S, Olstykke, Denmark) were attached bilaterally over the belly of the biceps brachii muscles with an inter-electrode spacing (center to center) of 3 cm. The inter-electrode spacing was chosen large enough to obtain information about the functioning of sufficient number of motor units. The reference electrodes were placed 6 - 7 cm laterally from the recording electrodes. A bipolar ME6000 EMG system (Mega Electronics Ltd., Kuopio, Finland) was used to record EMG continuously. Cables with distal preamplifiers assured good signal quality. A raw EMG signal was recorded with a sampling rate of 1000 Hz, analogically band-pass filtered with an anti-aliasing filter (Butterworth, band-pass $1 - 500$ Hz), amplified (differential amplifier, CMRR > 130 dB, total gain 1000, noise < 1 μV), analogue-to-digital converted (12-bit), and stored in a PC-computer for later analysis. In this study, two accelerometers were used to determine which side of the patient was more affected by tremor. They were attached to the wrists of the subjects with wrist supports.

During the measurements, subjects were asked to hold their elbows at an angle of 90 degrees with their palms up. The movement of the arm was not restricted because the acceleration of hand (tremor) was registered simultaneously with EMG measurement. The isometric contractions lasted for 10 seconds. EMG measurements were performed once with each level of muscle contraction (no weights and with weights of 0.5, 1, 2, 3, 4 and 5 kg). However, because the Parkinsonian EMG bursts are most visible in the signals of non-loaded condition, only the non-loaded condition is examined in this study. This means, that one EMG sample per subject was used for analysis.

2.2. Data Analysis

The following methods were used for analysis of EMG signal morphology in this study. All EMG signals were analysed by Matlab^{TM} (MathWorks Inc.) in three stages: 1) pre-processing, 2) calculation of feature vectors (histograms and crossing rate expansions) and 3) Karhunen-Loève transform of feature vectors. After these steps, the discriminant analysis of feature vectors can be performed.

2.2.1. Pre-processing Slow variations in EMG signals can be caused by movement artifacts or by instable electrode-skin interfaces (Merletti *et al* 1999). They are generally high pass filtered (cut-off 0-20 Hz) from the signal before analysis. However, in some cases there is relevant information about the firing rates of active motor units in the low-frequency components; in that case, the trend must be removed so that no relevant information is lost. In Parkinson's disease this trend can be due to tremor and therefore contain important information itself. For trend removal we used a detrending method

that is called the smoothness priors method. With that method it is possible to remove the trend from the EMG signal and store it for later analysis.

Smoothness priors works as a high-pass filter. However, there are two main advantages of the smoothness priors method when compared to traditional high-pass filtering. First, in smoothness priors method, the frequency response of the filter is adjusted with a single smoothing parameter while in high pass filtering with more variables such as the filter order, power of ripple at pass-band and cut-off-frequency. And second, smoothness priors method attenuates the filtering effect at the beginning and the end of data to avoid distortion of data end points.

A detailed description of the detrending method used with application to HRV analysis can be found in (Tarvainen *et al* 2002). Briefly, an EMG signal z can be considered a sum of nearly stationary EMG time series and a low-frequency trend component

$$z = z_{\text{stat}} + z_{\text{trend}}. \tag{1}$$

The trend component can be modeled with a linear observation model and solved as a regularized least squares solution. After that, it can be subtracted from the original signal.

EMG signals were pre-processed in this study as follows. Five-second long segments of EMG were first chosen from the end of the 10-second trial. The length of the segment was chosen to be five seconds, because in morphological analysis, it is important to choose the length of the segment in a way that the segment involves sufficient number of features that one wants to analyse. If one is interested in MU action potentials, it has been shown that the length of the segment can be 1 - 2 seconds with low levels of isometric constant force contractions (Merletti *et al* 1999). However, if one is interested in EMG burst modulation, the segment should contain several EMG bursts. Therefore, the length of the EMG segment was chosen on the basis of containing sufficient number of EMG bursts. It was checked visually that the EMG segments chosen did not involve peaked artefacts. EMG data of one Parkinsonian patient was discarded because of power-line interference. That is, 26 patients were measured but 25 patients were analysed in this study.

After choosing the EMG segments, they were detrended by the smoothness priors method (cut-off $\approx$ 10 Hz). They were also scaled before histogram and CR analysis. One possible choice for the scaling parameter could be the maximum value of EMG. However, in this study the scaling parameter was chosen to be the minimum value of the 50 highest values of the absolute EMG. This choice was made to exclude outliers.

2.2.2. Histogram EMG probability density was estimated for scaled signal segments by a sample histogram with 200 bins. In the previous study of EMG probability density (Clancy and Hogan 1999), it was shown that the real EMG densities are between theoretical Laplacian and Gaussian densities. The hypothesis of Gaussianity can be tested by calculating the kurtosis of the EMG data (Nakamura *et al* 2004). Kurtosis

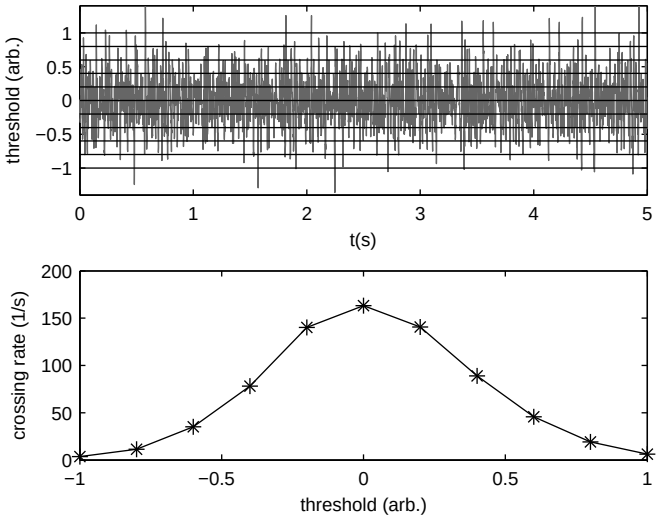

Figure 1. Formation of the CR expansion with eleven threshold levels (plotted as lines in relation to EMG signal).

(k) is defined as the fourth centred moment of the data z

$$k = \frac{E\{(z-\mu)^4\}}{\sigma^4} \tag{2}$$

where μ is the mean of the data and σ the standard deviation. The kurtosis value for normal distribution is 3. Because of the bursting and spiky behaviour of Parkinsonian EMG, the sample distributions of patients might differ from the distributions of healthy controls.

2.2.3. Crossing rate expansion CR expansions were calculated for all signal segments as the number of crossings at given threshold levels. A crossing occurs when two neighboring values in a time series are on opposite sides of the threshold level. The implementation of CR analysis with 11 different threshold levels is illustrated in figure 1. In this study, the number of different threshold levels was 201. The choice of the used number of threshold levels was made on the basis of obtaining as good representation as possible of the CR expansion.

2.2.4. Feature vectors Three different types of feature vectors were used in this study. The first type vectors contained histogram values for each EMG segment. The second type vectors contained values of CR expansion for each EMG segment. And the third type vectors contained concatenated CR expansion and histogram for each EMG segment. For clarification, few examples of concatenated CR expansions and histograms are represented in context with results in figure 4 (a).

2.2.5. Karhunen-Loève transform of feature vectors Karhunen-Loève transform is closely related to principal components, which can be used to reduce the dimensionality

of a data set without losing significant information in the original data. When the dimensionality of the data decreases, it becomes possible to represent multidimensional data graphically. In this study, principal components were used in context with KLT to discriminate EMG signals on the basis of signal morphology.

KLT can be used for approximation of feature vectors in the following way. First, the j'th feature vector $z_j \in \mathbb{R}^N$ is modeled with a linear model

$$z_j = H\theta_j + v_j. \tag{3}$$

In this model the matrix $H = (\phi_1 \ \cdots \ \phi_K) \in \mathbb{R}^{N\times K}$ is the model matrix. The column vectors ϕ_i are the basis vectors. The parameter $\theta_j \in \mathbb{R}^K$ contains the weights and v_j the model error for the j'th feature vector. In other words, vector z_j is expressed as the sum of vectors $\phi_1, ..., \phi_K$

$$z_j = \phi_1\theta_j(1) + \phi_2\theta_j(2) + ... + \phi_K\theta_j(K) + v_j. \tag{4}$$

If a data set consists of feature vectors for M subjects, the linear model (3) can be presented in matrix form. By denoting the feature vector for the j'th subject as z_j, the data matrix Z is then defined as

$$Z = (z_1 \ \cdots \ z_M) \tag{5}$$

and the linear model (3) can be written in matrix form as

$$Z = H\theta + v \tag{6}$$

where $\theta = (\theta_1 \ \cdots \ \theta_M) \in \mathbb{R}^{K\times M}$ and $v = (v_1 \ \cdots \ v_M) \in \mathbb{R}^{N\times M}$.

There are many ways to select the basis vectors ϕ_k in H. In KLT, the basis vectors are the eigenvectors of the experimental correlation matrix

$$R = \frac{1}{M}\sum_{j=1}^{M} z_j z_j^T = \frac{1}{M} ZZ^T. \tag{7}$$

It can be shown that the first basis vector ϕ_1 is then the best mean square fit for the data set and the second basis vector ϕ_2 is the best mean square fit for the residual of the first fit. Therefore, by using the eigenvectors that correspond to the K' ($K' < M$) largest eigenvalues $(\lambda_1, \ldots, \lambda_{K'})$, the best K'-dimensional approximation for the data set is obtained. The principal components $\theta_j(i)$ correspond to the fitting coefficients in the sum (4) and they can be solved in the least squares sense from the linear model (6) as

$$\hat{\theta} = (H^T H)^{-1} H^T Z = H^T Z \tag{8}$$

where $H^T H = I$ because the eigenvectors of R are orthonormal. In this study the principal components are used for discrimination between subjects.

In augmented KLT, multiple data sets can be combined. In the case of two data sets Z^1 and Z^2, the feature vector for the j'th subject consists of the concatenated

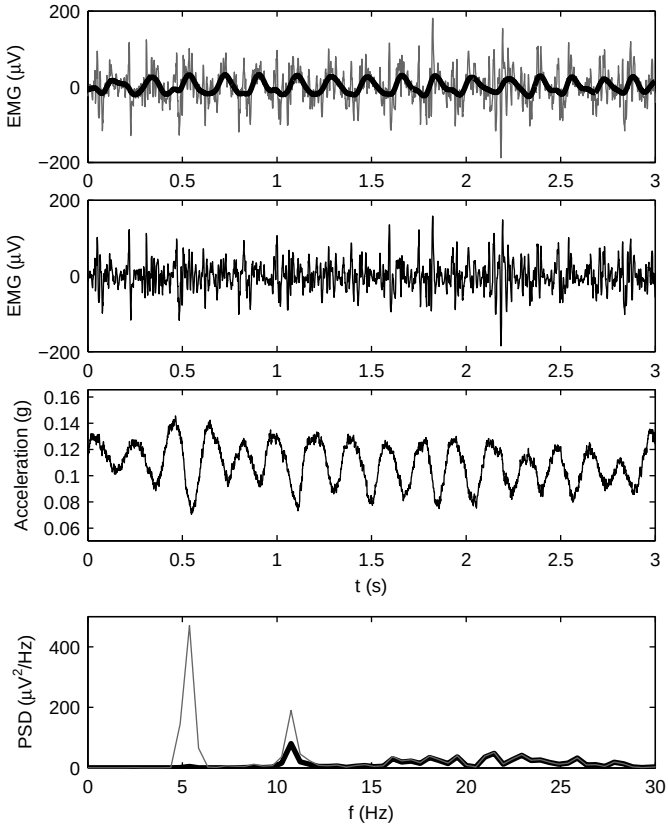

Figure 2. Original EMG and estimated trend for one patient (top). Detrended EMG (second from top). Acceleration signal (third from top). Power spectral density of original (gray) and detrended (black) EMG (bottom).

feature vectors z_j^1 of length N_1 and z_j^2 of length N_2. It is placed in the j'th column of the data matrix Z. For two data sets the data matrix for M subjects is

$$Z = \begin{pmatrix} Z^1 \\ Z^2 \end{pmatrix} = \begin{pmatrix} z_1^1 & \cdots & z_M^1 \\ z_1^2 & \cdots & z_M^2 \end{pmatrix}. \tag{9}$$

The data correlation matrix, eigenvectors and principal components are obtained similarly to the description with one data set.

3. Results

3.1. Detrending

All EMG signals were detrended by the smoothness priors method. Figure 2 illustrates the effect of the detrending method on the EMG measured from one patient with PD. The trend caused by tremor (~ 5 Hz) can be seen clearly in the original EMG but not in the detrended one. By comparing the trend with the acceleration signal, one can see that the removed trend is caused by movement and does not contain important information

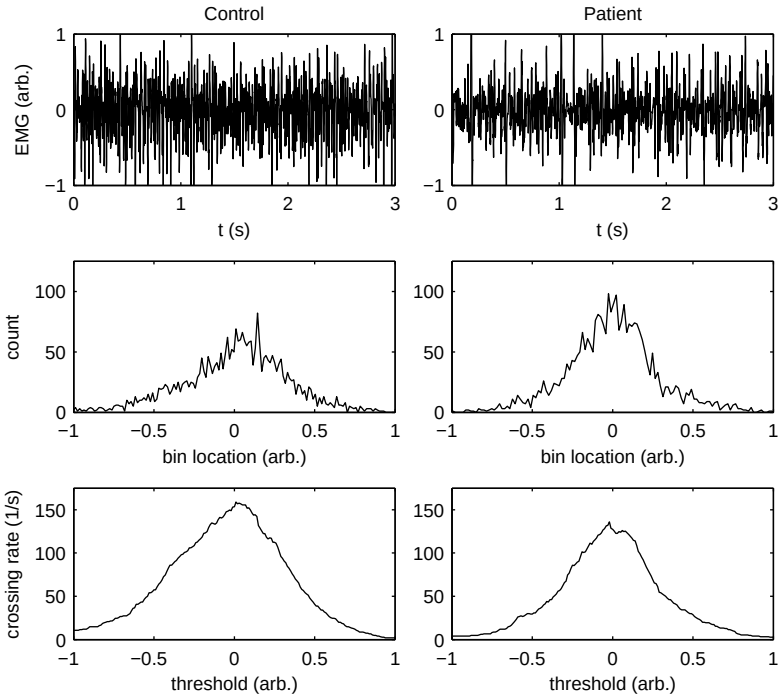

Figure 3. EMG of one control (top left) and one patient (top right). Corresponding histograms (middle) and CR expansions (bottom).

about EMG signal morphology. Figure 2 demonstrates the effect of detrending also in frequency domain. One can see, that the power of lower frequency components is decreased in the spectrum of the detrended EMG. The spectrum estimate was calculated by Welch's averaged periodogam with a window length of 2048 points and 75 % overlap.

3.2. Histograms and CR expansions

EMG histograms and CR expansions were calculated for all subjects. Typical detrended and scaled EMG signals for one patient with PD and for one healthy control are presented in figure 3. Furthermore, the corresponding histograms and CR expansions are presented in the same figure. One can see that the histogram of the patient is sharper than that of the control. Sharper histogram means higher kurtosis of the data. In fact, the mean $\pm$ std value of kurtosis for the whole patient group was 4.9 ± 1.0 and for the control group 4.0 ± 1.0. In addition, one can see in figure 3 that the CR expansion of the patient is lower and narrower than that of the control.

3.3. Feature vectors and Karhunen-Loève transform

Three different types of feature vectors were formed for all signal segments as described in section 2.2.4. KLT was used to reduce their dimensionality. After that, it became possible to discriminate measured EMG signals in a low dimensional eigenspace. KLT was performed for the set of histograms and for the set of CR expansions separately

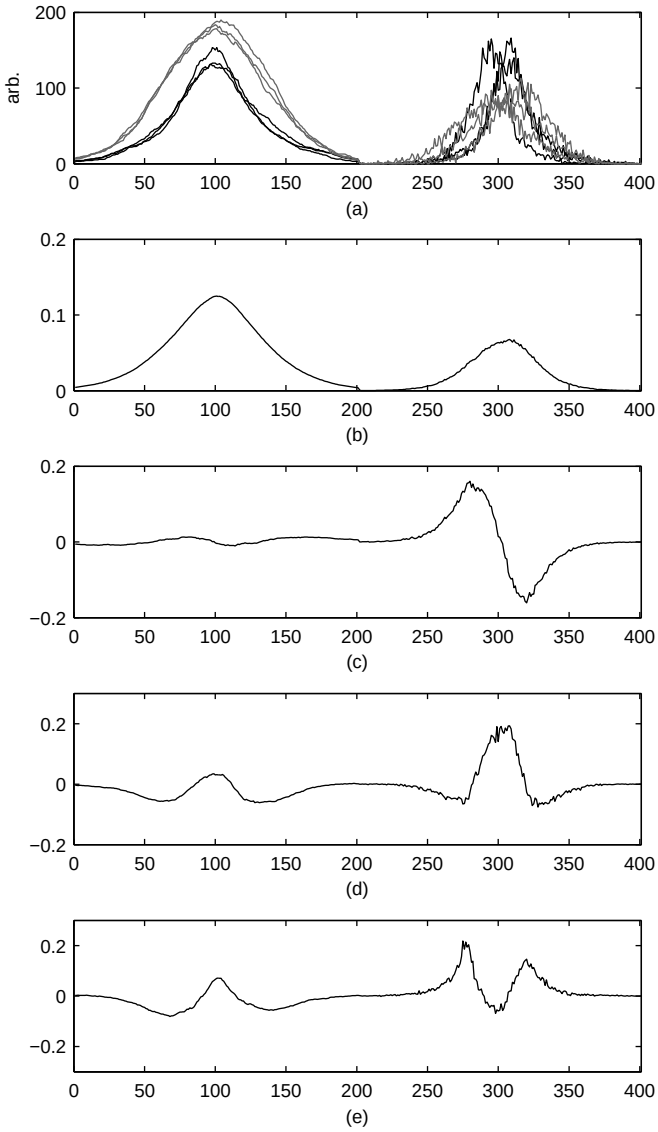

Figure 4. Concatenated CR expansions and histograms for three patients (black) and three controls (gray) in (a). Eigenvectors corresponding to the four largest eigenvalues in (b-e). Eigenvectors were calculated for the data set that contained the feature vectors of all subjects.

and augmented KLT for their combination.

3.3.1. Augmented KLT In augmented KLT, the feature vector for each subject consisted of the concatenated CR expansion and histogram. As an example, the feature vectors for three patients and three controls are presented in figure 4 (a). The feature vectors for all subjects were placed as column vectors in (9) by putting the values of CR expansion in the first 201 rows and the values of histogram in the 200 following rows. Therefore, the length of the feature vector for each subject was 401 points.

The next step in augmented KLT was to calculate the correlation matrix R in (7) and solve the eigenvalues and eigenvectors for it. Eight eigenvectors $(\phi_1, \ldots, \phi_8)$

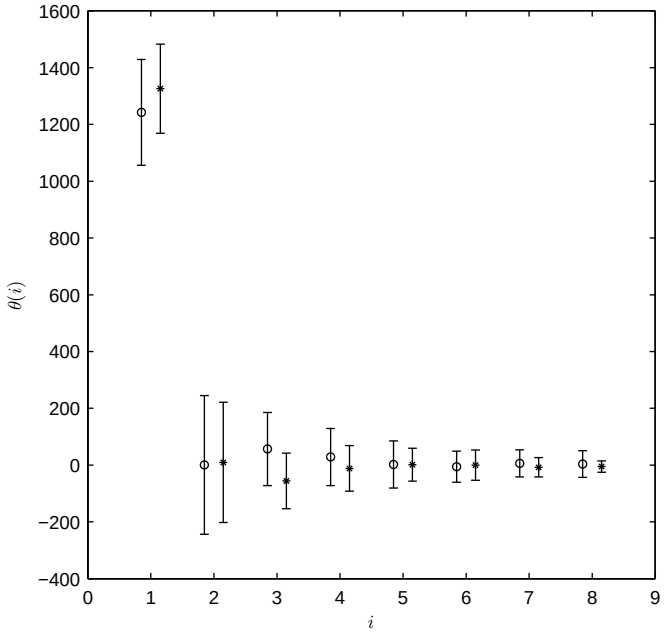

Figure 5. Mean ± std values of the principal components $\theta(i)(i = 1, ..., 8)$ for the patient (o) and control (*) group.

corresponding to the eight largest eigenvalues $(\lambda_1, \ldots, \lambda_8)$ were chosen as the basis vectors. The first four of them are presented in figure 4 (b-e).

Interpretation of the eigenvectors in figure 4 (b-e) and the principal components in figure 5: Eigenvectors in figure 4 (b-e) describe the correlations between the components of the feature vectors. That is, the feature vectors can be modeled as a sum of these eigenvectors. Principal components correspond to the fitting coefficients in the sum (4).

- The first eigenvector in figure 4 (b) is the best mean square fit for the data. Thus, it is similar to the mean of the feature vectors of all subjects. One can see in figure 5 that there are differences in the first principal component between the patient and the control group, but the differences are quite small.
- The second eigenvector in figure 4 (c) is approximately similar to the first derivative of the data. That is, in the linear sum (4) it tries to model the variation in the peaks (modes) of the histograms and CR expansions of all subjects. It can be seen that the variation is much stronger for the histogram than for the CR expansion. However, by looking figure 5 it can be seen that the differences in the second principal component between the subject groups are not significant.
- The third eigenvector in figure 4 (d) is approximately similar to the second derivative of the data. That is, it models variations in the heights and the widths of the histograms and CR expansions in the whole data set. The biggest differences between the patients and the controls are found in the third principal component.
- The fourth eigenvector in figure 4 (e) models the same kind of patterns in the

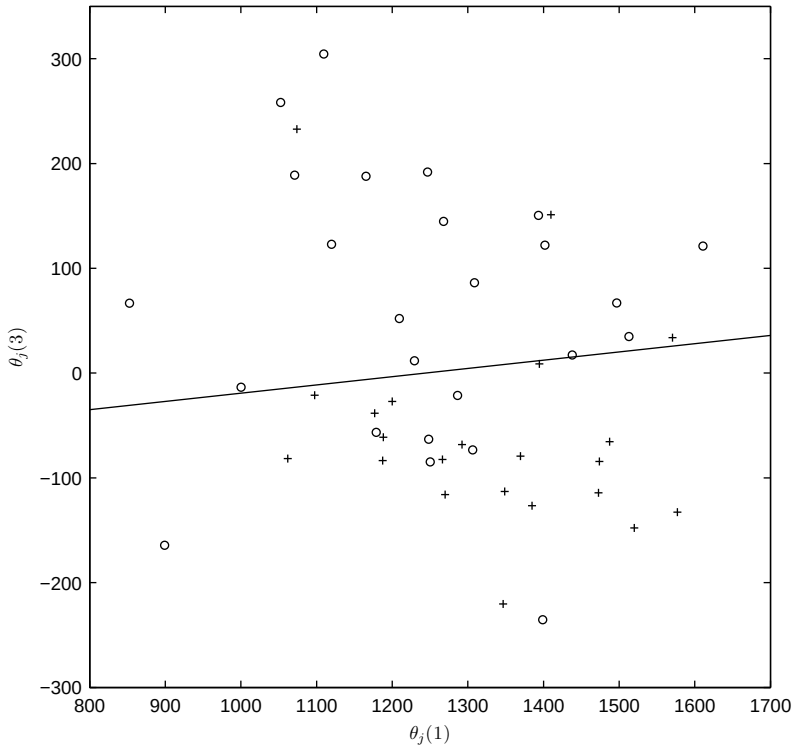

Figure 6. Discrimination results by augmented KLT: The third principal components $\theta_j(3)$ of healthy controls (+) and patients with PD (o) with respect to the first principal component $\theta_j(1)$.

feature vectors as the second and third eigenvector. The differences in the fourth principal components between the subject groups are small.

Because the largest differences between the control and the patient group are found in the third and first principal component, the discrimination between subjects is done in the subspace that is spanned by the first and the third eigenvector. The third principal components $\theta_j(3)$ for all subjects are presented with respect to the first principal component $\theta_j(1)$ in figure 6. A line is inserted into the figure to discriminate between subjects. One can see that most of the controls (19/22) are located below and most of the patients (18/25) above the discrimination line. If this line was used as a discriminative function for classification, the ratios of correct classification for the control group would be 86 % and for the patient group 72 %.

3.3.2. KLT of histogram In the KLT of histograms, the histograms of all subjects were placed as column vectors z_j in equation (5). The data correlation matrix and the corresponding eigenvectors and principal components were calculated for the data set the same way as in the augmented KLT. The best discrimination between patients and controls was obtained by analysing the third and the first principal component. These principal components for all subjects are presented in figure 7. Again, the line in the figure discriminates between subjects. One can see that most of the controls (16/22)

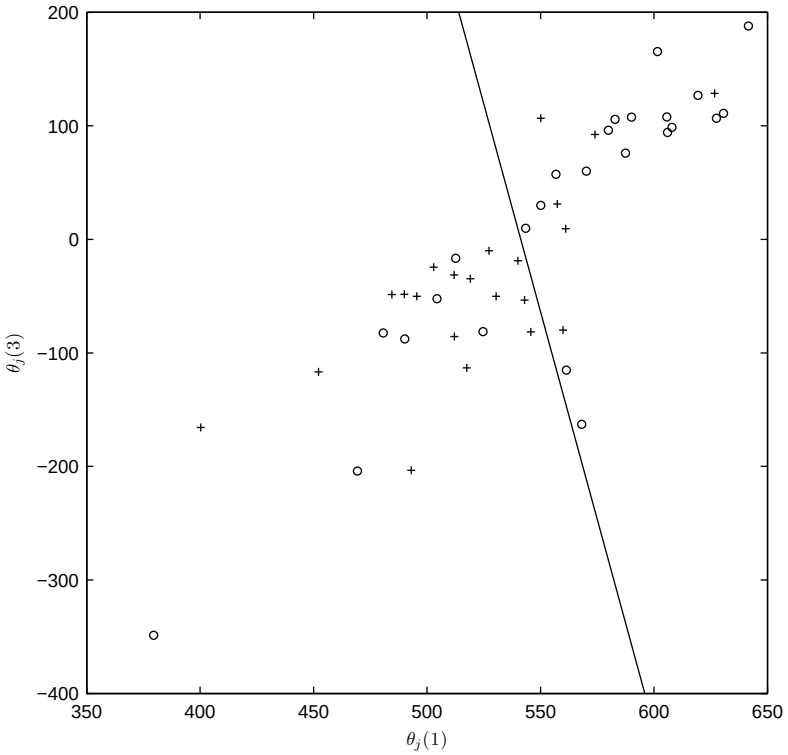

Figure 7. Discrimination results by KLT of histograms: The third principal components $\theta_j(3)$ of healthy controls (+) and patients with PD (o) with respect to the first principal component $\theta_j(1)$.

are located at the left side and most of the patients (18/25) at the right side of the discrimination line. In other words, in the histograms there are differences between the subject groups.

3.3.3. KLT of CR expansion Like in the KLT of histograms, the CR expansions of all subjects were placed as column vectors z_j in equation (5). The correlation matrix and the corresponding eigenvectors and principal components were calculated for the data set the same way as in the augmented KLT. Again, the best discrimination between the subject groups was obtained by analysing the third and the first principal component. These principal components for all subjects are presented in figure 8. The line inserted into the figure discriminates between subjects so that most of the controls (20/22) are located below and most of the patients (16/25) above the discrimination line. In other words, in the CR expansions there are differences between the patients and the controls.

4. Discussion

A method was presented here for analysis of surface EMG signal morphology in Parkinson's disease. The method, which was tested with EMG data measured from 25 patients with PD and 22 healthy controls, could reasonably well discriminate between

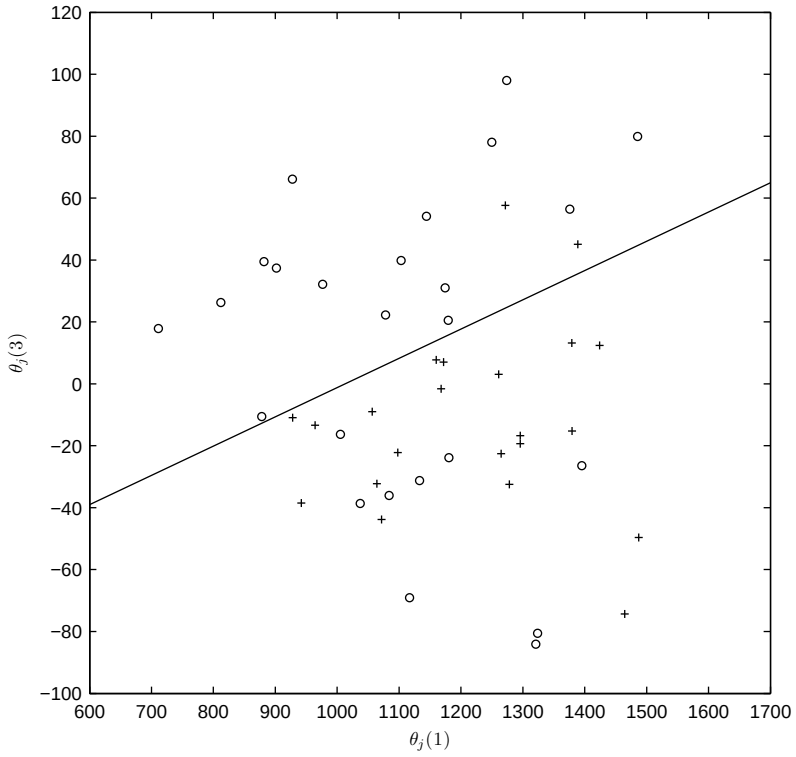

Figure 8. Discrimination results by KLT of CR expansions: The third principal components $\theta_j(3)$ of healthy controls (+) and patients with PD (o) with respect to the first principal component $\theta_j(1)$.

these two groups of subjects. Probably the best discrimination was obtained by using augmented KLT, in other words, by analysing both sample histograms and CR expansions together. The largest differences between the subject groups were obtained by analysing the third principal component, which illustrates the widths and the heights of the histograms and CR expansions. The sharpness of the histogram results from the spiky behaviour of the EMG signal and the spiky behaviour from the increased synchronization of motor units. In fact, the increased level of motor unit synchronization is a typical feature for patients with PD (Fattorini *et al* 2005). Moreover, the area under the curve of CR expansion was smaller for patients than for controls. That is, there are fewer crossing events in the signals of patients. Likewise, this could be a sign of MU synchronization.

According to the discrimination results obtained by using augmented KLT, 18 patients with PD and 19 controls were correctly classified as patients and controls, respectively. Seven patients were incorrectly classified as controls and three controls as patients. The reason for the incorrect classification of patients can be partly explained by three different things. First, three of the incorrectly classified patients had a low UPDRS -motor score (< 10). That is, their motor symptoms of PD were mild at the time of the measurements. However, it must be noted, that there were five patients with low UPDRS -motor score (< 12) that were correctly classified. This indicates that

the method could work at early stages of PD. Second, EMG signals of two incorrectly classified patients were strongly nonstationary i.e. the statistical properties of the signals changed strongly as a function of time. The nonstationary behaviour of EMG affected significantly on the shape of the histogram and furthermore the discrimination results. The problem with the nonstationarity could be probably solved by analysing the time dependent structure of EMG. However, the present study did not concern this issue. And third, one incorrectly classified patient had essential tremor in addition to Parkinson's disease, which clearly affected the obtained signal structure. At this point it is also important to note that all patients with PD were on medication at the time of the measurements, which may have weakened the discriminative power.

The method presented here differs from the traditionally used methods in EMG analysis. It is designed to detect bursting and spiky patterns in EMG signals. Traditional Fourier-based spectral analysis is a decomposition of the signal into harmonic basis functions of different frequencies. Therefore, it is not effective in analysing impulse like signals. Furthermore, traditional methods of amplitude analysis are able to detect increased level of muscle activity but not very effective in analysing signal morphology. The method presented here is based on sample histograms and CR expansions, which are easy to calculate and interpret. Parkinsonian EMGs have been previously studied by analysing the agonist-antagonist activity. In this study, only the EMGs of agonist muscles were measured and analysed.

A drawback of this method is that it requires good signal quality. Noises coming from other electrical sources have a significant effect on the EMG feature vectors used in this study. Furthermore, sometimes large motor units in the electrode neighbourhood may cause spiky patterns in the EMG resulting in the sharpness of the histogram and furthermore in different discrimination results by KLT. Therefore larger inter-electrode spacing may be advisable in order to obtain a good representation of the functioning motor unit pool. In this study, this was actually the case with one control who was incorrectly classified as patient by augmented KLT. It must also be noted that the detrending of EMG is especially important before morphological analyses.

It was not possible to cluster the subjects tested here strictly into two groups (patients and controls) on the basis of this method. However, this result was expected because the number of test subjects was small and the subject groups were quite heterogeneous. Moreover, the fact that patients were on medication at the time of the measurements may have reduced the group differences in the analysis. Still, despite of these shortcomings, the results of this study can be considered promising. After all, this study is a step towards objective and quantitative methods to assess motor function in PD.

The only well established method in PD diagnostics is the clinical evaluation of Parkinsonian symptoms by a clinician. This is often done by using UPDRS-examination. However, because the total UPDRS motor scores that are known about the patients do not directly measure the impairment in muscle function tested in this study and all the scores have not been defined at the same time with the measurement, it is not very

useful to compare them with the principal components used in this study. However, it is known, that the diagnosis of PD can be difficult. In fact, clinicopathological studies have shown that by using the subjective method of clinical evaluation the disease is diagnosed incorrectly in about 25 % of cases (Tolosa *et al* 2006). The reasons for misdiagnosis are partly explained by other Parkinsonian like diseases such as essential tremor and vascular Parkinsonism. Imaging techniques can be of help in the differential diagnostic, but they are not commonly used in the diagnostics of PD. This is partly because of the costs. Finnish study on [^{123}I]β-CIT SPECT showed, that in clinical practice, the differential specificity of [^{123}I]β-CIT SPECT in older patients (age $>$ 55 years) was 68.5 % (Eerola *et al* 2005). Patients with other neurological disorders were not examined in this study. However, when thinking of these percentages, EMG research should be regarded as a promising and cost-effective technique for quantification of symptoms and signs in Parkinson's disease.

The method presented in this study, is probably not yet specific enough for diagnostics, but if needed, the method can be later on easily improved by increasing the number of EMG features to be analysed. These features could include nonlinear and dynamic characteristics of EMG. In the future, one could also think of using more measurements per subject for classification. That was not possible with this study protocol. In addition, one could check, if it is needed to register longer samples of EMG because the severity of Parkinsonian symptoms and EMG bursting are fluctuating fast even within 30 seconds period.

The use of this method is not restricted to analysis of Parkinsonian EMG. Other applications of this method could include physiological or disease-based processes that lead to the synchronization of motor units (e.g. muscle fatigue, neuropathy, extrapyramidal tremor, anxiety and depression).

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