# NEUROFEEDBACK TOOL TO IMPROVE THE ONSET DELAY AND SEQUENTIAL MOVEMENTS DURING MOTOR TASKS

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Abstract: Neurofeedback can be useful in several neurological diseases if the user knows that his/her current mental state can be modify it to improve his/her development. The NFB here proposed provides modulation of cortical areas of the brain through motor imagery tasks (ERD-ERS). The topographic representation of the brain activity is shown for the user after motor task, .which are based on motor imagery of the left and right arm movements separately. Preliminary tests show that only some volunteers get good performance with feedback and have high activation level.

Keywords: Neurofeedback, Motor Imagery, ERD-ERS, EEG.

# Introduction

Movement disorders are neurological conditions that affect speed, fluency, quality, and ease of movement. Abnormal fluency or speed of movements called dyskinesia may involve excessive or involuntary movement (hyperkinesia) or slowed voluntary movement (hypokinesia). Movement disorders include several conditions, such as: ataxia, dystonia, progressive supranuclear palsy, tics and tremor. These conditions are observed in the Wilson disease, Parkinson disease (PD), and others [1].

Neurofeedback (NFB) is a rehabilitation therapy that can be employed on subjects with movement disorders to improve the motor function. These subjects can upregulate the higher cortical motor area and modulate the activity in basal ganglia circuits implicated in movement disorders. Activations during NFB have been observed in cortical motor areas and basal ganglia, including the subthalamic nucleus and globus pallidus, which are connected to the supplementary motor area (SMA) and crucial nodes in the pathophysiology of PD [2]. In humans, it was reported that SMA is crucial to the organization of both initial as well as sequential movements [3].

PD mainly consists of slowness of voluntary movements and progressive deterioration over time of motor performance during sequential movements. It is characterized by muscle rigidity, tremor, a slowing of physical movements (bradykinesia) and even loss of physical movements in an extreme situation (akinesia) [4]. These patients perform complex motor task (simultaneous/sequential) and their motor execution deteriorates significantly over time owing to the sequence effect. Furthermore, PD not only takes longer to complete each sequential sub-movement compared to the same motor task performed independently, but the time lapse between one sub-movement and the next is also significantly prolonged [5].

In PD the primary motor symptoms are the results of decreased activities of the motor cortices through the basal gangliathalamo-cortical loop, caused by the insufficient formation and action of dopamine, which is produced in the dopaminergic neurons of the brain [2].

The study conducted by [6], using fMRI, to investigated whether increased activity in the supplementary motor area (SMA) would result in improved motor function in patients with early stage of the PD. According to this study, the motor imagery task combined with fMRI neurofeedback showed higher activation than imagery control in several brain regions and improved motor speed (finger tapping) and clinical ratings of motor symptoms.

Other study [7] showed results of a comparison between a "cross-over" condition without NFB and subjects who received NFB immediately prior to Serial Reaction Time Task (SRTT). With NFB, these exhibited a significantly faster rate of learning, reflected in a greater reduction of reaction times across blocks. However no significant differences were observed between conditions in error rate or reaction time variability. This suggests that a single NFB session may be directly used to facilitate early acquisition of a procedural motor task, so as to boost behavioral performance and learning.

Research has demonstrated that the early component of Bereitschaftspotential (BP1) but not late component of Bereitschaftspotential (BP2) is reduced in amplitude compared to age-matched healthy subjects supporting the hypothesis of SMA underactivity in PD. BP1 occurs 1.5-0.5 s before movement onset in the SMA and then spreads to involve the lateral premotor areas bilaterally, and a BP2 preceding movement onset at 0.5 s or less [8]. Similarly, the desynchronization of sensorimotor rhythms can be used to study the cortical activation patterns during the planning of movements [9]. Such desynchronization is usually called Event-Related Desynchronization (ERD), and corresponds to a short lasting amplitude attenuation of EEG intrinsic rhythms by externally or internally paced-events. ERD was first quantified by, and starts over the contralateral sensorimotor areas on an average 1.7s before the movement, with a bilateral pattern after the movement onset [10].

Perhaps the most important observation in depth recordings up to date in PD has been the propensity for neuronal synchronization in the subthalamic nucleus (STN) and globus pallidus at frequencies between 8 and 30 Hz. The treatment reduces the level of synchronization over 8-30 Hz at rest and may increase the additional suppression of this activity that occurs prior to and during voluntary movements.

State of the Art -Bereitschaftspotential paradigm has been used to analyze the cortical processes involved in the preceding period of voluntary movement in PD. The study with ERD in PD was reported by [11] [12] founded that a topographical pattern of ERD, mainly over the front central area covering the SMA, appeared different in the Parkinsonian group as compared to the control group, when movement was performed with the akinetic hand. In order to obtain a clear distinction between BP and ERD [11] presented a study with simultaneous measure of the BP and ERD during self-paced voluntary movement in Parkisonian and control subjects. In the spatiotemporal maps of BP no apparent difference was noted between two groups. However, in the spatiotemporal maps of ERD (bandwidth of 9-11 Hz) difference between two groups was noted. In the control group, after wrist flexion, desynchronization began 1750 ms before the movement onset and was localized the contralateral sensorimotor over area. Desynchronization was also observed over the parietocentral zone with a contralateral predominance before the movement and an ipsilateral diffusion after the movement onset. In Parkinsonian group, ERD appeared also over the contralateral sensorimotor area, with a shorter latency than in the control group, 1250 ms before the movement onset for left flexion and 1000 ms for right flexion. ERD was also noted over the frontocentral area.

Recently, [4] has demonstrated that neurofeedback training is potentially useful for restoring BP1 amplitudes in PD, and helpful for increasing neural activity in cortical motor areas responsible for motor symptoms in PD. This study provides experimental evidence that support the NFB technique as a new nonpharmacological strategy helpful for increasing the neural activity in cortical motor areas.

The use of neurofeedback training with Parkinsons disease was presented by [13] in which a theoretical

framework for using a combination of EEG biofeedback and regular biofeedback with users who have movement disorders was introduced. This framework was demonstrated through the presentation of a case study. NFB training achieved a significant reduction in dystonic movements in this case.

The aim of this work is to develop a neurofeedback tool based on EEG signals (EEG-NFB) that allows users with movement disorders to modulate the mu rhythm to improve the coordination of sequential movements. Furthermore, this tool provides the possibility of evaluating the users progress. It is here developed an EEG-NFB tool that allows healthy subjects to increase their BP and modulate their mu rhythms. This tool can be used in a therapy that helps to improve the coordination of sequential movement and the quality of life of subjects with movement disorder.

# Materials and Methods

# Experimental protocol

An Emotiv EPOC neuroheadset (bandwidth from 0.1 to 100Hz) is employed for acquiring the EEG signals from fourteen electrodes on the scalp [9], placed according to the international 10/20 system [10]: AF3, AF4, F3, F4, F7, F8, FC5, FC6, P7, P8, T7, T8, O1, and O2. However, only the data from primary motor area (PMA) and SMA are used, because these region present a high activation power during motor tasks, and have less artefacts of ocular muscle activity compared to the frontal scalp regions. EEG channels are sampled with 128 Hz at 1.95  $\mu$ V, the least significant bit voltage resolution, and referred to A2 during of experimental protocol.

Four healthy subjects participated in the experiments (approved by the Ethical Committee of UFES/Brazil, number 048/08). Each subject is sit near to a computer screen in a chair, comfortably with their arms and hands on the armrests. In this protocol, the volunteer tries to produce similar suppression using his/her imagination. Before each trial his/her EEG signal are recorded for 2 min to determine the baseline. The task of testing the volunteer needs his/her imagination of the arms movements during 6 s and there is a rest of 6 s. A screen with a black background is then shown and a sound indicates when the task start and when ends. In the time between each task the NFB is show, which is the topographic representation of the brain activity.

**Wavelet power spectral (WPS)**: Although computing power spectral densities for EEG-based on emotion recognition is a very popular method amongst the available feature extraction algorithms, it assumes stationary of the signal over time periods. However, the EEG is not due to its dynamic behavior and this would constrain the Fourier Transformation to extract salient features which may be valuable for affect recognition. On the other hand, nonparametric methods for feature extraction can account for signal non-stationeries as are found in the joint time-frequency domain, such as Wavelet features. Thus, rather than analyzing the signal data set as a whole, wavelets provide a measure for the local frequency analysis, providing information that is likely to be obscured by other alternative time-frequency methods as Fourier analysis. The Wavelet power spectrum is computed by wavelet transform  $\psi(t)$ , defined in Equation 1.

$$\Psi(t) = \frac{1}{\sqrt{a}} \Psi(\frac{t-b}{a}) \tag{1}$$

Where "a" denotes a scaling parameter for the frequency represented by the wavelet, and "b" a shifting factor.

At arbitrary scales between sampling intervals containing the times series, it refers to the continuous wavelet transform (CWT) of a function of time, f(t), expressed as Equation 2, where factors "a" and "b" still determine scale and center of the wavelet.

$$W(f) = \int_{\infty}^{\infty} f(t) \psi(f)$$
 (2)

Given the continuous wavelet transform, we are able to obtain the wavelet power spectrum by essentially squaring the CWT as  $P_W = W (f)^2$ .

In this study, the bandwidth was 2 to 32Hz with a scale factor of 0.5 hz and a "Morlet" Wavelet function that characterizes the overall wavelet shape and covers the domain under study. The algorithm 1 shows the development of NFB ERD-ERS using WPS of the EEG signal for the motor channel (FC5 and FC6 channels of the Emotiv Epoch). The frequency of 10 Hz is selected as the target frequency.

Algorithm 1: Neurofeedback ERD-ERS supplementary area.

1: function [  $Out_1 Out_2$ ] = NFB\_ERD(EEG) 2: EEG(channels, Samples, Trial) 3: BaselineF<sub>C5</sub>,  $\leftarrow$  WPS(EEG, F<sub>C5</sub>,) 4: BaselineF<sub>C6</sub>  $\leftarrow$  WPS(EEG, F<sub>C6</sub>) 5: % obtain Wavelet Power Spectrum 6: % of 20 trials in relax state 7: lineF<sub>C5</sub>  $\leftarrow$  WPS(F<sub>C5</sub>) – BaselineF<sub>C5</sub> 8: lineF<sub>C6</sub>  $\leftarrow$  WPS(F<sub>C6</sub>) – BaselineF<sub>C6</sub> 9:  $Out_1 \leftarrow$  lineF<sub>C5</sub> /(lineF<sub>C5</sub> + lineF<sub>C6</sub>) 10:  $Out_2 \leftarrow$  lineF<sub>C6</sub> /(lineF<sub>C5</sub> + lineF<sub>C6</sub>) 11: return Out\_1, Out\_2

### Results

In this section, the preliminary tests are shown. Figures 2a and 2c show the topographic representation of the brain activity for imagery movement of the right and left arm. Figure 2b and 2d show the distribution on each hemisphere for 20 trials for the complementary arm movements.



(c) Imagery movement of right arm (d) Representation of 20 trials of imagery movement right arm

Figure 2: Representation of the imagery movement for left and right arms.

Table 1 shows the preliminary results for 4 volunteer using the system with feedback. The volunteers 1 and 2 (left-handed) present average results. One of the volunteers, user 4, was not able to active one of the hemisphere in several trials. The value in parentheses corresponds to the result of average percentage of specificity for 20 tasks.

Table 1: Performance using the Neurofeedback tool for 20 trials.

Users	Number of correct tasks (Activation Level)	
	Right Side	Left Side
User 1	13 (0.47)	8 (0.41)
User 2*	15 (0.42)	12 (0.60)
User 3	17 (0.76)	4 (0.70)
User 4	5 (0.44)	3 (0.52)
* Left-handed	user.	· · ·

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## Discussion

The low level activation shown in Table 1 confirms the results described for [14] because task single as hand movements implies low cortical activation.

Volunteer 4 is right-handed and carried out few tasks correctly. On the other hand, the classification for the as left-handed user got the better results: 15 (0.49) and 17 (0.59). Furthermore, this can be related to the fact that this task was very complex for him.

### Conclusions

The preliminary results show that the techniques implemented could be used to build a NFB tool. It can be used to obtain feedback necessary to improve the modulation of the cortical activity. The system does not require training process before the use, and the volunteer can imagine movements of some body parts, but cannot active specific region of the brain. It is worth to mention that the NFB tool shows the user his/her brain active regions. For future works, it is necessary to implement other features of the tool such as to identify the contralateral lateralization ipsilateral of hte user in order to correctly analyze his/her signals.

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#### References

- [1] K. Chaudhuri e W. Ondo, Movement Disorders in Clinical Practice, London : Springer, 2010.
- [2] A. Zaidel, D. Arkadir, Z. Israel e H. Bergman, "Akineto-rigid vs. tremor syndromes in Parkinsonism," Curr Opin Neurol, vol. 22, p. 387– 93, 2009.
- [3] P. E. Roland, E. Meyer, T. Shibasaki, Y. L. Yamamoto e C. J. Thompson, "Regional cerebral blood flow changes in cortex and basal ganglia during voluntary movements in normal human volunteers.," J Neurophysiol, vol. 48, p. 467–80, 1982.
- [4] T. Fumuro, M. Matsuhashi, T. Mitsueda, M. Inouchi, T. Hitomi, T. Nakagawa, R. Matsumoto, J. Kawamata, H. Inoue, T. Mima, R. Takahashi e A. Ikeda, "Bereitschaftspotential augmentation by neuro-feedback training in parkinsons disease," Clinical Neurophysiology, vol. 124, p. 1398 – 1405, 2013.
- [5] A. Suppa, "Boosting neural activity in cortical motor areas through neurofeedback in Parkinson's Disease," Clinical Neurophysiology, vol. 124, pp. 1262-1263, 2013.
- [6] L. Subramanian, J. Hindle, S. Johnston, M. Roberts, M. Husain, R. Goebel e D. Linden, "Real-Time Functional Magnetic Resonance Imaging Neurofeedback for Treatment of Parkinson's Disease," The Journal of Neuroscience, vol. 31, p. 16309–16317, 2011.
- [7] T. Ros, M. A. M. Munneke, L. A. Parkinson e J. H. Gruzelier, "Neurofeedback facilitation of implicit motor learning.," Biol Psychol, vol. 95, pp. 54-8, 2013.
- [8] H. H. Kornhuber e L. Deecke, "Hirnpotentialäderungen beim Menschen vor und nach Willkübewegungen, dargestell mit Magnetbandspeicherung und Rückwärtsanalyse.," Pflügers. Arch. Ges. Physiol., vol. 52, p. 261, 1964.
- [9] H. Jasper e W. Penfield, "Electrocorticograms in man: effect of the voluntary movement upon electrical activity of the precentral gyrus.," Arch. Psychiat. Z. Neurol., vol. 83, pp. 163-174, 1949.

- [10] G. Pfurtscheller e A. Berghold, "Patterns of cortical activation during planning," Electroenceph clin Neurophysiol, vol. 72, pp. 250-258, 1989.
- [11] L. Defebvre, J. L. Bourriez, K. Dujardin, P. Derambure, A. Destée e J. D. Guieu, "Spatiotemporal study of Bereitschaftspotential and Event-Related Desynchronization during voluntary movement in Parkinson's disease," Brain Topography, vol. 6, pp. 237-245, 1994.
- [12] R. Homan, J. Herman e P. Purdy, "Cerebral location of 10-20 system electrode placement.," Electroenceph. Clin. Neurophysiol., vol. 66, pp. 376-382, 1987.
- [13] M. Thompson e L. Thompson, "Biofeedback for movement disorders (dystonia with Parkinson's disease): Theory and preliminary results.," Journal of Neurotherapy, vol. 6 (4), pp. 51-70, 2002.
- [14] W. Kim, C. Gabbard, R. Young e J. Buchanan, "Reaching in contralateral hemispace by righthanders: A kinematic observation," Journal of Motor Behavior, vol. 39, n. 6, pp. 451-456, 2007.