



## Case study

## The use of EEG biofeedback in rehabilitation of a patient with amyotrophic lateral sclerosis with cognitive, mood and motivation disorders

**Joanna Białkowska<sup>1,2</sup>, Dorota Mroczkowska<sup>2</sup>, Joanna Wojtkiewicz<sup>3</sup>, Wojciech Maksymowicz<sup>2,4</sup>**

<sup>1</sup> Department of Public Health, Epidemiology and Microbiology, Faculty of Health Sciences, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland

<sup>2</sup> Clinical University Hospital in Olsztyn, Poland

<sup>3</sup> Department of Pathophysiology, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland

<sup>4</sup> Department of Neurology and Neurosurgery, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland

## ARTICLE INFO

## Article history

Received 12 April 2017

Accepted 19 October 2017

Available online 5 February 2018

## Keywords

Cognitive disorders

Degenerative neuromuscular disease

Mood

Motivation disorders

Neurofeedback

Neurorehabilitation

## Doi

10.29089/2017.17.00012

## User license

This work is licensed under a

Creative Commons Attribution –

NonCommercial – NoDerivatives

4.0 International License.



## ABSTRACT

**Introduction:** Amyotrophic lateral sclerosis (ALS) is primarily a degenerative disease of the nervous system, progressive, and of unknown aetiology. It leads to the limitation of movement (muscular dystrophy), impaired communication with others (speech disorders, dysarthria), dietary problems (dysphagia) and mental dysfunctions.

**Aim:** Our purpose was to assess the neurorehabilitation effectiveness of patient with ALS.

**Case study:** A 71-year-old male patient was diagnosed with ALS. The study of cognitive, mood and motivation disorders was performed using neuropsychological and neurophysiological methods. The authors assessed the impact of the neurofeedback method on EEG neurophysiological parameters: beta, beta2, sensorimotor (SMR) and theta waves. We used 4-channel headbox EEG DigiTrack BF.

**Results:** Neuropsychological diagnosis showed the presence of executive deficits: the ability to plan and perform complex tasks, and distraction in response to an external stimulus. Test for depression showed moderate mood decline and impulsiveness. Fear of the disease was manifested by excessive concentration on health, depersonalization, diurnal mood swings and intense obsessions. Reinforcement of behaviours responsible for an increase in SMR waves (the so-called high alpha 12–15 Hz) was aimed at reducing impulsive behaviour. Our goal was to diminish the amplitude and percentage share of: theta (4–8 Hz) and beta2 (frequency above 18 Hz) waves whose excess was manifested by emotional states, such as anxiety and psychomotor agitation. After a series of 10 sessions, the amplitude of SMR waves in the right hemisphere was increased. In addition, the desired reduction of beta2 waves was achieved.

**Conclusions:** The study suggests that neurofeedback can be used as a neurorehabilitation component of the personalized complex rehabilitation protocol for the ALS patients. The improvement of mental health is largely associated with better patient collaboration in the management of somatic disease by: enhancing motivation for rehabilitation, increasing tolerance of the disease and reducing anxiety.

## 1. INTRODUCTION

Amyotrophic lateral sclerosis (ALS) is primarily a degenerative disease of the nervous system, progressive, and of unknown aetiology. It leads to the limitation of movement (muscular dystrophy), impaired communication with others (speech disorders, dysarthria), dietary problems (dysphagia) and mental dysfunctions. In Poland, the incidence of ALS is 4–6 cases per 100,000 people. The mean age of incidence falls in the 6th to the 7th decade of life; it is more common in men than in women (about 1.5 : 1.0).

Neurofeedback (NFB) is one of the mechanisms known as electroencephalographic (EEG) feedback and involves monitoring of the brain activity by analysing EEG signals.<sup>1</sup> Changes in the bioelectricity of the brain are reflected in human cognitive processes. This assumption allows for the use of NFB in the process of diagnosing and treating neuropsychological defects, and mood and motivation disorders. NFB can also be used as a supportive psychotherapy procedure. It allows the establishment of the harmony of brain wave activity on the principle of bioregulation and self-learning of the brain through video training games.<sup>2</sup>

Studies show that while working with NFB, the human brain learns via conditioning: instrumental and classic.<sup>3</sup> Instrumental conditioning means rewarding the patient for obtaining a psychophysical state of the body which is consistent with thresholds determined for individual brain waves. By working with the therapist, the patient learns to react in the desired manner. In instrumental conditioning of brain waves, the patient acquires the ability to influence the information observed on the screen by changing his/her mental state (e.g., relaxation, concentration) until he/she gets a reward, i.e. a certain number of points (sound and visual feedback). Because the exercise is repeated many times, training activities are fixed and then automatically restored. Classic conditioning in NFB therapy is when the patient learns to transpose into a specific psychophysical state of the body, e.g., the state of concentration is connected with re-

duced free-wave activity and increased high-speed activity. Then, after the end of the training, the patient associates this condition with the performance of certain intellectual tasks.

Table 1 summarizes brain waves and their correlation with mental states.

Instrumental learning with the use of NFB is possible based on the following assumptions:

- mental states can be described by the record of bioelectrical activity of the brain;
- normal brain wave patterns correlate with disorders and psychiatric syndromes;
- a person can learn to influence the pattern of their own brain waves;
- changing brain pattern changes behaviour;
- brain mapping can help diagnose psychiatric syndromes and show patterns of communication between areas of the brain.

## 2. AIM

The aim of the study is to present the use of NFB in rehabilitation of a patient with ALS with cognitive, mood and motivation disorders.

## 3. MATERIAL AND METHODS

A 71-year-old male patient was diagnosed in 2012 with ALS. The study of cognitive, mood and motivation disorders was performed using neuropsychological and neurophysiological methods. The authors assessed the impact of the NFB method on EEG neurophysiological parameters: beta, beta2, sensorimotor (SMR) and theta waves. We used 4-channel headbox EEG DigiTrack BF. The purpose of the therapy was to reduce the number of theta and beta2 waves and strengthen SMR waves.

Quantitative EEG (QEEG) diagnosis was introduced before and after training, and both records were analyzed. The degree of NFB training difficulty was chosen taking into account the individual capabilities of the patient. Workouts were conducted in a series of 10 sessions, 3 times a week. Each session lasted 30 minutes. The study used standard training with mounting electrodes in the central band C3 : C4 (beta/theta : SMR/theta), with classic setting of the frequency bands. The speed of delivering audio feedback was 150-200 ms.<sup>3</sup>

Psychological diagnosis was made using the STAI-Spielberger State-Trait Anxiety Inventory, the Eysenck Personality Questionnaire (EPQ-R), the Beck Depression Inventory (BDI), the Points Matching Test - Common Task Test, and Raven's Progressive Matrices.

## 4. RESULTS

Neuropsychological diagnosis showed the presence of executive deficits. Disruptions in the executive functions

**Table 1. Bioelectric activity of the brain and mental states.<sup>3</sup>**

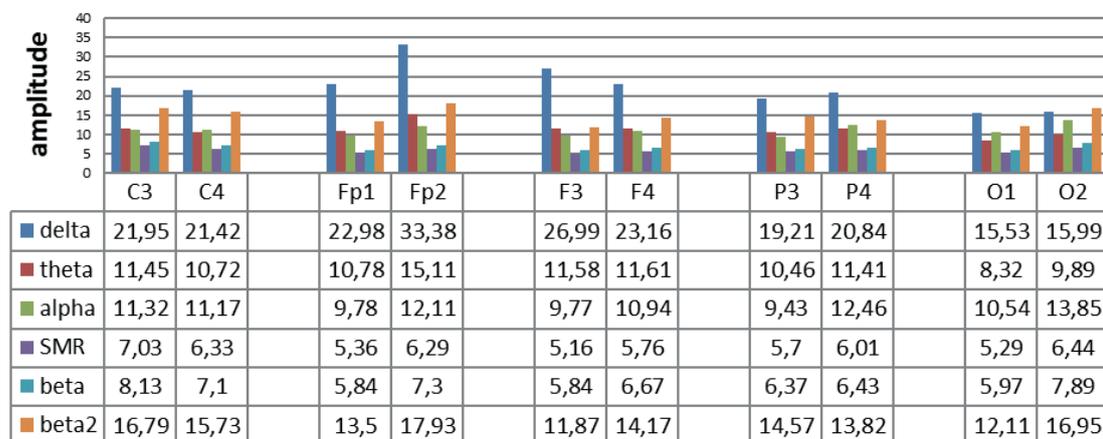
Frequency bands	Cerebral waves	Feature
0.5-3.0 Hz	delta	1. Learning disorders. 2. Brain damage. 3. Motion artefacts.
3-5 Hz	low theta	Drowsiness.
6-7 Hz	high theta	1. Lack of concentration on external stimuli. 2. Internal orientation. 3. Memory extraction and visualization (7.5-8.5 Hz).
8-10 Hz	low alpha	Internal orientation.
11-13 Hz		State of alertness, wide awareness.
13-15 Hz	high alpha	1. State of alertness, high concentration. 2. State of calmness, reduced anxiety in which action is preceded by thought.
16-20 Hz	beta	Cognitive activity, status of mobilization for problem solving.
19-23 Hz	beta2	Anxiety, emotional intensity of mental experiences.
24-36 Hz		Rumination.
38-42 Hz	gamma	Cognitive activity (high attention).

concerned the ability to plan and perform complex tasks, and distraction in response to an external stimulus (CTT1 – ten 43, centile 24; CTT2 – ten 31, centile 3). The following abilities were reduced: visual searching, maintaining and divisibility of attention and sequential processing of information. The assessment of intellectual capacity revealed the functioning of the patient as above average. The ability to concentrate and maintain attention in terms of auditory and visual modality was weakened. Test for depression (BDI-18 points) showed moderate mood decline. Fear of the disease was manifested by excessive concentration on health, depersonalization, diurnal mood swings and intense obsessions. The patient lost interest and willingness to act, work and devote time to hobbies. Slight slowdown in

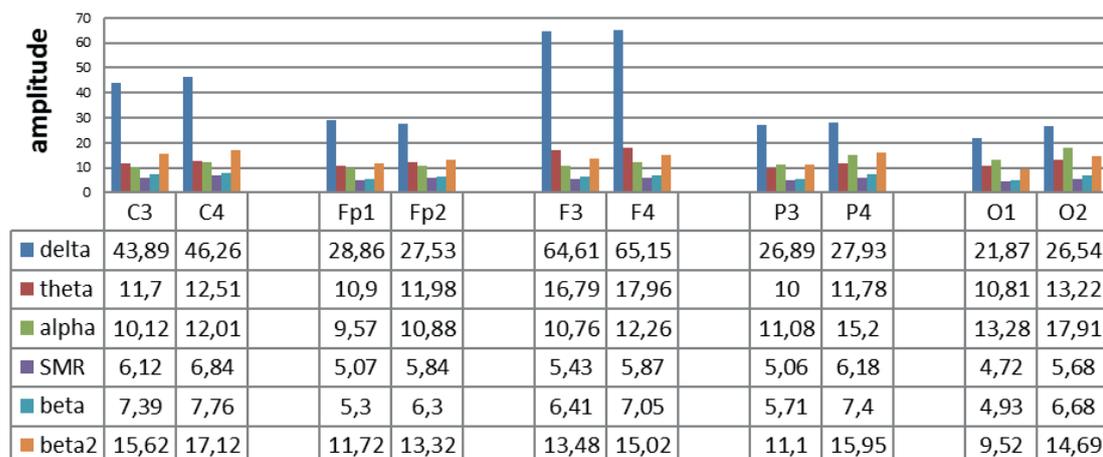
thinking and speech, weakening of concentration, tension and irritability were also reported. The patient felt guilty for disappointing other people, and was discouraged in life. The STAI study (L-feature sten 7, L-state sten 4) identified the elevated level of anxiety manifesting itself as a subjective, consciously experienced feeling of anxiety and tension, accompanied by arousal of the autonomic nervous system. No sleep disorders or suicidal thoughts were observed in the study.

The patient had a limited insight into his own functioning. He was oriented as to the place, time and schema of his own body. Orientation in space was slightly disturbed. Procedural learning and the ability to acquire motor skills and master specific tasks were preserved. In the sphere of

### Amplitudes variability - pre treatment



### Amplitudes variability - post treatment



**Figure 1.** Pre- vs. post-treatment changes in the amplitudes of the given waves for the respective leads in the quantitative EEG analyses.

personality, the EPQ-R questionnaire was used to evaluate mean scores for the following features: psychotism, extravagance, neuroticism, positive self-presentation, and tendency to addiction and criminality.

At admission to the clinical department of neurological rehabilitation the physical examination revealed: flaccid tetraparesis, more pronounced in the left limbs, extensive muscle atrophy within the shoulder girdle and muscles of the limbs, limited fine hand movements, weakening of muscle strength, numerous fasciculations especially in the upper torso, limited mobility of the cervical and lumbar spine, right-sided first degree scoliosis of the thoracic and lumbar spine, and decreased exercise and breathing capacity. In the march test, the patient stepped 192 m with the help of a walker. He complained of pain in the spine and joints (up to 10 points on the VAS scale).

The QEEG performed prior to the initiation of therapy showed the dominance of SMR (12–15 Hz) and beta (15–20 Hz) waves in the left hemisphere (beta band – normal, SMR – abnormal). Delta (1–3 Hz) and theta (4–8 Hz) waves in the central band slightly exceeded the norm. The beta2 frequency (20–34 Hz) in leads C3 and C4 was rated above the norm. The bilateral elevation of beta2 values suggested increased emotional tension, anxiety and mood disturbance. The aim of NFB therapy was to achieve a state of relaxation, reinforce attention and motivate the patient to exercise.

Reinforcement of behaviours responsible for an increase in SMR waves (the so-called high alpha, 12–15 Hz) was aimed at reducing impulsive behaviour. Our goal was to diminish the amplitude and percentage share of: theta (4–8 Hz) and beta2 (frequency above 18 Hz) waves whose excess was manifested by emotional states, such as anxiety and psychomotor agitation. After a series of 10 sessions, the amplitude of SMR waves in the right hemisphere was increased. In addition, the desired reduction of beta2 waves was achieved.

Figure 1 shows pre- and posttreatment changes in the amplitudes of the given waves for the respective leads in the quantitative EEG analyses.

Kinesiotherapy included: respiratory exercises, gait improvement exercises, manipulation exercises, shoulder joint relief exercises, classic shoulder and upper limb shoulder massage, relaxation exercises, PNF exercises, equivalent exercises, occupational therapy, lower limbs relief exercises, lasers for painful areas, lower extremity drooping exercises, pain trigger points mobilization, warm gel packs for painful areas, and TENS currents for the lumbar spine.

Initially, kinesiotherapy was hindered by the depressed mood of the patient and his weak motivation for exercising, and after a few minutes he gave up the rehabilitation. NFB therapy was introduced. NFB training, adjusted to the patient's deficits, improved his mental state: his mood stabilized, motivation for physical rehabilitation increased, and concentration improved.

Complex rehabilitation improved the following parameters: muscle strength in the upper and lower limbs as well as postural muscles, movement coordination, balance and

fine hand movements, respiratory and exercise capacity. In addition pain reduced (up to 3 points on the VAS scale) and gait quality improved. In the march test, the patient stepped 224 m with a walker.

## 5. DISCUSSION

ALS is an incurable disease. In its initial stage, the patient denies the existence of the disease. He cannot accept the diagnosis and starts to become isolated from other people. In the second phase, when he realizes that he can no longer deny the disease, he becomes angry at his inability to control the situation. Before the patient starts to accept the disease and the inevitability of death, he tries to 'bargain.' The awareness of the ineffectiveness of therapeutic actions causes mental disorganization: mood disorders, aggression, passivity, apathy, suicidal thoughts, or addiction. The loss of self-mobility is the end of an important stage in life. Realizing dependence on others often means limiting the socio-occupational functioning.<sup>4</sup> People with fatal illness often suffer from depression, high levels of anxiety, and a lack of motivation to participate in rehabilitation. Patients complain of severe pain. Pain has a huge negative effect on the human psyche, and this directly affects how the patient copes with illness and disability.<sup>5</sup> Pain can be intensified by anxiety or environmental factors. It enhances the feeling of loneliness and rejection, lowers mood, and increases the risk of depression.

Psychotherapy plays an important auxiliary role in the treatment of patients with somatic disease and anxiety disorders. The treatment not only leads to a symptomatic improvement resulting from the cessation of functional disturbances, but also causes a betterment of the somatic status. As the general condition gets better, the patient more eagerly collaborates in the management of the somatic disease by:

- (1) abolition of anxiety that prevents participation in diagnostic tests and necessary consultation;<sup>6</sup>
- (2) increased ability to tolerate illness and discomfort, and acceptance of limitations;
- (3) dealing with internal and external conflicts, and regulating negative emotions that may affect the course of the somatic disease;
- (4) successful resolution of problems resulting from somatic disease-related limitations, mainly through redevelopment of the value system;
- (5) strengthening of motivation for rehabilitation.<sup>7</sup>

The limbic system plays an important role in recognizing and evaluating stressful situations.<sup>8</sup>

The hippocampus and entorhinal cortex are involved in the creation of memory traces and cognitive schematics. Chronic stress can lead to degeneration of dendrites in hippocampal neurons, which explains the cognitive decline.<sup>9</sup> The amygdala holds emotional response programmes throughout life. Despite the lack of real danger, the conditioning of fear generates anxiety in certain situations. Fear attacks may be due to pathological discharges in the

amygdala neurons, as confirmed by functional tests in PET, SPECT.<sup>10</sup> The poor development of connections between the neocortex and limbic system is responsible for alexithymia (difficulty with reading emotions). Inhibitory neurons run from the frontal lobes to the amygdala, but projections in the opposite direction are much more numerous. Perhaps that is why emotions more easily dominate thoughts, and it becomes more difficult for thoughts to control emotions.<sup>8</sup> The presence of coupling between the frontal cortex and the limbic system provides justification for psychotherapeutic activities restoring balance, which create a chance of controlling the disease.<sup>11</sup>

There is evidence for the negative impact of stressors on immune responses.<sup>12</sup> It has been shown that those who experience stress related to the loss of a family member have decreased activity of B and T cells and weakening of NK (Natural Killer) cells.<sup>13</sup>

Since 1997, research into neuroimmunology of depression has been closely linked to studies on stress neuroimmunology. Depression is associated with the inhibition of some elements of the immune response and the concomitant stimulation of, for example, acute phase proteins and proinflammatory cytokines.<sup>14</sup>

On the other hand, the immune system may have an impact on the mental state, mainly by increasing the secretion of cytokines that alter the metabolism of monoamines: serotonin, noradrenaline, dopamine, which are important in the pathogenesis of depression. Cytokines involved in fighting infections, such as interleukin-6 and interleukin-1, can cause depressive symptoms. They reach the hypothalamus by triggering a response to stress (h-h-a axis stimulation) with all its physiological consequences.<sup>15</sup>

NFB is a therapeutic-assistive approach aimed at achieving a psychophysical state characterized by emotional silence, increased motivation, and improved memory and concentration. Thanks to the performed actions, the patient learns to inhibit the excess and amplify the deficiency of specific brain waves and thus change behaviour in a desired manner. In therapy, it is important to strengthen frequencies that are typical but distorted in a given area.<sup>2</sup> The studies have shown that the therapeutic effects of NFB persist for many years, particularly in the cognitive rehabilitation of non-psychiatric individuals.<sup>16,17</sup>

In patients with severe neurological deficits and uncertain prognosis, standard psychological therapy and rehabilitation often do not produce expected results. Incorporating an original element in the form of NFB therapy creates new therapeutic possibilities. In our patient, the therapy brought a significant reduction of pain, improved motivation for rehabilitation, along with improved physical performance and mood. However, doubts about the introduction of new therapeutic methods to standard treatment could ultimately be resolved only in randomized, long-term, double blind studies, with a control group not receiving treatment.<sup>18</sup> Unfortunately, longterm placebo-based research in rehabilitation is often not feasible, because it generates high costs and may be unethical.<sup>19</sup>

In our work, we have highlighted the possibility of organizing clinical trials using biological feedback as a complementary therapy to existing treatments. Choosing exercises (training protocols) and duration of therapy requires further studies.

## 6. CONCLUSIONS

1. NFB can be used to treat psychological disorders and as supportive therapy in rehabilitation.
2. The improvement of mental health is largely associated with better patient collaboration in the management of somatic disease by: enhancing motivation for rehabilitation, increasing tolerance of the disease and reducing anxiety.

## Conflict of interest

None declared.

## Acknowledgement

This is supported by the National Centre for Research and Development Grant STRATEGMED1/234261/2NCFBR/2014.

## References

- 1 Mroczkowska D, Białkowska J, Rakowska A. Neurofeedback as supportive therapy after stroke. Case report. *Post Psych Neurol.* 2014;23(4):190–201. <https://doi.org/10.1016/j.pin.2014.09.002>.
- 2 Okupińska A, Krzywowiąza A. [*Biofeedback as an effective method in cognitive deficit therapy in the theory of mind. Neurocognitive science in the pathology and health*]. Szczecin: Pomoravian Medical University in Szczecin; 2013:195–202 [in Polish].
- 3 Thompson M, Thompson L. [*Neurofeedback. Introduction to the basic concepts of applied psychophysiology*]. Wrocław: BioMed; 2012 [in Polish].
- 4 Adamek D, Tomik B. [*Amyotrophic Lateral Sclerosis*]. Kraków: ZOZ Centre of UMEA Shinoda-Kuracejo; 2005 [in Polish].
- 5 Białkowska J, Sowa M, Maksymowicz W. Exploration of assistance and rehabilitation possibilities for neurosurgical patients with late complications after craniocerebral injuries based on one patient case. *Pol Ann Med.* 2012;19(1):58–62. <https://doi.org/10.1016/j.poamed.2012.04.001>.
- 6 Skulimowska K, Siwak-Kobayashi M, Galińska E. [Problems in psychotherapy of patients with a chronic life-threatening disease, treated in the Neurosis Clinic]. *Post Psych Neurol.* 2000;9:121–128 [in Polish].
- 7 Skulimowska K. [Interactions of the somatic and psychological states in patients diagnosed with somatic disease and neurosis]. *Psychoterapia.* 2011;3(158):41–59 [in Polish].
- 8 Świerkocka-Miastkowska M, Klimarczyk M, Mazur R. [Comprehending the limbic system]. *Psychiatr Prakt Ogólnolek.* 2005;5(1):47–50 [in Polish].

- <sup>9</sup> Gould E, Tanapat P. Stress and hippocampal neurogenesis. *Biol Psychiatry*. 1999;46(11):1472–1479. [https://doi.org/10.1016/S0006-3223\(99\)00247-4](https://doi.org/10.1016/S0006-3223(99)00247-4).
- <sup>10</sup> Aggieton JP. The contribution of the amygdala to normal and abnormal emotional states. *Trends Neurosci*. 1993;16(8):328–333. [https://doi.org/10.1016/0166-2236\(93\)90110-8](https://doi.org/10.1016/0166-2236(93)90110-8).
- <sup>11</sup> Magdoń M. [Integrative way in diagnostics and therapy]. *Sztuka Leczenia*. 1995;1:81–86 [in Polish].
- <sup>12</sup> Miller AH. Neuroendocrine and immune system interaction in stress and depression. *Psychiatr Clin North Am*. 1998;21(2):443–463. [https://doi.org/10.1016/S0193-953X\(05\)70015-0](https://doi.org/10.1016/S0193-953X(05)70015-0).
- <sup>13</sup> Solomon G. [Emotions, immunity and disease. Historical and philosophical perspectives]. *Nowiny Psychologiczne*. 1990;1–2:109–126 [in Polish].
- <sup>14</sup> Connor TJ, Leonard BE. Depression, stress and immunological activation: the role of cytokines in depressive disorders. *Life Sci*. 1998;62(7):583–606. [https://doi.org/10.1016/S0024-3205\(97\)00990-9](https://doi.org/10.1016/S0024-3205(97)00990-9).
- <sup>15</sup> Landowski J. Neuroendokrynology. In: Bilikiewicz A, Pużyński S, Rybakowski J, Wciórka J, eds. [*Psychiatria*]. Wrocław: Urban & Partner; 2002:191–212 [in Polish].
- <sup>16</sup> Egner T, Gruzelier JH. Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *Neuroreport*. 2001;12(18):4155–4159. <https://doi.org/10.1097/00001756-200112210-00058>.
- <sup>17</sup> Fomina T, Lohmann G, Erb M, Ethofer T, Schölkopf B, Grosse-Wentrup M. Self-regulation of brain rhythms in the precuneus: a novel BCI paradigm for patients with ALS. *J Neural Eng*. 2016;13(6):066021. <https://doi.org/10.1088/1741-2560/13/6/066021>.
- <sup>18</sup> Preston E, Ada L, Dean CM, Stanton R, Waddington G. What is the probability of patients who are nonambulatory after stroke regaining independent walking? A systematic review. *Int J Stroke*. 2011;6(6):531–540. <https://doi.org/10.1111/j.1747-4949.2011.00668.x>.
- <sup>19</sup> Kwolek A, Podgórska J, Rykała J, Leszczak J. Use of biofeedback in neurological rehabilitation. *Prz Med Univ Rzesz Inst Leków*. 2013;3:379–388.