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Biofeedback and Neurofeedback in the Treatment of Migraine

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Abstract

Biofeedback is a noninvasive method of measurement of physiological functions where precise instruments measure the slightest changes in body functions. Many of the studies have shown that using biofeedback can reduce the occurrence of migraine or reduce the strength of the pain. Some results from a study suggest that the use of biofeedback in combination with medication is more successful than medication alone in treating migraines. Also, holistic approach by using behavioral technic is necessary to provide maximal results by methods. To more precisely work with patients who suffer from a migraine, it is also important to know the pathophysiology of a migraine. According to relevant research, we combined biofeedback treatment that consisted of a combination of three forms of biofeedback treatment: neurofeedback, breathing, and vascular biofeedback. Combination of treatments in 25 sessions helped the patient with a long history of a severe migraine. Further research of patients suffering from a migraine with different treatment protocols is needed to establish the method.

Keywords: biofeedback, neurofeedback, migraine treatment, pathophysiology, breathing technique

1. Introduction

Migraine headache is one of the most common headaches in the general population, 15% suffering from the European Union population [1] which with disabling symptoms significantly decreases the quality of life of the patients [2]. According to the International Classification of Headache Disorders [3], a chronic migraine is a type of a primary headache occurring in 15 or more days per month for more than 3 months, in which more than 8 days per month of

headache meet the criteria for a migraine with or without aura or respond to specific migraine treatment. Not only in Europe, but also in the world, migraine has today a high incidence. The prevalence of a migraine in Europe is 15%—ranges depending on the individual countries, 12–27.5% [1]. According to data published in 2006, Croatia, with Germany and Denmark, has the highest prevalence of migraine in Europe [1].

Migraine is a disabling neurological condition characterized by episodic attacks of usually unilateral headache, with pulsating character and light and sound intolerance, associated with nausea and vomiting. The tendency to suffer from a migraine has a genetic component, but attacks can be triggered by a series of internal and external factors. Two types of migraine have been described: episodic migraine (EM) (with subtypes migraine with aura and migraine without aura)—in which a typical headache occurs on fewer than 15 days per month—and chronic migraine (CM) with headaches in 15 or more days per month for at least 3 months [3]. It is not rare that an episodic migraine has progression to a chronic migraine. The development of a chronic migraine has been associated with the presence of many risk factors: female sex, older age, low level of education, low-income populations, predisposition for anxiety, depression, sleep apnea or snoring, overweight, history of frequent headache, stressful life events or major life changes, asthma, allergic rhinitis, and caffeine consumption [4]. Because of all these facts about migraines, it is not difficult to think about complexity and longevity of the treatment. Also, many of people who suffer from migraine in their life use more than one treatment to get better results, which is reduced pain and number of migraines. For more than four decades, many different experts have been trying to find the best way to treat a migraine. Because the causes of migraine are not fully clarified as well as the physiology of migraine, so no unique treatment has yet been conceived.

The annual costs of migraine such as diagnosis, treatment, reduced productivity, and absence from work are estimated to be 5 billion euros in the European Union [5]. It follows from the above that a migraine is not only a medical but also a socioeconomic problem. Apart from the economic, the lack of influence of migraine is manifested in the social sphere. This recurrent disease significantly reduces the quality of life of the diseased, as it limits them to perform daily activities. This directly affects both the near and the outer environment and above all the patient's family. Thus, the consequences of a migraine are reflected in all areas of life—family, professional, and social—resulting in dissatisfaction with their own achievements in all these spheres and creating a sense of inefficiency and intolerance, creating a vicious cycle with negative consequences [2]. Therefore, the comprehensive approach to solving this problem is very important, and education, of both the general population and the patients, and raising health care to a higher level, with ongoing support for migraine-sick patients, are indispensable for shaping a healthier society.

The incidence of migraine before puberty is greater in boys than in girls [6]. It grows up to 12 years in both sexes and is the highest in the age range of 30–40 years. After puberty, the ratio changes and increases in favor of women and with 40 is 3.5:1. After 40 years, the strength of the symptoms is reduced (except for women in perimenopause), and the beginning of migraine headaches in the fifties is rare [7]. The prevalence of migraine is higher in the case of white races than in black races and, on the other hand, is proportional to the socioeconomic status [6].

Migraine is a disease with many faces. The most common form is migraine without aura, occurring in about 80% of patients, while migraine with aura occurs in about 20% of the patients [8].

2. Pathophysiology of migraine

Pathogenesis of a migraine has long been a subject of discussion among scientists. It has been considered that typical headaches are caused by intracranial vasodilation preceded by vasoconstriction causing aura—vascular theory. Today it is known that this is not the case, and although new findings have emerged, the exact mechanism and genetic determinants are not yet fully clarified. The admitted neurovascular theory states that causes of migraine lie in neurogenic processes, followed by secondary changes in brain perfusion [7].

For a long time, it was thought that the cause of the aura, which precedes headaches, is cerebral vasoconstriction. Today, this theory is denied, and the aura is explained by neural dysfunction rather than ischemia due to vasoconstriction. The process of cortical widespread depression, described in 1944 by Brazilian scientist Leão, is now associated with the emergence of visual aura [9]. It is a self-stimulating process that is thought to be due to hyperexcitability of the brain.

There is a release of potassium and neuroexcitatory amino acids of glutamate from neuronal endings, whereby the surrounding tissue depolarizes and then a longer period of neuronal activity is observed. Impulses travel by tissue at a rate of 2–6 mm/min—which is the first feature to retrieve parallel with the rate of appearance, progression, and spread of characteristic visual auric symptoms. During this process, there are also molecular events that cause sterile inflammation and changes in brain perfusion. During the aura seizures, studies using positron emission tomography showed initial hyper-phase, followed by reduced cortical blood flow caused by reduced metabolism due to depolarization and associated decreased neuronal activity. Changing the blood flow in the post-anterior direction is followed by the spread of the impulse through the cortex and is not anatomically linked to the site and during the cerebral blood vessels [10].

During the functional magnetic resonance imaging study, blood oxygenation was found to be initially increased, followed by a decrease in oxidative clearance in the occipital cortex, which ranged at 3–6 mm/min—which may again be related to the appearance of visual symptoms of aura [11]. In addition to being associated with oligemia, corticosteroid depression also influences the trigeminal activation of the trigeminovascular system and changes the permeability of the blood-brain barrier and thus generates migraine headaches [12]. Cortical widespread depression leads to activation of trigeminovascular afferent fibers. Because of this activation, prolonged blood flow increases through the middle meningeal artery and extravasation of plasma proteins in the pituitary mater. There is the opening of the neuronal panicles and the release of proinflammatory cytokines. Consequently, there is a sterile inflammation and pain that affects the brain veins [12].

2.1. Pathogenesis of migraine headaches

When trigeminal ganglion stimulation occurs, neuropeptides are released that are key to the emergence of neurogenic inflammation. The key substances are P and calcitonin gene-related peptide (CGRP) [13, 14]. Substance P is released primarily from thin non-ligated C fibers, while CGRP releases A and C fibers. They, within neurogenic inflammation, cause vasodilation (CGRP), protein extravasation, and dural mast cell activation.

There is the release of ions, cytokines, and other inflammatory mediators in the environment of sensory fibers that inject the brain envelope. Due to the presence of these substances, prolonged activation of peripheral nociceptors occurs, which is eventually perceived as pain. Neurogenic inflammation prolongs and enhances migraine headaches. Because of inflammation, there is also sensitization [13, 14]. Sensitization of neurons and neural fibers indicates an increase in their susceptibility. The threshold is lowered, and the magnitude of irritability and area of the irritable area grow [12]. Because of this, the weaknesses of the stimuli at perhaps atypical sites can be perceived as pain. Spontaneous neuronal activation also occurs. There are two forms—peripheral and central sensitization. In peripheral sensitization, it is about capturing primary afferent neurons, while in central sensitization, it is more susceptible to “higher” neurons—those in the trigeminal nucleus and other parts of the brain stem and hemisphere. Sensitization is believed to be responsible for many of the clinical symptoms of migraine. Pulsating pain, strengthening pain due to physical activity, hyperalgesia, and allodynia are associated with sensitization.

2.2. Genetics of a migraine

The association of genetic factors with the onset of a migraine has been first proven in patients with familial hemiplegic migraine (FHM). This is a migraine subtype where an aura appears to be fully reversible motor deficiency [12]. There are three types of family hemiplegic migraines:

- FHM1 is linked to the mutation of the CACNA1A gene, located on chromosome 19p13.1, and encodes for the $\alpha 1$ subunit of the P/Q calcium channel neurons [15]. The P/Q calcium channel manifests multiple expressions in the central nervous system, regulates serotonin and glutamate release in central and peripheral synapses, and is associated with increased susceptibility to cortical widespread depression [16]. With the mutation of this gene, episodic ataxia type 2, paroxysmal disorder causing cerebellar ataxia, migraine-like symptoms, nystagmus, and cerebellar atrophy [17] are associated.
- FHM2 occurs due to the mutation of the ATP1A2 gene encoding the $\alpha 2$ subunit of Na/K ATPase. This gene is found on the 1q23 chromosome, and the mutation causes reduced ATPase oligodendrocyte activity and decreased affinity for potassium ions, leading to the reduced removal of the same from the extracellular space and reduction of retention of glutamate from the synaptic cracks [18]. The elevated concentration of potassium ions and glutamate in the extracellular space results in hyperexcitability of the brain [16]. Because of the emergence of isolated FHM, it is also possible to combine with cerebellar symptoms, childhood convulsions, and the emergence of mental retardation epilepsy [19].
- The FHM3 mutation affects the SCN1A gene (on the second chromosome), which encodes the Nav1.1 voltage channel. The Nav1.1 voltage channel is key to generating and spreading neuronal action potential, and genetic mutation causes excessive activation of action potentials and can alleviate cortical widespread depression through several mechanisms: high trigger rates can lead to increased extracellular potassium concentrations and further depolarization and increase the release excitatory neurotransmitter glutamate [15]. In addition to being associated with the emergence of family hemiplegic migraine, this gene is also recognized as a cause of generalized convulsion in adult and childhood epilepsy [16], generalized epilepsy with febrile convulsions, and myoclonic epilepsy in early childhood [20].

Discovery of the mutations of these genes explains very few migraine cases, but their detection is very important for a better understanding of pathogenesis [21]. Other forms of migraine are most likely to be complicated genetic disorders, where multiple genes are responsible for the occurrence of migraine and in which the gene base is intertwined with environmental factors [12].

3. Diagnosis of a migraine

Diagnosis of a migraine is based on the clinical picture or diagnostic criteria set by the Headache Classification Committee of the International Headache Society [3]. There are two types of a migraine—migraine without aura and migraine with aura. Headaches that occur 15 or more days a month for more than 3 months and 8 or more days of migraine headache are diagnosed with chronic migraine [3].

Specific diagnostic tests for migraine do not exist, and image methods are in most cases not necessary. According to the American Academy of Neurology, the use of radiographic image methods (MSCT, MR) is recommended only if an abnormal neurological status is found and in patients with an atypical clinical history of headaches or headaches that cannot be classified into either a migraine headache or some other primary headache [15]. Differential diagnosis of a migraine without aura includes primarily tensile headache, whereas the differential diagnosis of migraine with aura also involves transitory ischemic attack and partial epileptic seizure. At the setting of diagnosis of a migraine can help presence of auras (the presence of positive phenomena following negative phenomena), the sequence of their occurrence, progression, duration, and possibly the existence of associated symptoms [12].

Also, at diagnostic, it is very important to take an extensive interview to get detailed information on all spheres of life of the person with migraine (frequency, pain, time of occurrence, association with other events, relationship with some period of time, place of appearance of pain and description of pain, susceptibility to events in their own surroundings—greater expectations of oneself or others—sensitivity to criticism, events that could have caused migraines). Being a good listener to hear all the details of the person with migraine is of crucial importance because it also depends on proposing the possible treatment. After an initial interview where we collect all the necessary information, we shall decide together with the person about how to treat a migraine. For biofeedback as a method of treatment, it is very important to find out how much the person is motivated to invest in and separate the time they will devote to these treatments. At some people, it is still a bigger motive to take some medications that will quickly solve their problem.

4. Treatment of a migraine

Migraine headache therapy according to European Federation of Neurological Societies (EFNS) recommendations' indication for individual drugs was elaborated according to EFNS guidelines at three levels [22]:

- Level A—the drug is effective, ineffective, or harmful, as demonstrated by at least one convincing first-level research (prospective, randomized, double-blind clinical study in a representative population sample or systematic review of prospective, randomized, double-blind clinical studies in a representative sample of populations) or with two consistent, convincing second-level studies (prospective, cohort, double-blind research in a representative sample of populations or randomized, controlled research in a representative population sample).
- Level B—the drug is probably effective, inefficient, or harmful, as proven by at least one persuasive research other levels or superior trials of the three (all other controlled studies in the representative sample of the population where the expected outcome is independent of the treatment of the patient).
- Level C—the drug is probably effective, ineffective, or harmful this is proven by at least two-level three trials.

4.1. Abortive migraine therapy

Abortive migraine therapy involves interrupting the headaches in a short time. The choice of the drug and the way it is administered depend on the clinical picture; the strength of a headache; whether it is associated with additional symptoms, such as nausea and vomiting; and the health of the patient itself—the presence of cardiovascular and/or other illness—and pregnancy. Symptomatic therapy works best if early, immediate headaches are given, with a larger dose being more effective than many smaller ones [23]. In the treatment of weaker to moderate headaches without nausea and vomiting, the nonsteroidal anti-inflammatory drugs (NSAIDs) are prescribed for oral use. If a headache is followed by nausea and vomiting, the NSAID and antiemetics are used parenterally [24]. Moderate to severe headaches without nausea and vomiting are treated with specific drugs—triptans in oral or triptan combinations and NSAIDs (sumatriptan and naproxen) [24]. A moderate to severe headache with nausea and vomiting indicated the use of triptan subcutaneously or intranasally [23].

If the number of headaches varies from attack to attack, the patients are taught and prescribed two or more drugs, either orally or parenterally, which they use as needed [25].

Pregnancy is contraindicated in the use of all medicines used to treat a migraine except paracetamol and aspirin and ibuprofen in the second trimester. Triptans may be used with specialist consensus and if the risk to the child for attack and vomiting is greater than the risk of using triptan [26]. Ibuprofen and paracetamol from the NSAID group, domperidone from the antiemetic group and sumatriptan nasally from the triptan group [26], may be used for childhood treatment and adolescent treatment.

4.2. The prophylactic treatment of migraine

The prophylactic treatment of migraine is prescribed to patients to prevent or reduce the incidence and strength of symptoms. Prophylactic migraine treatment should be initiated if headaches significantly reduce the quality of life (family and professional), headaches occur

twice a month or more often, abortive treatment is inadequately effective, and common, long-lasting, and unpleasant aura occurs [26].

It is important to emphasize that the introduction of prophylactic therapy should be discussed with the patient, who should be familiar with the possible adverse effects of therapy and adapt the drug and the dose to each individual patient [26]. The main goals of prophylactic therapy are to reduce the incidence and duration of headaches, improve the quality of life of patients, and prevent progression of transient episodic to chronic migraines. Prophylactic therapy should also be introduced if the patient is suffering from a specific form of migraine that can lead to permanent neurological damage, hemiplegic migraine, basilar migraine, persistent aura without migraine infarction, and migraine infarction [27]. Pregnancy is recommended only for magnesium and metoprolol [28], and flunarizine, propranolol, or topiramate may be used in childhood and adolescence [26]. In the prophylactic treatment of migraine, certain drugs are used such as antihypertensive, antidepressant, antiepileptic, and nonsteroidal anti-inflammatory drugs.

4.3. Non-pharmacological prophylactic treatment of migraine

In addition to drug therapy, preventive procedures include life-changing practices that include sleep hygiene, regular meals, exercise, and avoiding known trigger for reducing migraine frequency. Using techniques such as relaxation exercises, cognitive-behavioral techniques, biofeedback, acupuncture, and transcutaneous electrical stimulation of the nerve (TENS) can also contribute to the prevention of migraine headaches [23].

According to preventive treatment, it is recommended in patients with high frequency of migraine attacks (usually more than 4–5 days per month)—which is always the case in a chronic migraine, but also when attacks are rare, but very severe and disabling (intense pain), or when patients have contraindications or no response to triptans [29]. Relaxation training and biofeedback focus on the perception of pain, biofeedback training focus on the physical response involved in pain persistence, and cognitive-behavioral techniques target the experience of feeling pain. Knowing the factors that produced chronic headaches may allow the patient to modulate the pain. Patients are taught self-regulation techniques to enhance individual control of pain and coping strategies for a chronic headache and reduce migraine-related stress [30]. In the treatment of a migraine, we can certainly combine different methods. Some people can take certain analgesics only in the period of headaches; some take preventive therapy. With the use of nonpharmacological treatments such as biofeedback, according to our experience, the analgesia is gradually diminishing. The use of multiple combinations of treatments always goes on the assumption that a combination of multiple treatments will sooner and faster produce better results.

5. Biofeedback and neurofeedback in the treatment of migraine

Biofeedback is a noninvasive method of measurement of physiological functions. Precise instruments measure the slightest changes of different body functions—which are then

in a clear and understandable manner shown in the form of feedback. The person gets an insight into what is going on inside the body and thus learns to change patterns of behavior to improve health and performance. Any changes that are wanted are rewarded, which leads to learning of the new patterns of behavior [31]. Biofeedback is a common intervention in pain management. For migraine treatment, the most frequently used biofeedback methods have been peripheral skin temperature biofeedback, blood-volume-pulse feedback, and electromyography feedback [31]. Neurofeedback is a method of obtaining feedback on brain processes, that is, a type of training that observes wave activity and is presented to the individual through the screen through video games. It is based on the measurement of specific brain activity patterns that are characteristic of certain cognitive processes or conditions such as attention, concentration, depression, insomnia, anxiety, fears, stress, headache, or migraine. The neurofeedback method functions as a “mirror that you hold in front of the brain.” It gives information on how the brain works. That way the brain can train to function better. It is possible to alleviate feelings of anxiety and anger and increase self-esteem, concentration, and organizational skills. According to symptoms and problems that a person could have, neurofeedback could be used to increase self-esteem and concentration and decrease anxiety and anger. Brain activity can be targeted by the brain function that we call neuroplasticity. Neuroplasticity refers to the ability to change brain activity over time and represents one of the fundamental parts of the human evolution process and is found to be the basis of some mental and health disorder acquisition.

Before training the target change activity in the brain, it is necessary to see what the initial activity is. Electrical activity in the brain is recorded with electroencephalograph, or EEG. The program analyzes brain activity through brain waves and uses, as well as feedback data presented through video games (motion of missiles, cars, changing image size, etc.). Brain activity by measuring brain waves is monitored via a computer interface, whereby the neurofeedback trainer follows brain activity and the client looks at his screen where he traces his brain activity in the form of video games and sound signals. If we want to achieve that the brain produces more rapid waves (important for mental functions and attention), when brain activity is increased precisely at those frequencies, the client will get better in the game or gain more points. If activity increases with slow-wave frequencies (those we want to reduce), then the success in the game will be weaker. Gradually, the brain will react more and more to these instructions and learn a new pattern of brain activity. The client does not have to think about the process of controlling activity that occurs at a subconscious level. The client just needs to relax and let the brain use its own ability to self-regulate. Observing brain activity information through real-time senses is what makes neurofeedback unique and successful. While some aspects of neurofeedback are automated using modern computer technology, each brain is unique, and each individual situation is different. It is therefore very important that the trained neurofeedback therapist takes an individual approach to the treatment of each client.

Focus and emotional balance are important to outstanding performance in all areas of our work and activities. By training certain brain wave patterns in certain areas, we can develop the skill of entering the “zone,” at those times when it is most important for us to be excellent and regardless of the circumstances in which we are located. We must not forget that one of the most important preconditions for excellence is a quality dream. Stress and anxiety can

seriously affect the amount and quality of sleep. Neurofeedback training can reduce anxiety and strengthen the brain activity that will help us sleep again well.

Some studies have shown that using biofeedback can reduce the occurrence of a migraine or reduce the strength of the pain. A German meta-analysis of the efficacy of biofeedback for migraine—account 55 studies—showed medium effect size for all biofeedback interventions and proved stable over an average follow-up phase of 17 months. The frequency of migraine attacks and perceived self-efficacy demonstrated the strongest improvements. Blood-volume-pulse feedback yielded higher effect sizes than peripheral skin temperature feedback and electromyography feedback [32].

Sharff et al. [33] conducted a study to examine the effects of hand-warming biofeedback, as compared to hand-cooling biofeedback and no treatment at all. Sharff et al. found that the children who were in the hand-warming biofeedback group improved more than the comparison groups and sustained this improvement for up to 6 months later.

Results from a study conducted by Grazzi et al. [34] suggest that the use of biofeedback in combination with medication is more successful than medication alone in treating migraines. Results showed a relapse rate of 42.1% (16 of 38) for participants in the medication only group vs. a relapse rate of only 12.5% (2 of 16) for the medication plus biofeedback group at year 3 of follow-up. This study, therefore, suggests that a combination of medication and biofeedback rather than either by itself may perhaps be the best means of treating migraines, specifically transformed migraines.

Vasudeva [35] conducted a study to examine whether migraine sufferers who experienced aura reacted differently to biofeedback/relaxation than those without and if this was accounted for by blood flow velocity. The results have shown the biofeedback group experienced a decline in the severity of their migraine pain and reported about using less medication to treat/control the pain. Furthermore, no association between biofeedback-assisted relaxation and blood flow velocity was found. Therefore, this study provides corroborating evidence for the notion that biofeedback is an effective treatment for migraines. Another migraine study with 62% of participants using neurofeedback reported major or total improvement in their migraines [36]. Per the study, most patients had long histories of migraines and had tried multiple pharmaceutical treatments prior to trying neurofeedback. Most were on medications during the study. Participants took part in an average of 40 sessions over 6 months. For neurofeedback training, they used a different site such as temporal locations (T3, T4), central areas (C3, C4), frontal areas (F3, F4), prefrontal areas (FP1, FP2), and parietal areas (P3, P4) for typically one to two sessions at each location. Seventy percent of the 37 participants showed a 50% or greater reduction in the frequency of their migraines, and only 16% failed to improve at all. Of those who improved, 62% reported major or total improvement in their migraines. The goal of neurofeedback, however, is to reduce, on an ongoing basis, the number and intensity of migraines. Based on these results—and on clinical experience from clinicians around the country—neurofeedback offers the potential for significant relief for anyone still struggling with migraines.

From some studies, in the electrophysiological activities of migraine sufferers, there are certain abnormalities, so it is understandable that interventions using EEG may be beneficial

at migraines [37, 38]. For example, a study in children with a migraine, with or without aura, shows an increase in frequency theta compared to the control group [37]. One of the used neurofeedback protocols for a migraine emphasizes the brain activity reward of 12–15 HZ at T3 and T4 sites [37]. Siniatchkin et al., in their research, showed a significant reduction of migraine in 10 young people after 10 sessions of neurofeedback in central frontal and central areas by teaching them control of sporadic cortical potential activity that represents cortical sensitivity and reactivity [39]. Michael Tansey in his work with four people with migraine after neurofeedback training in the central frontal and central area showed a decrease in low frequency that became less dominant and strengthened faster frequency [40]. Also, neurofeedback training includes a newer method called hemoencephalography (HEG) that is used on the frontal lobe with gaining information on heat value and learning to increase the frontal temperature or forehead temperature [41]. The elevation of PIR HEG signals reflects the composite thermal activity generated by vascular supply, vascular return, and brain cell activity. Changes of the heat signal from the underlying prefrontal cortex reflect the degree of engagement and increase of neuronal activity. This method also, according to literature, helped to reduce migraine pain as well as the frequency of their headaches in people with migraine diagnoses [37, 41, 42].

According to relevant research, we started a combined biofeedback treatment as a combination of three forms of biofeedback treatment: neurofeedback, breathing, and vascular biofeedback. After an initial assessment and extensive interview, we decided together with the person to combine multiple treatments at one visit. First, we did neurofeedback treatment, followed by breathing with the diaphragm for 10 min and then vascular training, with the aim of learning to achieve vasoconstriction. Treatments started at the beginning of September 2015 and completed at the end of December 2015. During the treatment, a headache diary was conducted by the person.

Our treatment goals were an improvement of the quality of life and increase in everyday functioning by reducing the symptoms related to the primary diagnoses.

Before we started treatment, we have done an initial assessment which included:

- Analyses of medical documentation (conducted diagnostic and therapeutic procedures).
- Structured interview.
- Measuring of baseline EEG (one channel, Cz).

With neurofeedback as a method, we chose to train a relaxed focus or sensorimotor rhythm (SMR) with maintaining muscular relaxation and reducing the internal anxiety and tensions most commonly occurring in the fast beta activity (high beta amplitude) above 22 Hz and may be associated with stress as well as other psychological events. Given that it was a young person, but also loads with her law study, the idea was to strengthen the relaxed focus with neurofeedback and reduce internal tension. Along with the effectiveness of neurofeedback that we have been able to see in improving performance as well as at some difficulties during

our decades of use, we thought that it is a good choice to work with a migraine. After neurofeedback, breathing training with the diaphragm lasted for 10–15 min with the aim of relaxing the body through muscle relaxation and breathing training that would lead to vasodilation and relaxation of the whole body by stimulating the parasympathetic action of the autonomic nervous system. Subsequently, we have been using vasoconstriction-vasodilation training with the aim of enabling a person to learn that vasoconstriction occurs when a migraine occurs. The training is done in such a way that the sensor is set to a temporalis with a signal that must be at least 10 μ V. During the 21-min training sessions, we had 4-min vasoconstriction training and 1-min relaxation training alternate. Choosing all three training sessions at the same time was to prompt positive changes in pain reduction as well as migraine rates.

The reason for choosing that particular way of training (neurofeedback, breathing, vasoconstriction training) was to have one relaxation training between two difficult pieces of training. It is not so easy to work one hour and a half with maximum work of the person. We also could recommend monitoring peripheral temperature as a secondary parameter during vasoconstriction training, but we did not consider it necessary to include it because the effectiveness of the training achieves vasodilation, that is, a certain blood flow, which also increases the peripheral temperature. Of course, it would be useful to put the sensor in peripheral temperature for future research. We did not use EMG biofeedback, it was not necessary in this particular case. HEG would certainly be useful as a method and probably use it to have it in the software. The device we used in training was NEXUS-10 MARK II and software BIOTRACE+, of the Dutch company Mind Media.

The training lasted 4 months during which the person came two to three times a week for complete training; breathing training was used daily. During the 4 months, a headache diary was also conducted. During the training session, there was no migraine, or a headache was on pain scale 3, on the scale of 1–10.

Implementation of neurofeedback was by using protocols that are determined individually according to the initial assessment, and mean duration of each session was 30 min. Electrode position was according to the international 10–20 systems, and frequency bands were inhibited or rewarded. Administered protocols were on Cz, C4, and C3. Neurofeedback was used to increase sensory motor rhythm (SMR) in the sensory-motor area, to decrease high beta activities and to learn to maintain a relaxed state with a clear focus and concentration. Below is a description of the neurofeedback training with a detailed performance about working on individual points according to the international 10–20 systems:

Cz: inhibition of theta waves (4–9 Hz), strengthening of SMR and beta waves (12–15 Hz), and inhibition of high beta (22–30 Hz)—ten sessions.

C4: inhibition of theta waves (4–9 Hz), strengthening of SMR and beta waves (12–15 Hz), and inhibition of high beta (22–30 Hz)—seven sessions.

C3: inhibition of theta waves (4–9 Hz), strengthening of SMR and beta waves (14–18 Hz), and inhibition of high beta (22–30 Hz)—eight sessions.

Figure 1. Vascular training with BVP sensor on a. temporalis (session 2).

Figure 2. Vascular training with BVP sensor on a. temporalis (after 1 month).

Implementation of biofeedback included vascular training which means 25 sessions of vascular training on a. temporalis, learning the modality of vasoconstriction and vasodilatation. Each training session was 30 min. We used the device that had the vascular training. The goal was to have the amplitude of BVP signal smaller at training phase than relax phase. The results of the few training we shown in **Figures 1–3**.

Figure 3. Vascular training with BVP sensor on a. temporalis (after 3 months).

5.1. Breathing technique

Breathing is a willing and reluctant function, which means we can, but we do not have to pay attention to breathing. Breathing is under control of the breathing center located in the extended spinal cord, which means that the breathing takes place without our influence. It is controlled by an autonomic nervous system that manages various functions in the body and is often not recognized or affected by them.

How then to affect the function of breathing? Breathing can be controlled by a conscious mechanism of breathing an attempt to relax the body and slow down the physiological processes. Today, when the challenges—and therefore stressors—in everyday life are very intense, body relaxation for the sake of health is very important. People who use certain breathing techniques feel their benefit. They are more relaxed, they feel happier, and they do not feel in their own body of great pressures of everyday life. To explain why breathing is important and how it affects a person, we need to look at the vascular system that is also affected by the autonomic nervous system. The most frequent changes due to the great pressures in life most people feel in that system. Mostly, it is a disruption of heartburn, accelerated breathing, a sense of losing air, and “kicking in the heart”—all these are symptoms that lead to bad feelings and the inability to function every day.

Common respiratory changes are associated with retardation or acceleration of the blood-stream and are caused by the sympathetic and parasympathetic autonomic nervous system. The first accelerates it, and the other serves to slow down all the functions in the body. The same happens with breathing—in the sympathetic work of the autonomic nervous system, the breathing is faster, shallow, and irregular, while in the parasympathetic effect, breathing is slower, which most commonly occur during rest and sleep without our influence. For the body to function normally, it is necessary to balance both systems.

Figure 4. “Breathe in” with the diaphragm.

Experts engaged in the vascular system of research have found that reducing cardiac variability leads to a greater inclination to the bloodstream disease, and deaths are also more common. Also, biofeedback use aims to increase heart rate variability by using breathing techniques. So, breathing is a tool that we use not only to relax but to influence the well-being of the bloodstream system. The breathing used in the training itself is breathing with the diaphragm (breathing with belly) that breathes the lowest part of the respiratory system—the diaphragm—as we show in **Figure 4**.

Breathing training at a migraine was used by breathing with the diaphragm in the duration of minimum 10 min at one session. The person had instructions to use this breathing also at home at least once a day taking care that the breathing is taking place without the influence of external distractors. Breathing technique is a recommended method for relaxation and to reduce stress which is important for people with a migraine. Breathing with the diaphragm is the healthiest breathing we can use. The aim is to provide the best possible exchange of oxygen and carbon dioxide. The body of the belly breathing curves upward, so that the lower part of the chest is enlarged and the lower part of the lung fills with air, as the belly breathes. With this breathing, we get a full effect: the fullness of the ribs, the bones, the spine, and the scurvy and thus the higher lung capacity.

In people who are often affected by different stressors, the use of breathing techniques is of utmost importance. Breathing encourages the parasympathetic action of the autonomic nervous system that slows down and relaxes our body. Breathing is a mechanism that affects HRV and changes that will lead to optimum functioning of the individual.

The daily use of breathing with the diaphragm for at least 10 min leads to the stimulation of protective mechanisms in our body and the creation of so-called protective “receptors” that protect the body from long-term adverse effects of stress. Below is a detailed description of breathing by using the diaphragm.

5.2. Instruction for breathing training—10 min

1. Take a comfortable position and remove anything that sticks around you.
2. Relax the upper body muscles (face, neck, shoulders, upper back, arms).

Figure 5. Slowly “breathe in” through your nose about 3 seconds and inflate your belly such as balloon.

Figure 6. With open mouth slightly, and slowly exhale by counting to six (until you out all the air).

3. If you are feeling comfortable, place your palms on the belly, at the height of the navel.
4. Close your eyes and relax your body.
5. Slowly breathe through your nose counting in yourself by three, inflating your belly as if you were blowing a balloon (**Figure 5**).
6. Try to keep the rest of the body relaxed and not lift it up.
7. It is important that you take as much air as is enough to fill your belly.
8. When you are breathing out, open your mouth slightly, and slowly exhale by counting to six (until you breathe all the air) (**Figure 6**).

Only the body will stimulate your next breath and repeat the cycle. Exercise at least once a day. You can use it several times if you feel the need for relaxation.

6. Results and discussion

According to the treatment of the person with a migraine, we have done a total of 25 treatments. Three modalities were used at each treatment: neurofeedback, vascular training, and breathing techniques. Duration of one treatment was 1 h and 30 min. The training sessions were done 2–3 times per week. The results were a reduction in the frequency of migraine attacks, as well as a reduction in pain severity during the attacks. The reduction in the frequency of migraine attacks and in the strength of pain was gradual. In September,

the person had nine migraines, with pain ranges between 8 and 9. In October, the person had six migraine attacks (3 less), pain strengths from 5 to 8. In November there were five migraine attacks, with a pain score from 2 to 7. In December the number of migraine attacks was four, which is a reduction in frequency by more than 50% since the beginning of the training in September. The pain strength was from 3 to 5, which is also a reduction of more than 40% from September. The person learned to apply the vasoconstriction method when she was experiencing migraine, which reduced the strength of the pain. Also, breathing technique became normal training used daily. The efficacy of biofeedback is evident in the application of the method, for the reduction of a migraine as well as pain relief. Within 4 months of treatment, the person had fewer migraine attacks, and the pain was reduced to such extent that it did not require the use of analgesics. Our experience has shown that it is important to take a good initial assessment and know all relevant information of a migraine as well as to the person itself and to agree with the person on possible treatment goals as well as the incidence of arrivals. The emphasis is on the motivation of the person as well as encouraging motivation, which is always it happens as the frequency and strength of headaches are reduced.

7. Conclusion

Our case study has shown the good curative potential of biofeedback and neurofeedback treatments at a migraine. We confirmed earlier research results that different types of biofeedback methods could have some benefits to people with migraine. Also, we gave some our way of training. Combination of treatments (neurofeedback, breathing, and vascular training) in 25 sessions helped the female patient with a long history of a severe migraine. Further research of patients suffering from migraine with different treatment protocols is needed to establish the method. Therefore, a comprehensive approach to solving this problem is very important, and education, to both the general population and the patients, and the raising of health care to a higher level, with ongoing support for patients suffering from a migraine, are indispensable for the formation of a healthier society. Combination of pharmacologic and behavioral treatments such as relaxation training and cognitive behavior therapy can lead to faster and better results with people who suffer from migraine [43, 44]. Empirically validated behavioral treatments include biofeedback training; relaxation training, combinations of the two, stress management training, and cognitive behavioral therapy could be helpful for people with migraine headache. According to earlier research and our case, we could have recommended a different type of biofeedback methods such as temperature, vascular training, neurofeedback, HEG, and EMG biofeedback. The choice is depending on therapist and client with migraine. Migraines as a disease with many faces need the multidisciplinary and combined approach which provides the possibility of faster achievement of the goals of a therapist and a person with a migraine, which are a reduction pain and the number of migraines. By achieving these goals, we enable a person to have a good and quality life.

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References

- [1] Stovner LJ, Zwart JA, Hagen K, Terwindt GM, Pascual J. Epidemiology of a headache in Europe. *European Journal of Neurology*. 2006;**13**(4):333-345. DOI: 10.1007/s10194-010-0217-0
- [2] Menken M, Munsat TL, Toole JF. The global burden of disease study implications for neurology. *Archives of Neurology*. 2000;**57**(3):418-420. DOI: 10.1001/archneur.57.3.418
- [3] Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 3rd edition (Beta version). *Cephalalgia*. 2013;**33**:629-808. Available at <https://www.ichd-3.org/> [Accessed: 05.12.2017]
- [4] Diener HC, Solbach K, Holle D, Gaul C. Integrated care for chronic migraine patients: Epidemiology, burden, diagnosis and treatment options. *Clinical Medicine*. 2015;**15**(4): 344-350. DOI: 10.7861/clinmedicine.15-4-344
- [5] Linde M, Gustavsson A, Stovner LJ, Steiner TJ, Barré J, Katsarava Z. The cost of headache disorders in Europe: The Eurolight project. *European Journal of Neurology*. 2012;**19**(5):703-711. DOI: 10.1111/j.1468-1331.2011.03612.x
- [6] Lipton RB, Stewart WF, Diamond S, Diamond ML, Reed M. Prevalence and burden of migraine in the United States: Data from the American migraine study II. *Headache*. 2001;**41**(7):646-657. DOI: 10.1046/j.1526-4610.2001.041007646.x
- [7] Chawla J. Migraine Headache. U: Medscape, Lutsep HL ur. Medscape [internet] New York, NY: Medscape; 2016. Available at: <http://emedicine.medscape.com/article/1142556-overview> [02.1.2018]
- [8] Hansen JM, Lipton RB, Dodick DW, Silberstein SD, Saper JR, Aurora SK, et al. Migraine headache is present in the aura phase: A prospective study. *Neurology*. 2012;**79**(20):2044-2049. DOI: 10.1212/WNL.0b013e3182749eed
- [9] Ayata C, Jin H, Kudo C, Dalkara T, Moskowitz M. Suppression of cortical spreading depression in migraine prophylaxis. *Annals of Neurology*. 2006;**59**(4):652-661. DOI: 10.1002/ana.20778

- [10] Woods RP, Iacoboni M, Mazziotta JC. Brief report: Bilateral spreading cerebral hypoperfusion during a spontaneous migraine headache. *The New England Journal of Medicine*. 1994;**331**(25):1689-1692. DOI: 10.1056/NEJM199412223312505
- [11] Lauritzen M. Cortical spreading depression in a migraine. *Cephalalgia*. 2001;**21**(7):757-760. DOI: 10.1111/j.1468-2982.2001.00244.x
- [12] Cutrer FM, Huerter K. Migraine aura. *Neurologist*. 2007;**13**(3):118-125. DOI: 10.1097/01.nrl.0000252943.82792.38
- [13] Goadsby PJ, Lipton RB, Ferrari MD. Migraine—Current understanding and treatment. *The New England Journal of Medicine*. 2002;**346**(4):257-270. DOI: 10.1056/NEJMra010917
- [14] Durham PL. CGRP-receptor antagonists—A fresh approach to migraine therapy? *The New England Journal of Medicine*. 2004;**350**(11):1073-1075. DOI: 10.1056/NEJMp048016
- [15] Silberstein SD, Dodick DW. Migraine genetics: Part II. *Headache*. 2013;**53**(8):1218-1229. DOI: 10.1111/head.12169
- [16] Nye BL, Thadani VM. Migraine and epilepsy: Review of the literature. *Headache*. 2015;**55**(3):359-380. DOI: 10.1111/head.12536
- [17] Ophoff RA, Terwindt GM, Vergouwe MN, Eijk R, Oefner PJ, Hoffman SMG, et al. Familial hemiplegic migraine and episodic ataxia Type-2 are caused by mutations in the Ca²⁺ channel gene CACNL1A4. *Cell*. 1996;**87**(3):543-552. DOI: 10.1016/S0092-8674(00)81373-2
- [18] Ferrari MD, Klever RR, Terwindt GM, Ayata C, van den Maagdenberg AMJM. Migraine pathophysiology: Lessons from mouse models and human genetics. *Lancet Neurology*. 2015;**14**:65-80. DOI: 10.1016/S1474-4422(14)70220-0
- [19] Di Lorenzo C, Grieco GS, Santorelli FM. Migraine headache: A review of the molecular genetics of a common disorder. *The Journal of Headache and Pain*. 2012;**13**(7):571-580. DOI: 10.1007/s10194-012-0478-x
- [20] Dichgans M, Freilinger T, Eckstein G, Babini E, Lorenz-Depiereux B, Biskup S, et al. Mutation in the neuronal voltage-gated sodium channel SCN1A in familial hemiplegic migraine. *Lancet*. 2005;**366**(9483):371-377. DOI: 10.1016/S0140-6736(05)66786-4
- [21] Burstein R, Nosedà R, Borsook D. Migraine: Multiple processes, complex pathophysiology. *Journal of Neuroscience*. 2015;**35**(17):6619-6629. DOI: 10.1523/JNEUROSCI.0373-15.2015
- [22] Brainin M, Barnes M, Baron JC, Gilhus NE, Hughes R, Selmaaj K, et al. Guidance for the preparation of neurological management guidelines by EFNS scientific task forces—Revised recommendations 2004*. *European Journal of Neurology*. 2004;**11**(9):577-581. DOI: 10.1111/j.1468-1331.2004.00867.x
- [23] Bajwa ZH, Smith JH. Preventive treatment of migraine in adults. U: UpToDate, Post TW ur. UpToDate [Internet]. Waltham, MA: UpToDate; 2016 [Pristupljeno 15.11.2017]. Dostupno na: http://www.uptodate.com/contents/preventive-treatment-of-migraine-inadults?source=search_result&search=preventive+treatment+migraine&selectedTitle=1~150

- [24] Becker WJ. Acute migraine treatment in adults. *Headache*. 2015;**55**(6):778-793. DOI: 10.1111/head.12550
- [25] Taylor FR, Kaniecki RG. Symptomatic treatment of migraine: When to use NSAIDs, triptans, or opiates. *Current Treatment Options in Neurology*. 2011;**13**(1):15-27. DOI: 10.1007/s11940-010-0107-4
- [26] Evers S, Afra J, Frese A, Goadsby PJ, Linde M, May A, Sandor PS. EFNS guideline on the drug treatment of migraine—Revised report. *European Journal of Neurology*. 2009;**16**:968-981. DOI: 10.1111/j.1468-1331.2009.02748.x
- [27] Silberstein SD. Practice parameter: Evidence-based guidelines for migraine headache (an evidence-based review): Report of the quality standards Subcommittee of the American Academy of Neurology. *Neurology*. 2000;**55**(6):754-762. DOI: <https://doi.org/10.1212/WNL.55.6.754>
- [28] Goadsby PJ, Goldberg J, Silberstein SD. Migraine in pregnancy. *British Medical Journal*. 2008;**336**(7659):1502-1504. DOI: 10.1136/bmj.39559.675891.AD
- [29] Stanescu I, Dogaru G. Treatment in chronic migraine: Choice of rehabilitation strategies. *Balneo Research Journal*. 2015;**6**(4):217-223. DOI: 10.12680/balneo.2015.1108
- [30] Carod-Artal FJ. Tackling chronic migraine: Current perspectives. *Journal of Pain Research*. 2014;**7**:185-194. DOI: 10.2147/JPR.S61819
- [31] Schwartz MS, Andrasik F. *Biofeedback: A Practitioner's Guide*. Guilford Press. 2003. Chapt;**14**:275-334. DOI: 10.5298/1081-5937-44.4.09
- [32] Nestoriuc Y, Martin A. Efficacy of biofeedback for migraine: A meta-analysis. *Pain*. 2007;**128**(1-2):111-127. DOI: 10.1016/j.pain.2006.09.007
- [33] Sharff L, Marcus DA, Masek BJ. A controlled study of minimal-contact thermal biofeedback treatment in children with migraine. *Journal of Pediatric Psychology*. 2002;**27**:109-119. DOI: <https://doi.org/10.1093/jpepsy/27.2.109>
- [34] Grazzi L, Andrasik F, D'Amico D, Leone M, Usai S, Kass SJ, Bussone G. Behavioral and pharmacologic treatment of transformed migraine with analgesic overuse: Outcome at 3 years. *Headache*. 2002;**42**:483-490. DOI: 10.1046/j.1526-4610.2002.02123.x
- [35] Vasudeva S, Claggett AL, Tietjen GE, McGrady AV. Biofeedback-assisted relaxation in migraine headache: Relationship to cerebral blood flow velocity in the middle cerebral artery. *Headache*. 2003;**43**:245-250. DOI: 10.1046/j.1526-4610.2003.03048.x
- [36] Stokes DA, Lappin MS. Neurofeedback and biofeedback with 37 migraineurs: A clinical outcome study. *Behavioral and Brain Functions*. 2010;**6**:9. <http://www.behavioraland-brainfunctions.com/content/6/1/9>
- [37] de Tommaso M, Trotta G, Eleonora Vecchio E, Ricci K, Siugzdaitė R, Stramaglia S. Brain networking analysis in migraine with and without aura. *The Journal of Headache and Pain*. 2017;**18**:98. DOI: 10.1186/s10194-017-0803-5

- [38] Schoenen J. Neurophysiological features of the migrainous brain. *Neurology Science*. 2006 May;27(Suppl 2):S77-S81. DOI: 10.1007/s10072-006-0575-1
- [39] Siniatchkin M, Averkina N, Andrasik F, Stephani U, Gerber WD. Neurophysiological reactivity before a migraine attack. *Neuroscience Letter*. 2006;400(1-2):121-124. Epub 2006 Mar 15. DOI: 10.1016/j.neulet.2006.02.019
- [40] Tansey MA. A neurobiological treatment for migraine: The response of four cases of migraine to EEG biofeedback training. *Headache Quarterly: Current Treatment and Research*; 1991:90-96
- [41] Carmen JA. Passive infrared hemoencephalography: Four years and 100 migraines. *Journal of Neurotherapy*. 2004;8(3):23-51. DOI: 10.1300/J184v08n03_03
- [42] David CS, Evans JR. *Clinical Neurotherapy, Application of Techniques for Treatment*. San Diego, CA: Elsevier Inc.; 2014. ISBN: 978-0-12-396988-0
- [43] The Medical Roundtable: Behavioral Approaches to Headache and Migraine Management; General Medicine Edition. 2017;1(2):131-144. Available at https://themedicalroundtable.com/journal/general_medicine. Article ID: GM68961
- [44] Sullivan A, Cousins S, Ridsdale L. Psychological interventions for migraine: A systematic review. *Journal of Neurology*. 2016;263:2369-2377. DOI: 10.1007/s00415-016-8126-z