I. INTRODUCTION

Pain is a necessary “evil.” To feel pain is to be forewarned that something is wrong in the body. Moreover, when the pain becomes chronic and debilitating, it is definitively related to more than a specific event or body area. It has been demonstrated that the chronic pain patient shows dysfunctional structural and emotional impairment in cortical regions (Acerra and Moseley, 2005). Fox and Raichle (2007) indicate that long-term pain disrupts the balance of the “default mode network: (DMN), producing a widespread impact on overall brain function. These disruptions of the DMN are believed to be related to the associated depression, anxiety, sleep disturbances, and faulty decision-making which plague chronic pain patients.” Pain has become one of the most common symptoms for which patients are requesting medical attention (more than 80% of medical visits). This care has cost an excess of $80 billion each year in the US (as reported by the Institute of Medicine (Osterweis et al., 1987).

As defined by the International Association for the Study of Pain (IASP), “Pain is unquestionably a sensation in a part or parts of body but is always unpleasant and therefore also an emotional experience” (Merskey 1986). Pain symptoms have been evaluated and addressed variously over time, projecting numerous models for understanding the overall pain experience. From ancient times until Descartes, in the 1700s, pain has been considered a disease condition. Thus, within this “biomedical pain model,” pain is considered a symptom associated with physical damage, purportedly having an objective element connected with the sensation. However, identifying physical pathology does not predict severity of pain, intensity or level of disability (Holroyd et al., 1999).

II. PSYCHOLOGICAL PAIN ASSOCIATED WITH PHYSIOLOGICAL CONDITIONS

The first attempt to integrate the physiological with psychological pain aspects was achieved by Melzak and Wall in 1965 through development of the “gate theory” of pain. This theory incorporated the three systems that are involved in pain...
experience: sensory-discriminative, motivational-affective, and cognitive-evaluative. This model integrates the notions that pain can be somatic as well as psychogenic.

In the light of the gate theory of pain control, one must understand the pathways involved in pain experience. In short, the pain is recorded by specialized peripheral sensory receptors, and the information is brought to the brain through the spinal cord, via afferent pathways. A half century ago, Penfield and Jasper (1954) described these pathways wherein the brain receives the information in specialized centers and performs analysis of the pain’s intensity, quality, and the peripheral localization. This information is integrated in other areas as well, such as limbic system memory of pain, and the emotional encounter caused by the pain sensation. The brain and spinal cord also have the capacity for diminishing the pain sensation by sending, via efferent pathways, messages to reduce the pain experience (the pain control “analgesia” systems are described in any neurophysiology manual).

The psychological aspects described by the gate theory were revisited and considered incomplete by others (Sufka and Price 2002). In spite of the limitations of the mechanisms proposed within this theory (Nathan, 1976), the gate theory of control of pain is considered to have provided an extraordinary contribution to the understanding that chronic pain includes brain function. The mechanisms of pain, incorporating peripheral receptors, pain pathways, and cortical and subcortical centers where pain is perceived, have brought emphasis to the importance of the corticalization of pain. Understanding corticalization of pain (Birbaumer et al., 1995), and neuroplasticity (Ramachandran and Rogers-Ramanchandran, 2000), may explain why neurofeedback is such a valuable technique. Neurofeedback proposes that by teaching self-regulation, a patient can reduce or even eliminate pain sensations. Since the advent of the gate theory, numerous therapeutic modalities have evolved based on the neurophysiological mechanisms of pain (e.g., neurostimulation procedures, pharmacological interventions, behavioral treatments, and neuromodulation techniques through neurofeedback).

There are no two individuals who perceive pain or other physical symptoms similarly. The perception of pain is based partially on an individual’s pain tolerance and pain threshold. The bio-psychosocial make-up determines the pain perception of different individuals as well. Often the subjective complaints cannot be corroborated by any objective measurements. To understand chronic pain, we need to understand the distinction between “disease” and “illness.” To be “diseased” implies that an individual presents objective anatomic-pathological disturbances in their body, while “illness” represents the “subjective experience” of that individual. Thus chronic pain, as a subjective phenomenon, must be evaluated using the bio-psychosocial model that focuses on illness.

III. THE BIO-PSYCHOSOCIAL MODEL OF PAIN

The intricate interaction among biological, psychological, and social aspects of one’s life must be considered. The biomedical model is too limited as a formulation
to understand the complexity of chronic pain syndromes. A comprehensive historical perspective of the bio-psychosocial model is thoroughly presented by Gatchel and Turk (1996) and Turk and Gatchel (1999), and also in Craig's statements including the theory on social learning mechanisms (1986).

Operant learning mechanisms of pain, as discussed by Fordyce (1976), represented a unique direction, and were expanded over the years by others such as Cairns and Passino (1977). Pain is a symptom that is acute as a response to an injury. If the pain persists for more than 6 months since the onset, it becomes chronic. Chronicity of pain is based on the respondent learning mechanisms. However, such learning theory approaches ignore the brain/body interface. Not only are the assaults on the body, which first generated the experience of pain, ignored, but the brain's emotional responses to bodily insults are not considered. To accomplish a successful self-regulatory program through the biofeedback/neurofeedback (BF/NF) treatment approach, considerations should be given to evaluate the underlining etiologies of chronic pain syndromes as well as overlapping diagnosis or co-morbidities (see Table 16.1). For example, patients who suffered from chronic pain usually suffer from depression, anxiety, fatigue, sleep disorders, hypertension, and cognitive disabilities, diabetes, etc. (Turk et al., 1995).

The frequent pairing of chronic pain with several other chronic disabilities may not be coincidental. As Baliki et al. (2008) demonstrated in chronic pain patients, the default-mode network (DMN) shown by fMRI is imbalanced and shown to be disrupted, and this disruption may account for the development of accompanying diagnostic conditions. Therefore, neurofeedback’s effect in restoring the brain functions in general may be based on its contribution to aid in the correction of this cortical disruption. These findings are elaborated in the discussion section of this chapter.

There are various factors perpetuating pain syndromes that contribute to the chronicity of pain, which also need to be addressed (Table 16.2).

Pain is a symptom that affects many people in our society, causing an alarming dependence on pain medications (Kudrow 1982; Mayo Clinic 2007; News-Medical.Net 2007). Chronic pain’s effect on perpetuating other disabilities is enormous. According to the 2005 National Survey on Drug Use and Health, the incidence of new non-medical users of pain relievers is now at 2.2 million

<table>
<thead>
<tr>
<th>TABLE 16.1</th>
<th>Etiology of Chronic Pain of our patients’ population and Overlapping Diagnoses of our patients’ population</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiology of Chronic Pain</strong></td>
<td><strong>Overlapping Diagnoses</strong></td>
</tr>
<tr>
<td>Head Injuries due to different accidents: STBI, MTBI, CVA</td>
<td>Anxiety and/or Depression</td>
</tr>
<tr>
<td>Work related injuries due to repetitive muscle activities</td>
<td>ADHD</td>
</tr>
<tr>
<td>Post surgery</td>
<td>Asthma</td>
</tr>
<tr>
<td>Post inflammation (ie: post shingles)</td>
<td>Cancer – different localizations</td>
</tr>
<tr>
<td>Psychological or Idiopathic</td>
<td>Essential Tremor or Parkinson's</td>
</tr>
<tr>
<td>Chronic degenerative diseases</td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Sleep Disorders</td>
</tr>
<tr>
<td></td>
<td>Memory impairment</td>
</tr>
</tbody>
</table>
TABLE 16.2 Factors perpetuating and aggravating pain syndromes

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mechanical stressors</td>
</tr>
<tr>
<td></td>
<td>Structural asymmetry</td>
</tr>
<tr>
<td></td>
<td>Poor Posture</td>
</tr>
<tr>
<td>2</td>
<td>Nutritional deficiencies</td>
</tr>
<tr>
<td></td>
<td>Avitaminosis, Poor or imbalanced diet</td>
</tr>
<tr>
<td>3</td>
<td>Metabolic and Endocrinologic abnormalities</td>
</tr>
<tr>
<td></td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td></td>
<td>Hyperuricemia</td>
</tr>
<tr>
<td>4</td>
<td>Secondary Psychosocial Factors</td>
</tr>
<tr>
<td></td>
<td>Adjustment Disorder</td>
</tr>
<tr>
<td></td>
<td>Psychosomatic Disorder</td>
</tr>
<tr>
<td></td>
<td>Secondary Gain</td>
</tr>
<tr>
<td>5</td>
<td>Chronic Infections</td>
</tr>
<tr>
<td></td>
<td>Bacterial, viral, fungi, etc</td>
</tr>
<tr>
<td></td>
<td>Immune Deficit Syndromes</td>
</tr>
<tr>
<td>6</td>
<td>Sleep Disorders</td>
</tr>
<tr>
<td></td>
<td>Sleep Apnea</td>
</tr>
<tr>
<td></td>
<td>Bruxism</td>
</tr>
<tr>
<td>7</td>
<td>Neurologic Disorders</td>
</tr>
<tr>
<td></td>
<td>Radiculopathies</td>
</tr>
<tr>
<td></td>
<td>Entrapment neuropathies</td>
</tr>
<tr>
<td></td>
<td>Peripheral neuropathies</td>
</tr>
<tr>
<td></td>
<td>Multiple Sclerosis</td>
</tr>
<tr>
<td>8</td>
<td>Rheumatologic Disorders</td>
</tr>
<tr>
<td></td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td></td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>Systemic lupus erythematosus</td>
</tr>
</tbody>
</table>

Americans age 12 and older, surpassing the number of new marijuana abusers (2.1 million). Also, more than 6 million Americans reported current non-medical use of prescription drugs—more than the number abusing cocaine, heroin, hallucinogens, and inhalants combined.

IV. THE USEFULNESS OF BF/NF WITH CO-MORBIDITIES ASSOCIATED WITH CHRONIC PAIN

It would be erroneous and an oversimplification to suggest that biofeedback/neurofeedback alone would effectively assist the chronic pain patient to overcome co-morbidities associated with their pain. Other therapies that have been shown to be beneficial in pain management are mentioned in Mosby’s Complementary and Alternative Medicine (Freeman, 2004). They include behavioral modification techniques (Block et al., 1980), acupuncture (Price and Meyer, 1995) and Chinese medicine, Alexander technique, Bachflower remedies, chiropractic, craniosacral therapy, Feldenkrais, Hellerwork, homeopathy, meditation, physiotherapy techniques such as heat, CES, TENS, ultra sound, laser, and Shiatsu, massage, yoga, etc.

Nonetheless, using BF/NF as a central modality, many of the causes, co-morbidities, and perpetuating factors of pain often have been addressed successfully with BF/NF techniques. These co-morbidities include high blood pressure and cognitive dysfunctions (Ibric and Grierson, 1995), and sleep disorders (Ibric, 2001). NIH recommends
the use of BF/NF in pain syndromes and sleep disorders, and the same recommendations also are found in Freeman (2004). The addictive behaviors associated with chronic pain have been addressed with NF, resulting in remarkable success (Ibric 2002; Guyol, 2006). Some of the patients presented in the case study section of this chapter completely renounced their pain medication regimen, as the NF training progressed.

Biofeedback (BF) protocols designed to address the peripheral correlates of arousal, such as temperature (TMP), muscle tension (EMG), sweat gland activity (SCR), and heart rate variability (HRV), indirectly affect pain parameters. By comparison, neurofeedback (NF) directly affects the processing of pain perception. By modifying the electrical activity of the central processing units at cortical and sub-cortical areas, the pain perception, pain memory and pain affect are modulated, and pain tolerance and pain thresholds enhanced. We also hypothesize that NF produces an enhanced process of analgesia, due to endorphine and enkephaline stimulation (see the discussion section of this chapter).

Over the last 50 years numerous studies reported biofeedback (BF) and relaxation as useful complementary techniques to medical approaches in treating acute and chronic pain syndromes (Arena and Blanchard, 1996). Examples include: headaches (Brown 1974; Budzynski et al., 1973; Tansey 1991); temporomandibular joint (TMJ) pain (Ham and Packard, 1996); neck, shoulder and back pain (Carlsson 1975; Cairns and Passino, 1977); and myofascial pain syndrome (MPS) (Toomim, 1987; Ibric, 1996). Early in its history neurofeedback (NF), or EEG-BF, as a sub-specialty of BF, was reported useful in epilepsy (Sterman 1973), in obtaining a relaxation response (Jacob and Benson 1996) and enhancing creativity (Brown and Klugg, 1974; Budzynski, 1976; Fehmi, 1970; Green, 1972, and Kamiya, 1968), and in attention deficit problems (Lubar, 1985; Tansey, 1993). As the years went by, the applications of NF expanded to psychological and physical dysfunctions, such as migraine in children (Sinatchkin et al. 2000, Kropp et al., 2002), headaches (McKenzie et al., 1974), sleep disorders and pain syndromes (Ibric and Jacobs, 1997, Ibric and Kaur, 1999, Ibric, 2000, 2001, 2002; Donaldson et al., 2004a-d; Othmer, 2001) as well as depression, anxiety, and chronic fatigue (Rosenfeld et al., 1995; Hammond, 2000, 2001).

V. OVERVIEW OF THE COMPLEXITY OF TREATING CHRONIC PAIN

This presentation of our clinic’s experience demonstrates the difficulties in stylizing protocols for chronic pain treatment. The co-morbidities often overlap—one person’s pain experience itself is very unlike another’s, social and home conditions superimpose upon the chronic pain experiences, and the EEG patterns associated with pain demand their own unique training locations and training modifications. Over 10 years of practice, 147 patients were referred to us for biofeedback training for different chronic pain syndromes, ranging from migraine to complex regional pain syndromes (see Table 16.3). These patients had been previously treated with other modalities, but had little resolution of pain.

From the total of 147 patients who were statistically analyzed at the end of this chapter, 10 cases are presented in Table 16.4, and will be detailed below.
Three longitudinal studies (Case studies 8, 9 and 10) are included among these 10 patients. Case number 10, presented in more detail, demonstrates the progress of NF training and its effects on subjective and objective measures. The QEEG evaluation of the real-time changes on connectivity during NF and HEG modification during a NF session are also shown to indicate the totality of changes of all measurements on the brain. The efficacy study of the NF training in pain syndromes will conclude this chapter.
FIGURE 16.1  Psycho-physiological evaluation on Biocomp instrument.

A. Data collection of subjective and objective symptoms

Our clinic’s methods of evaluation of chronic pain patients are comprehensive, and include a complete medical history followed by a psycho-physiological profile (PPP) using a Biocomp instrument. The PPP consists of measures of the peripheral level of arousal via electromyography (EMG), of skin temperature (TMP) and sweat gland activity, or skin conductance response (SCR), as patients are engaged in resting, then mental and emotional stress. An example of PPP is presented in Fig. 16.1.

The evaluation process includes objective cognitive tests such as TOVA (test of variables of attention) or IVA (integrated visual and auditory), and the MAS (memory assessment scale). Results of stress tests, depression or CES-D (Radloff 1977), anxiety tests, SCL–90R, and the level of pain evaluated with the chronic pain grade questionnaire (CPG) as VAS (visual analog scale) and/or the McGill questionnaire (Melzack 1975), are very important subjective parameters that aid in measuring progress.

The Electroencephalography (EEG) was obtained with an NF instrument, such as the Neurocybernetics or ROSHI. This provides us with information concerning electrical activity of the brain. We are especially interested in the sensory–motor area corresponding to the Penfield homunculus (Fig. 16.2) (Penfield and Jasper, 1954).

A quantitative EEG (QEEG), using the Lexicor Neurosearch–24, gives us further important and detailed information regarding various parameters such as relative and absolute magnitude, asymmetry, phase, and coherence using various software programs, e.g., Neuro–Guide, Nx–Linx, NeuroRep and LORETA. We are using the QEEG primarily in cases of traumatic brain injuries (TBI). The results obtained guide us more precisely in designing an appropriate protocol to assist in the neuromodulation of the client’s pain perception. Recently we introduced the
Neurofeedback in pain management

Hemoencephalogram (HEG) measurements to monitor the blood perfusion in the NF trained areas in some of our chronic pain patients. (An example is presented in the case studies section.)

B. Training strategies

The specific training strategy chosen is decided primarily by the BF modality that we, and others, have found to produce the best result in lowering the pain perception. In our practice, peripheral BF was useful in correcting the perception of tension headaches, myofascial pain syndromes, some TMJ, and in conducting neuromuscular re-education. Others have reported such BF useful as well, e.g., Sime (2004).

Nevertheless, in our practice NF has been the modality of choice in treating complex chronic pain syndromes, such as fibromyalgia, and RSD or complex regional pain syndrome I, CRPS type I (Ibric, 1996). In certain complex pain syndromes associated with many co-morbidities, the NF training is enhanced by light and/or electromagnetic stimulation, using ROISHI instruments. In short, the ROISHI instrument, designed to aid in the neurofeedback training, uses an algorithm, developed by Chuck Davis, named complex adaptive modality. This involves extracting the error aspects of one’s EEG (for example, features such as transients) from the wideband EEG, and feeding back to the client non-error features of his or her brain activity in the form of colored LED light flashes and/or electromagnetic pulses, very much in real-time (Ibric and Davis, 2007).

After a comprehensive evaluation (Ibric, 1996), electrodes are placed in positions individualized for each case based on the type or localization of pain, and located according to 10/20 international system (Jasper, 1958).

FIGURE 16.2 Penfield’s homunculus presenting the sensory and motor cortical areas where we first analyze the EEG, and often where the NF training starts.

- Motor cortex: movement
- Somatosensory cortex: somatic sensation
Patients trained for 20 consecutive NF sessions, 45 minutes long, were then re-evaluated with the same battery of tests as those used at the intake. The number 20, for the number of NF sessions, before first re-evaluation followed the literature (Marzano et al., 2001) that describes the “learning curve” for any learned skill. We also have found and reported earlier (Ibric and Dragomirescu, 2005) that 20 sessions were “necessary and almost sufficient” for the NF to be effective. The results of our statistical analyses on NF efficacy are presented at the end of this chapter.

To monitor progress, stress tests and depression/anxiety scales were used periodically, and the VAS for pain was used pre/post each NF training. NF was done either as “traditional NF” (audio-visual NF) using a Neurocybernetics system, and/or “NF enhanced by light or electromagnetic closed-loop EEG” (CL-EEG), using the ROSHI instrument. For example, we had discovered earlier that myo-fascial pain syndrome (MFPS) responded well to “traditional NF,” versus fibromyalgia, or CRPS (chronic repetitive pain syndrome) that required NF enhanced by light and/or electromagnetic CL-EEG to have positive results. The employment of various light colors (LED or light emitting diodes) in the “eyesets” or goggles, and electromagnetic stimulation, was determined case by case, taking into consideration each person’s emotional response to colors. For example, it has been observed that red and indigo colors are useful in cases were pain is accompanied by depression, while green and blue are necessary to reduce associated anxiety. Patients with different pain syndromes required different numbers of NF sessions.

VI. CASES STUDIES 1–7

Case 1


Symptoms: Right side face shoulder/arm, right eyelid spastic ptosis, depression, sleep disorder.

Etiology: MVA 3 years prior to investigating BF.

Other therapies: Neck surgery, physical therapy, electrical stimulator implanted.

Medications: Antidepressants, Vicodin.

NF training: 15 sessions, NF protocols were done mostly at C3 site (where Beta [15–18 Hz] was enhanced while Theta [4–7 Hz] and High Beta [22–30 Hz] suppressed.) Neurocybernetics was the instrument used. The C3 site corresponds to the right eye projection, at the sensory-motor area of the homunculus (see Fig. 16.1 above).

NF results: Patient was able to open the right eye as presented in Figs 16.3a and 16.3b, and have minimal pain over the right face, neck, shoulder and arm. In parallel, after 30 minutes of NF, the EEG activity normalized (Figs 16.3c and 16.3d). After the first session, the eye stayed open for 1 hour. As the NF continued, after each session the same effect occurred as described in the first session, except that it was
Neurofeedback in pain management

FIGURE 16.3A Patient with RSD—right eye presents palpebral ptosis—before NF.

FIGURE 16.3B Same patient as in Fig. 16.3a after 30 minutes of NF training, on Neurocybernetics instrument trained at the C3 position/Beta (15–18 Hz) enhanced.

FIGURES 16.3C and 16.3D EEG presentations of Case 1 (recorded on Neurocybernetics), corresponding to Figs. 16.3A and 16.3B respectively.
sustained for a longer and longer time. After 15 sessions, NF training was terminated due to her family leaving the state. This case was presented at the Myofascial Pain Syndrome Symposium sponsored by Discovery International (Ibric, 1996).

Case 2

Patient description: A 67-year-old retired actress (recently widowed) with chronic headaches, neck and low back pain, spasticity post-meningitis, and hypertension as co-morbidity.

Symptoms: Headaches, right side neck spasticity, gait dysfunction, depression/anxiety, sleep disorders, memory and concentration impairments, urinary incontinence.

Etiology: Three neck surgeries after an MVA that occurred 20 years prior to investigating BF. Last surgery was done a year before BF and that induced meningitis.

Other therapies: Surgery, psychotherapy, physical therapy, acupuncture.

Medications: Lorcet, Neurontin, Elavil, Restoril, Depakote, Norvasc, Baclofen, Hydrochlorothiazide, Duragesic patches, Synthroid, Cardura, Norco 10-350, Vitamin B complex, Vitamin E.

NF training: 145 sessions. Neurocybernetics and ROSHI instruments were used for her NF training. The electrode positioning varied from CZ to C3, or C4, C3-Cz, Cz-C4 and F3/F4, or C3/C4, enhancing either SMR, 15 Hz, or correcting coherence. EEG patterns modified, from a great variability to a more stable activity.

NF results: Pain perception modified and decreased to none, less depressed or anxious, better and more restful sleep, lowered blood pressure, better gait, and better quality of life. Patient was able to enjoy travel, and she was able to move to a new house, which she was unable to do, since her husband died. Able to reduce her meds by half, under her physician’s supervision.

Case 3

Patient description: A 33-year-old retired construction engineer, neuropathy post-TBI and spinal cord injury.

Symptoms: Neck, shoulder, upper/lower back, arms and legs pain, spasticity, tremor, gait dysfunction, memory problems, depression, panic attacks, sleep deprivation, neurovegetative deregulation (temperature fluctuations with profuse sweats, paroxysmal tachycardia, blood pressure with large fluctuations!), which were exacerbated by Elavil (discontinued).

Etiology: TBI due to work injury that affected the brain stem (post-16 ft ladder fall).

Medications: Zoloft, Mirapex, Baclofen, Buspar, Vicodin, Sonata, Ambien, Elavil.

NF training: Total of 82 sessions. Protocols on NC (8) and on ROSHI (74).

NF results: Decreased pain, tremor and spasticity reduced, less depression, better sleep, reduced medication.
As an example, the Case 3 protocols were designed following the peripheral symptoms, such as for legs spasticity, and pain; the electrodes were placed over the central sensory motor area over the vertex at the Cz position. The ROSHI training was set for complex adaptive modality (CAM) for the light stimulation, to inhibit high beta frequency over 25 Hz, or HiBeta[I]. The effect was enhanced by using the electromagnetic stimulation, concomitantly. The hands tremor ceased when the electrodes were placed over the C3/C4 positions (where the motor control projection of the hands is located on Penfield's homunculus), and the training was designed to enhance S14 (SMR 14) or SMR, 12–15 Hz (while Theta and HiBeta were discouraged). The sessions done on ROSHI I, monitored and recorded on Neurocybernetics, and the changes in EEG presentation shown in Figs 16.4A and 16.4B.

In both Fig. 16.4A and 16.4B there are three panels. The first, at the top, represents the EEG evaluation at the C3 position; the second, in the middle, is at C4 position and the third, at the bottom, at Cz position. The changes, from before NF (Fig. 16.4A) and after NF (Fig. 16.4B), in the amplitude and the variability of all the frequencies analyzed (Theta, SMR and HiBeta) are obvious.

FIGURE 16.4A Examples of brain waves of case 3 recorded on NC—before NF training on ROSHI.
Case 4

**Patient description:** A 20-year-old student with RSD left foot.

**Symptoms:** Chronic pain of left foot migrating to the right, headaches, and cognitive dysfunctions due to meds.

**Etiology:** A heavy metal object fell on her left foot, 2 years prior to investigating BF.

**Other therapies:** Physical therapy, acupuncture.

**Medications:** Various antidepressants, Vicodin, Motrin.

**NF training:** 20 sessions. Protocols mostly over the central sensory area at the Cz or Cz/C4 positions using the Neurocybernetics (11) followed by the ROSHI (9) NF instruments.

**NF results:** Pain reduced from 8 (0–10 VAS) to 2–1. Able to return to school.

Case 5

**Patient description:** 63-year-old retired engineer with idiopathic neuropathy and hypertension as co-morbidity.

**Symptoms:** Pain in both legs, level 9 (0–10 VAS) anxiety, sleep disorder, hypertension.
Neurofeedback in pain management

Other therapies: Physical therapy.
Medications: Neurontin, Norvasc.
NF training: 22 sessions. Protocols on NC Cz SMR (2), and on ROSHI (20) with light and electromag stimulation F3/F4 alpha inhibit.

Results: Pain reduction down to none, anxiety controlled, better sleep, reduced Neurontin.

Case 6

Symptoms: Chronic pain low back and legs, level 8 (0–10 VAS), depression, attempted suicide, addiction to pain meds.
Etiology: Laminectomy for chronic low back pain 8 yrs prior to NF.
Other therapies: Surgery, psychotherapy, Palade exercises, yoga.
Medications: Neurontin, Wellbutrin, Vicodin.
NF training: 46 sessions. Protocols on NC (4), Cz SMR, and on ROSHI (42), F3/F4 the protocols varied from AO[I] or alpha only inhibit (8), to S14 reward (4), B16 reward (16), B17 (5) and Sync enhance (9). The complex adaptive modality (CAM) of light stimulation was always used with ROSHI.1

NF results: Pain reduction down to none, no more depression, reduced meds, no more Vicodin.

Seven years after the NF training ended, the learned skills continued to benefit the client, and enhanced his performance.

Case 7

Patient description: A 35-year-old student with MFPS, chronic headaches (multiple origins) and (PTSD), Bruxism.
Symptoms: Headaches due to dental problems or sinus infections or allergies, left TMJ, teeth grinding, depression, anxiety, anger, sleep disorders.
Other therapies: Chiropractic, massage therapy, sinus surgeries, psychotherapy.
Medications: Neurontin, Depakote, Vicodin, Acetaminophen, Motrin, Relafen, Diazepam, Loracet, Relafen, Baclofen, Tegretol, Serozone, Lidocaine Infusions, Antihistamines (Zyrtec, NavCon-A, Albuterol, as needed).
NF training: 15 sessions. Protocols used as needed at Cz or C4 SMR and C3 Beta (some sessions done with alternation of C3 beta followed by C4 SMR) using Neurocybernetics instrument.

NF results: Headache and TMJ pain reduced from 8–9 to 2–1, and emotional correction of depression. Anxiety reduced from 8 to 2–0. Improved cognitive functioning with the reduction of the meds. Three months post the 15th session the normalization of the brain wave activity sustained, and she was able to resume school.
VII. LONGITUDINAL CASE STUDIES (8–10)

Case 8

*Patient description:* A 51-year-old teacher with chronic neck/shoulder or N/S pain, and TMJ/RSD; co-morbidity, rheumatoid arthritis.

*Symptoms:* Severe chronic pain (left neck/shoulder, TMJ, ear), numbness of left hand; sleep disorders—insomnia, teeth grinding; fatigue, nervousness.

*Etiology:* MVA 4 years prior to investigating BF.

*Medications:* Relafen, Plaquenil, Prozac, Serozone (Elavil, Zoloft, Sinequal in the past), HRT for menopause.

*BF:* 2 sessions without any positive results.

*NF training:* 22 sessions on NC, resumed NF after a 5-month break, then continued to session 51 on NC, followed by three re-evaluations.

*NF training:* At Cz or C4—SMR enhanced, and theta and high beta discouraged.

*NF results:* TMJ and neck/shoulder pain level was lowered from 8–9 to 2–4, and was gradually reduced and kept at acceptable levels of 1–3 on the visual analog scale, or VAS (see Figs. 16.5–16.9) for a longer time, even after the NF ended.

The pain reduction for session number (n) was estimated by subtracting from the declared pain at the end, the declared pain at the beginning of each session, or Postn–Pren. For every session n, the relative reduction of pain (Rn), compared to the pain perceived before the first session (Pre1), was calculated by using the formula: Rn = (Postn–Pren)/Pre1. (See for cases 8 and 9, the Figs 16.6 and 16.8, and Figs 16.11 and 16.13, respectively).

FIGURE 16.5A  TMJ pain, pre and post each NF session in time. Note: Session 22 done after a 5-month break; session 51 marked the end of NF series; session 52 is a re-evaluation.
FIGURE 16.5B  N/S pain, pre and post each NF session in time. Note: session 22 done after a 5-month break; session 51 marked the end of NF series; session 52 is a re-evaluation after another year.

Case 8—temporomandibular joint pain

FIGURE 16.6  TMJ pain relative reduction \( R_n \), based on VAS. \( R_n = (\text{Pre}_n - \text{Post}_n) / \text{Pre}_1 \). \( \text{Pre}_n \) and \( \text{Post}_n \) are VAS values Pre and Post, respectively, in session number \( n \).

Case 8—learning curve TMJ

FIGURE 16.7  The learning curve for controlling TMJ pain, following NF training sessions (where \( C_1 = R_1 \) and \( C_n = C_{n-1} + R_n \)).
To quantify the clients’ learned control of pain over n sessions, we constructed the cumulative learning curves presented in Figs 16.7 and 16.9 and 16.12 and 16.14, for cases 8 and 9 respectively. For example, the client, after session one learned to reduce pain in proportion $R_1$, therefore $C_1 = R_1$. After a second session the client learned from previous session ($n-1$) plus the current session (n), or $C_n = C_{n-1} + R_n$.

**Case 9**

*Patient description:* 60-year-old housewife with chronic pain. Co-morbidities: Parkinson’s disease (PD), skin and colon cancer, chronic lymphatic leukemia (CLL).

*Etiology:* 2 years prior BF fell and injured left knee, diagnosed also with CLL and PD.

*Symptoms:* Severe lower back and left knee pain, level 8–9 (0–10 VAS), spasticity of left foot, numbness of the left hand, tremor, depression, anxiety, sleep disorder, tinnitus.

*Medications:* Cinemet, Trazadone, Zoloft, Lodosyn, HRT, Oscal and multivitamins.

*NF training:* Protocols total of 56 sessions on NC (Session 54 after a 6-month break; session 55 and 56 follow-up evaluations at 1½ and 2½ years after NF ended,
Results: Spasticity lowered from 8–10 to 3 post 20 sessions, and reduced to none after 50 sessions. Tremor more controlled and better after each session, imperceptible after 50 sessions. Pain reduction to 2–1, anxiety/depression controlled, better sleep, reduced meds (under physician control). Pain continued to be under control even after 1½ or 2½ years, after the NF training ended (see Figs 16.10–16.14).

NF treatment effectiveness in back pain control

FIGURE 16.10A Case 9 presents the reduction of back pain following each NF session. Note: Session 54 after a 6-month break; session 55 and 56 follow up evaluations 1½ and 2½ years after NF ended, respectively.

NF treatment effectiveness in knee pain control

FIGURE 16.10B Case 9 presents the reduction of knee pain following each NF session. Note: Session 54 after a 6-month break; session 55 and 56 follow up evaluations 1½ and 2½ years after NF ended, respectively.
FIGURE 16.11  Back pain relative reduction ($R_n$), based on VAS. $R_n = (\text{Pre}_n - \text{Post}_n)/\text{Pre}_1$.

FIGURE 16.12  The learning curve for controlling back pain, following the NF training ($C_1 = R_1$ and $C_n = C_{n-1} + R_n$).

FIGURE 16.13  Knee pain relative reduction ($R_n$), based on VAS. $R_n = (\text{Pre}_n - \text{Post}_n)/\text{Pre}_1$.

FIGURE 16.14  The learning curve for controlling knee pain, following the NF training ($C_1 = R_1$ and $C_n = C_{n-1} + R_n$).
Neurofeedback in pain management

Case 10

Patient description: A 42-year-old carpenter with left inguinal pain post-surgery and kidney stones (185 over the years), MTBI, chronic colitis, addictions to pain killers/marijuana and smoking.

Symptoms: Depression/anger, chronic fatigue, left inguinal pain, low back pain.

Etiology: Hernia surgery and epidydimectomy 4 years prior to investigating BF; work injury (hit with a construction wood log of 2 by 4 at the posterior right side of the head).

Other therapies: Acupuncture, chiropractic, herbs.

Medications: Vicodin, Morphin (repeated ER visits), Iboprufen 2400 mg, Epinephrine. Note: All meds stopped since the NF training started to help (after the first 15 sessions).

NF training: 112 neurofeedback sessions. Neurocybernetics (9); C4 SMR, C3 Beta, P3 Alpha [E]; ROSHI (103); enhanced NF by light or electromagnetic closed-loop EEG: F3/F4 alpha only inhibit, AO[I]; theta only inhibit, TO[I], or theta 4, T4[I]; P3/P4, alpha only enhanced, AO[E]; C3/C4 or Cz SMR; F3/F7, synchronization inhibit, Sync [I]; Fp1/T3 Sync [I]. When NF was completed, continued the home training with a pROSHI (personal ROSHI entrainer/disen-trainer, non-NF instrument).

NF Results: Pain reduction from 9–10 immobilizing pain to 3–1, and complete elimination of painkillers. Able to go back to work, and produced musical CDs due to enhanced mental performance.

The progress during the NF training monitored by periodic re-evaluations of the stress response, SCL-90R, and depression scale (CES-D) is shown in Figs. 16.15A, 16.15B and 16.15C. These three figures show continued improvement of this patient’s subjective responses. The cognitive re-evaluation also shows increased mental performance, while during pain episodes altered cognition (Fig. 16.15D).

QEEG

The case’s 10 results of the QEEG connectivity map, using NeuroRep program, are presented in Fig. 16.16A, and the response to the ROSHI light enhanced NF is shown in Figs. 16.16B and 16.16C. There are disconnected (blue) areas of the brain and other areas hyper-connected (dark orange). These data are part of an experiment we have done to evaluate real-time changes in the brain connectivity during NF, which is described in detail in Hudspeth and Ibric (2004) and Ibric et al., 2007 (in press). The nZ-score in eyes-closed condition is 335 (Fig. 16.16A), which was lowered during the NF to 179 (Fig. 16.16B) and was still lower even after the NF stopped, nZ score 169 (Fig. 16.16C). Our interpretation, for the results obtained, is that the lower the nZ score, the
Victoria L. Ibric, M.D., Ph.D. BCIAC and Liviu G. Dragomirescu, Ph.D.

0–25 mild; 26–50 moderate; 51–75 severe; 76 + very severe

FIGURE 16.15A  Case 10: Stress test evaluations over the years of NF training.

Max = 360; Min = 0

FIGURE 16.15B  Case 10: SCL-90R evaluations over the years of NF training.

Note: CES-D max = 60; over 16 + depression

FIGURE 16.15C  Case 10: Depression CES-D evaluations over the years of NF training.
FIGURE 16.16A Case 10: QEEG—Connectivity map in eyes-closed condition (nZ score 335, shows a high level of disconnection).

better the brain should function. Therefore the NF, by modifying the connectivity parameters, proves to be an effective training tool for patients post-TBI and suffering with chronic pain.
FIGURE 16.16B  Case 10: QEEG—Connectivity map in eyes-closed condition during the ROSHI NF training (nZ score 179 shows a reduction of the disconnection).

FIGURE 16.16C  Case 10: QEEG—Connectivity map in eyes-closed condition after ROSHI NF training (nZ score 169 shows a continued correction of the connectivity).
FIGURE 16.17A  Case 10: HEG during the NF on ROSHI NF light enhanced.

FIGURE 16.17B  Case 10: HEG during the NF on ROSHI, light and electromagnetic enhanced NF. The spike in HEG at the end of the training corresponded to patient's expression of happiness, due to the complete resolution of pain for the first time.

HEG (Hemoencephalography) was done during the NF training on ROSHI instrument. Figs. 16.17A and 16.17B shows that the perfusion of the frontal area increased and corresponded to lowering of the pain perception.
VIII. STATISTICAL ANALYSIS OF THE NF EFFICACY IN PAIN SYNDROMES

The analysis of the responses to the NF training in our chronic pain patient group offered many interesting results, which are reported here. Firstly, we evaluated the relationship between the number of sessions and the outcome; secondly, we verified the efficacy of NF in cases treated for more than 19 sessions; thirdly, we checked if there is a correlation between the age of our patients to the number of sessions needed; and fourthly, we evaluated the influence of gender on the number of NF sessions needed to correct their pain perception.

Table 16.5 presents the total number of patients (147) used in the study, from which 52 were males and 95 females. The outcomes of the NF training in relation to the number of NF sessions are presented. It can be seen that there is a direct correlation between the number of sessions and the positive outcome. When the NF training was done only for up to 19 sessions, the rate of success is very reduced (only three cases ameliorated), while in the case of the patients who completed more than 19 sessions of NF the success rate was evident (68 out of 74 cases with clinical significant improvement (CSI), or 92%, and up to 95% total success if we consider all CSI cases plus two ameliorated).

We chose the value 20 (first number >19) as a minimal limit for the number of NF sessions (CSM), following the literature (Marzano et al., 2001). We also found and reported previously (Ibric and Dragomirescu, 2005) from our patient group, and in particular from Cases 8 and 9 that a minimal number of 20 sessions was necessary to obtain CSI, or to attain 70–80% of the learning curve as Marzano described in his study on students’ learning skills.

A. NF training efficacy relative to number of sessions (NS)

We compared NS for the three types of results obtained: Clinical significant improvement = CSI, Ameliorated = A, and those without any positive results or zero results = 0 results. The results are presented in Tables 16.5 and 16.6.

TABLE 16.5

<table>
<thead>
<tr>
<th>No. cases</th>
<th>Positive results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients with 1 NF session after evaluation</td>
<td>15</td>
</tr>
<tr>
<td>Number of patients who completed 2–10 NF sessions</td>
<td>33</td>
</tr>
<tr>
<td>Number of patients who completed 11–19 NF sessions</td>
<td>25</td>
</tr>
<tr>
<td>Number of patients who completed &gt;19 NF sessions</td>
<td>74</td>
</tr>
</tbody>
</table>

Success rate CSI* = 68/74 = 92%
Success rate of POSITIVE RESULTS = 70/74 = 95%

*CSI is abbreviation for “Clinical Significant Improvement”
TABLE 16.6  The minimum (Min), maximum (Max) and the three quartiles [lower (Q₁), median (Me) and upper (Q₃)] of NS (# Sessions) for the “0 or no results”, (A) ameliorated (A) and clinical significant improvement (CSI) patients

<table>
<thead>
<tr>
<th>Efficacy Results</th>
<th>Number of patients</th>
<th>Minimum (Min)</th>
<th>Lower Quartile, Q₁ (25%)</th>
<th>Median (Me) (50%)</th>
<th>Upper Quartile, Q₃ (75%)</th>
<th>Maximum (Max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 results</td>
<td>73</td>
<td>1</td>
<td>2</td>
<td>6</td>
<td>13</td>
<td>32</td>
</tr>
<tr>
<td>A</td>
<td>6</td>
<td>8</td>
<td>14</td>
<td>18.5</td>
<td>25</td>
<td>34</td>
</tr>
<tr>
<td>CSI</td>
<td>68</td>
<td>20</td>
<td>27</td>
<td>35.5</td>
<td>64.5</td>
<td>250</td>
</tr>
</tbody>
</table>

The differences (Kruskal-Wallis test) between medians 6.0, 18.5, 35.5 are significant (p < 0.001).

Using the MINITAB program for plotting data presented in Table 16.6, we generated Fig. 16.18 showing that there are statistical outliers in the CSI and the “0 results” categories. These values are outside the Q₃ + 1.5(Q₃ - Q₁) quartile interval in their respective subsets.

TABLE 16.7 Case control study of the Efficacy of NF training for “more than 19 sessions”

<table>
<thead>
<tr>
<th>NF exposure</th>
<th>Cases—with illness (0 effect)</th>
<th>Controls—without illness (Positive effect)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposed to enough NF training (More than 19 sessions)</td>
<td>4</td>
<td>70</td>
</tr>
<tr>
<td>Not exposed to enough NF training (1–19 NF sessions)</td>
<td>69</td>
<td>4</td>
</tr>
</tbody>
</table>

From Table 16.7, a case-control study was considered and (Odd Ratio OR) = 0 was obtained. Because one or more expected values are less than 5 we used the exact confidence limits according to the Fisher Exact test, and they are: exact lower 95% confidence limit = 0, and exact upper 95% confidence limit = 0.02. Because OR < 1 and
the confidence interval (0, 0.02) does not include 1, our data indicate that treatment with more than 19 sessions is a protective factor for chronic pain. This is consistent with other previous reports in the NF literature (Lubar, 1985, Steinberg and Othmer, 2004).

B. Relationship between the patient’s age and the number of sessions (NS) of NF

Figure 16.19 shows a scatter diagram of age of our pain patient group versus log (NS). This was done for exploratory purposes only. The cloud of the data points is distributed, approximately, parallel to the horizontal axis. This result shows that there is no correlation between the age of our pain patient group and the number of NF sessions needed, expressed as log (NS).

C. Relationship between the patient’s gender and the response to the NS of NF

Table 16.8 presents the quartiles of NS for both sexes from the “CSI” group of patients. According to the Mann–Whitney test, the two median values, 32 and 46.5, for females vs. males respectively, are not significantly different (p < 0.128). This p value is very close to 0.1, which may be significant from some statisticians’
444 Neurofeedback in pain management

![Log (NS) vs Age Scatter Plot](image)

FIGURE 16.19 The scatter diagram shows statistical independence between log (NS) and the age of the pain patient group.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Number of patients</th>
<th>Minimum (Min)</th>
<th>Q1 (25%)</th>
<th>Median (Me)</th>
<th>Q3 (75%)</th>
<th>Maximum (Max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>40</td>
<td>20</td>
<td>24.5</td>
<td>32</td>
<td>60</td>
<td>250</td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>20</td>
<td>31.5</td>
<td>46.5</td>
<td>73.5</td>
<td>164</td>
</tr>
</tbody>
</table>

The differences (based on Kruskal-Wallis test for two groups equivalent to Mann-Whitney test or Wilcoxon on Two-Sample Test) between 32 and 46.5 are not significant ($H = 2.314, DF = 1, p = 0.128$).

IX. DISCUSSION

Why is neurofeedback useful in the modulation of pain perception? If we are to follow the localization of the central parameters of pain as mentioned above, we can design protocols that can rather directly address these parameters. For example, for pain intensity, by considering the SI area or the main sensory area over the central SM, and following the homunculus map, we can address the pain intensity for the particular area that needs attention (Tolle et al., 1999). If the emotional issues
are associated with the pain they are usually feeding the pain; then the area to address with NF training will be the anterior cingulate gyrus (Rainville, 1997).

Insofar as psychological factors are concerned, the cognitive processes of the patient are important to investigate in order to understand pain and disability. Coping mechanisms with chronic pain differ from patient to patient, as mentioned by Hanson and Gerber (1990) and Haythornthwaite et al., 1998. Psychological and social factors impacting on pain may change the level of arousal, and modify body chemistry. Thought effects on sympathetic arousal and muscle tension could be measured with peripheral BF. Thoughts apparently can change the body biochemistry, producing endogenous analgesia. For example, Bandura (1987) examined and demonstrated the direct effect of cognitive control of pain on central opioid activity. And Birbaumer et al. (1995) and Maihöfner et al. (2003) showed that the brain changes its functional organization at the level of the somato-sensory cortex in chronic pain patients. Thus, it should not be especially surprising that NF, with its emphasis on volitional control, has been found useful in pain modulation as demonstrated by Ibric (1996) and Donaldson et al. (1998, 2004).

**X. CONCLUSIONS**

Pain or DOLOR is one of the five symptoms described in any kind of inflammation process. That is why so many anti-inflammatory drugs are administered at the
Neurofeedback in pain management

The first sign of persistent and debilitating pain. However, when pain persists for longer than 6 months, and the symptoms are labeled as chronic pain, other medications such as morphine or its derivatives tend to be prescribed. A major problem is that often the pain is accompanied by other symptoms such as depression, anxiety, sleep disturbances, and decision-making abnormalities (Apkarian et al., 2004a). As stated by Baliki et al. (2008, p. 1398), “chronic pain hurts the brain, disrupting the default-mode network dynamics (DMN).” Their study, using fMRI, showed that the brain of a chronic pain patient is not simply a healthy brain processing pain information, but rather is altered by the persistent pain in a manner reminiscent of other neurological conditions associated with cognitive impairments (Baliki et al. 2008, p. 1402).

In their study, these co-morbidities are found accompanying the pain syndromes, possibly having been produced by disrupted DMN. During a task, there are areas of activation while other areas are deactivated. The research of Baliki et al. (2008) showed that chronic pain patients presented much smaller areas of brain deactivation in several key areas, suggesting widespread disruptions of the DMN, thus indicating that this phenomenon may underlie the cognitive and behavioral impairments.

During the last half-century, NF has become a state-of-the-art training modality. With each passing year, more sophisticated computer programs have been developed, and improved NF protocols designed based on comprehensive standardized evaluations. Adding QEEG evaluations has played an important role in enhancing NF by guiding training protocol designs. Training strategies evolved as more accurate electrode positioning, and more brainwave enhancement/suppression possibilities became available. Specifically with regard to NF for pain syndromes, training is being dictated by the characteristics of pain and the multitude of overlapping diagnoses.

There are many examples of clinical and research findings of special significance for the understanding and treatment of pain. For example, NF training applied over the frontal cortex has been found to be followed by a change in pain affect as reported by patients with acute or chronic pain syndromes. And this effect correlates with the localization of the unpleasantness of the pain as described by Rainville et al. (1997). Apkarian (2004b), using functional MRI techniques, revealed that chronic back pain appears in a different part of the brain (prefrontal cortex) than the discomfort of burning a finger, for example. The acute sensory pain of the burned finger appears in the sensory part of the thalamus with projection into the cortical homunculus. That may explain why NF may be successful when applied to the areas of the brain corresponding to the peripheral perception of an acute pain. However, chronic back pain shows up in the prefrontal cortex. By contrast, with the acute pain representation, the chronic pain leaves an imprint in the prefrontal cortex; thus, it seems that the longer the suffering of pain, the more activity in the prefrontal area.

QEEG evaluations have provided similar results to fMRIs, and have guided NF practitioners to train the prefrontal cortex in patients who have suffered from
chronic pain for a very long time. This “cumulative memory” may be modulated, or erased through NF.

However, NF training in patients with acute or chronic pain syndromes, applied over the contralateral central cortex area corresponding to the peripheral painful regions, can bring significant changes in the perception of pain, expressed as pain threshold and pain tolerance. There are no significant differences between the number of NF sessions and the age of the patient population analyzed in this study. There are no significant differences between the male and female responses to NF training regarding the change in pain perception. Thus, we can conclude that the ages of the pain patient population do not influence the response in modulating the pain perception. The number of NF sessions does influence the outcome. Twenty sessions were “necessary and almost sufficient” to produce some positive effects on the pain perception and affect of our patients.

Due to the relatively small number of cases analyzed in this study, it was hard to determine how the different diagnostic cases responded to NF training. Also, we could not conclude that gender influenced the outcome like others observed (Derbyshire, 2008), since our sample population was too small. As a final conclusion, however, we can say that NF training, if carried out past 19 sessions, can modify pain perception and pain affect, perhaps permanently. Correction of sleep and emotional dysfunctions has also been noted.

NF training directly addresses cortical areas corresponding to pain perception, memory, and affect. NF, based on neuromodulation, achieves its positive and sustained results largely through operant conditioning. However, it also can impinge favorably upon the pain experience indirectly through arousal regulation, as well as through enhanced central nervous system and autonomic nervous system stability, and improved homeostatic control. Commensurate with the neuroplasticity of the nervous system, the localization of pain perception at the central level seems capable of being changed by events at the periphery (see work on the re-mapping of the brain sensory area in amputees [Ramachandran and Rogers-Ramachandran, 2000]). The corticalization of pain perception described by Birbaumer et al. (1995), Apkarian et al. (2004 a and b), and Baliki et al. (2008) also helps explain the basis of the pain modulation obtained through NF techniques.

DeCharms et al. (2005), using fMRI, reported that the anterior cingulate is an area involved in pain severity, and found the fMRI imaging to be responsive to feedback control. Since then greater attention has been given to the possibility of using fMRI in pain management. In 2007, the Institute of Medicine’s report states that functional imaging is under study and possibly can be used not only to detect brain areas affected by chronic pain, but also act as a therapeutic intervention. However, fMRI used in therapy may not be cost-effective. Unfortunately, despite many clinical and research findings regarding EEG correlates of pain, and a great many cases of successful treatment of pain through NF, nothing was mentioned in this report about the promise of NF.

The use of QEEG as the basis for identifying the location and type of brain disturbances, or the efficacy of using neurofeedback for modifying or eliminating
pain, should also be considered. The research and clinical cases cited in this chapter attest to the fact that through the application of neurofeedback it is possible for pain patients to extend the brain’s natural self-healing powers, and perhaps re-establish a “default mode network” needed for optimal brain function. These non-invasive techniques, QEEG and NF, must continue to be integrated in the medical care for those who suffer with chronic pain.

REFERENCES


Victoria L. Ibric, M.D., Ph.D. BCIAC and Liviu G. Dragomirescu, Ph.D.


