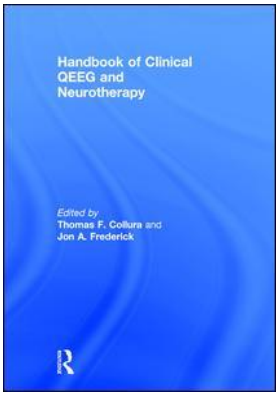

Handbook of Clinical QEEG and Neurotherapy

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Investigating the Neuroplasticity of Chronic Pain Utilizing Biofeedback Procedures

Publication details
https://www.routledgehandbooks.com/doi/10.4324/9781315754093.ch5
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Published online on: 17 Nov 2016

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INVESTIGATING THE NEUROPLASTICITY OF CHRONIC PAIN UTILIZING BIOFEEDBACK PROCEDURES

Stuart Donaldson, Mary Donaldson and Doneen Moran

Abstract

Norman Doidge in his book *The Brain That Changes Itself* (2007) describes chronic pain as a negative consequence of neuroplasticity or neuroplasticity gone wrong. Using their clinical experiences, the authors explore the issues involving chronic pain and its relevance to neuroplasticity. Changes in the peripheral and central nervous systems are examined through the use of surface electromyography (SEMG), quantitative electroencephalography (QEEG) and stress profiling techniques. The use of these techniques is combined with relevant research literature to produce a working model of the complex phenomena known as neuroplasticity. A case study concludes the presentation.

Neuroplasticity, also known as brain plasticity, is an umbrella term that encompasses both synaptic plasticity and non-synaptic plasticity—it refers to changes in neural pathways and synapses which are due to changes in behavior, environment and neural processes, as well as changes resulting from bodily injury (Pascual-Leone et al., 2011). Neuroplastic changes may occur on a number of different physiological levels from cellular to cortical remapping after injury and is also involved in normal learning processes, performance enhancement and growth (maturation).

Clinically the neuroplasticity model provides a theoretical model in which to explore and understand the neurological changes that occur with chronic pain.

Chronic pain is thought to represent a maladaptation of the nervous system to repeated stimulation which in time produces a change in the nervous system itself. Examination of these changes requires an extensive knowledge of the three branches of the nervous system: (a) the central nervous system, (b) the peripheral nervous system and (c) the autonomic nervous system and the disturbances in one system that can cause alterations in the other systems.

In most cases chronic pain initially involves repeated stimulation at a peripheral site which involves noxious stimuli and inflammation. This repeated stimulation over time elicits a neuroplastic response at the cortical level leading to changes in somatotopic organization including central sensitization (Seifert & Maihöfner, 2011). Norman Doidge introduced the concept of neuroplasticity in its numerous forms in his book *The Brain That Changes Itself* (Doidge, 2007). Outlining the numerous ways changes in the central nervous can enhance the quality of life, he also points out that these changes can be maladaptive, as seen in chronic pain.

Doidge postulates that when a part of a body is lost the brain hungers for stimulation, releasing little sprouts to grow and develop looking for nerves or sites in the brain that are similar in type to those lost. Often this occurs but occasionally cross wiring errors happen leading to a mixing of nerve impulses. Also the brain appears to be constantly changing (Doidge, 2007) as repeated mapping of the brain shows that it changes its contours dependent upon the stimulation. Doidge suggests that these factors play a role in the development of the neuroplastic aspects of chronic pain. In a few rare cases repeated stimulation is not evident, being replaced by singular overwhelming trauma such as a motor vehicle accident.

Numerous researchers have demonstrated these central nervous system changes. Maihöfner, Handwerker, Neundorfer and Birklein (2003) demonstrated that individuals experiencing complex regional pain syndrome (in the hand) demonstrate a diminished cortical somatotopic representation of the hand contralaterally as well as a decreased spacing between the representation of the hand and the mouth in the somatosensory cortex. Reduction of the volume of gray matter in the prefrontal cortex and thalamus was reported by Apkarian et al. (2004) in chronic pain. Similar results have been reported for phantom limb pain (Karl, Birbaumer, Lutzenberger, Cohen & Flor, 2001), chronic low back pain (Flor, Braun, Elbert & Birbaumer, 1997) and carpal tunnel syndrome (Napadow et al., 2006).

The peripheral nervous system also appears to be impacted or involved with neuroplasticity on two levels. Evidence from numerous authors suggests that the peripheral nerves also respond to repeated stimulation demonstrating that dendrites grow in diameter and length while their axons spread the field of innervation. For example, recent research (Navarro, Vivo & Valero-Cabre, 2007) suggests that peripheral nerve injury induces a cascade of events at the systemic, cellular and molecular levels progressing throughout the spinal cord to the brainstem relay nuclei, thalamus and cortex.

This brief overview of the literature is not intended to be comprehensive but to give the reader an idea of the theoretical basis for the working model and rationale for the assessment of chronic pain.

Clients

During the year 2013 there were 251 new clients seen ranging from children with Attention Deficit Disorder to adults with severe chronic pain of various etiologies. Of the clients seen 133 were of the chronic pain variety with the majority suffering from fibromyalgia or myofascial pain syndromes. These chronic pain sufferers were on the average 47.8 years old, had been in pain for 2–30 years (median 5 years) and were predominantly female and right-handed.

The clinic is private in nature with the client responsible for their own expenses. This produces a skewed distribution of type of client, usually upper middle to upper class with a higher education. Participation in the clinic programs was voluntary in nature with a full disclosure of treatment effects and risks occurring before participation.

Evaluation

The clinical investigation is directed towards understanding all the factors which contribute to the presence and maintenance of the chronic pain. The question to be addressed is: how does neuroplasticity develop and how is it then maintained in chronic pain? To address this question a five-part evaluation is conducted. The five parts include: (a) presenting problem, (b) history as relevant to the presenting problem, (c) central nervous system contributions, (d) peripheral nervous system contributions and (e) autonomic nervous system contributions.

Presenting Problem(s)

The chronic-pain patients seen are usually suffering from: (a) chronic muscle pain (i.e. headaches, low back pain and muscle pain throughout the body) with (b) cognitive complications or issues (i.e.

problems with concentration, loss of focus, poor memory) and/or (c) emotional issues (anxiety or depression). During this phase of the interview these three primary tracks are being explored.

Approximately 90% of these individuals report that they have been diagnosed by their health care provider as having fibromyalgia. The emphasis on assessing and treating chronic muscle pain did not take off until the introduction of the concept of fibromyalgia in the late 1990s. The release of the Task Force Report by Wolfe et al. (1990) started the trend to diagnose most chronic muscle pain as fibromyalgia.

Most patients start the interview stating, "I want to get rid of my pain and get back to the way I was before the start of the pain." Time is spent discussing their pain, describing it in detail and exploring how it started and when it started. This emphasis on the pain at the start serves to validate the individual as most people have been told it's in their head and there is no objective data to support their complaints. The second thing that happens is somebody is listening in a non-judgmental way.

As the interview proceeds the ideas of trigger points and tender points are introduced as the cause of their pain, plus the concepts of myofascial pain syndromes and of fibromyalgia are introduced (if needed). The textbooks of *Myofascial Pain* (Travell & Simons, 1983) are introduced, with individuals shown pictures of what is expected to be the source of their pain (i.e. trigger points) and the referred pain patterns. This is very validating to individuals who feel neglected by the health care system, with more than a few tears seen at this point.

It is a big help to have a diagram of a human body with views of both front and back. Most people will circle specific areas indicating back pain, or headaches or whatever. Some will circle the entire body, both front and back, while others will circle several specific areas of pain. This represents the first clues for diagnosis. The person who circles the whole body probably has some central summation process occurring, probably involving allodynia and hyperalgesia consistent with fibromyalgia. The person with a specific site or two of pain probably has a myofascial pain syndrome involving the peripheral nervous system. The individual with multiple but specific sites presents a greater challenge as there may be central, peripheral and autonomic issues which need further investigation.

Time is spent exploring when and where the pain developed. Answers to this question are varied, ranging from on such and such a date when the motor vehicle accident happened, to that of being not sure as there was more of an insidious onset. The fixed date answer represents more of a challenge to determine the time of onset because the fixed date of the accident represents the date of the onset of acute but not chronic pain. This is important to remember because the development of trigger points (myofascial pain syndrome) takes about six weeks to occur after the trauma and the length of time to show marked neuroplastic changes is not well known.

The focus of the interview then changes to that of establishing how they are doing cognitively. Issues concerning memory, focus, concentration and the ability to multi-task are discussed. Decreased abilities in these areas are red flags for a need for further investigation of central processes and how the brain is functioning. Most people are confused by the noted cognitive decline, thinking they may be developing Alzheimer's disease. If the onset is associated with a trauma (i.e. motor vehicle accident) it is important to detail if there was a loss of consciousness or if they were dazed or violently thrown about as this points to a possible closed head injury or whiplash involving the neck.

While all this is occurring a mental status examination is being conducted. It is important to know the psychiatric status of the person as individuals with significant emotional issues are screened out at this time. The clinic's focus is on rehabilitation of chronic pain, not the cure of psychiatric disorders. People eliminated from the program at this point are usually (a) those delusional about putting electrical signals into their brain, (b) those grossly angry with control issues (usually directed at insurance companies) and (c) individuals on certain types of medications that interfere with the biofeedback signals.

Conversations after this point are wide ranging and varied, discussing how the pain has impacted their lives in any area the patient wants to discuss and includes their interests and hobbies. All the time the question in the background is: what factors are causing and or maintaining the pain?

History

During the wide ranging conversation a review of the individual's medical, social and employment history will be conducted. Details of these parts of people's lives are important to neuroplasticity and how it has developed.

The medical background is reviewed to see if a pattern of illness existed before the development of the chronic pain. The thinking is, the more the individual was ill before the onset of the pain, the greater the likelihood that the nervous system is wired to be responsive to the new pain (directly reflecting the concepts of allodynia and hyperalgesia). Also at the same time factors pertaining to secondary gain can be explored. Secondary gain is not considered as important as the number of times the individual got sick or suffered some severe illness except in extreme cases such as Munchhausen by Proxy.

Illness such as severe viral infections can cause an increase in Theta activity during the course of the illness (Westmoreland, 1993). Patients will report that after the illness was over and they had fully recovered, cognitively they felt different with some type of diminished cognitive capacity. Also their reaction to immunizations is explored as a few chronic pain sufferers report an extreme reaction to the shots. Another issue examined is the number of general anesthetics they received.

Social and employment issues are examined, exploring what type of social network they have and if the network is meeting their needs. Socially isolated individuals seem to take longer to recover. The thinking is that having others around, if nothing else, serves as a form of distraction from the pain. Getting people back to work is extremely important, for not only do they have the social network that work provides, but being active reduces the impact of depression.

A key predictor as to whether or not the individual will recover from a trauma type onset of the chronic pain is the level of anger or rage the person feels. Often they have feelings of being victimized, of being in the wrong place at the wrong time and yet having to prove they were injured and they are not faking it. They want their just reward and the insurance company is going to pay. These cases usually involve a lawyer as well, introducing a secondary factor as to how much the lawyer reinforces the complainant.

On the basis of the information as gathered above the individual is given the option of being evaluated or they can drop out. A comprehensive evaluation involves: (a) a quantitative electroencephalograph (QEEG), (b) a mini cognitive functioning test, (c) a trigger point and dolorimeter examination, (d) a surface electromyographic (SEMG) evaluation and (e) a psychophysiological stress test. Points (a) and (b) are designed to assess central nervous system functioning, points (c) and (d) are designed to assess peripheral nervous system functioning and point (e) is designed to assess autonomic functioning.

Central Nervous System Contributions

A routine QEEG is performed on any person with suspected central nervous system (CNS) contributions to the chronic pain. Indications for testing include but are not limited to: (a) cognitive issues such as decreased memory, needing to reread articles several times, unable to recall words when appropriate, poor focus and concentration, and decreased ability to multi-task; (b) changes in emotional status such as increased irritability, depression and anxiety; and (c) poor results from numerous physical therapies. (Patients usually report they have seen 3 to 5 therapists, usually reporting an initial decrease in pain of 30% with any treatment, then plateauing and not responding to further treatments.)

The QEEG is performed following standard protocols and utilizes the Neuroguide database as developed by Thatcher (2012) for data interpretation. The reader is referred to Thatcher (2012) for details of the procedures and database.

The raw data from the QEEG is inspected, examining for epileptiform phenomena. With any evidence of this in the tracings the patient is immediately referred back to the physician for investigation by a neurologist and a recommendation that treatment can continue.

If the raw tracings are acceptable the entire QEEG is reviewed for deviance from normal. There is no one pattern that is associated with chronic pain so all aspects of the data are examined. Deviances are most commonly found in absolute power, on the Mild Traumatic Brain Discriminate Index (MTBI), coherence, to a lesser extent in phase lag and in Brodmann areas 21 and 23 on the LORETA.

Absolute power most often shows increased Beta activity (18–25 Hz) frontally in the fibromyalgia population (Donaldson, Donaldson, Mueller & Sella, 2003). Consistent with this is decreased Delta throughout the cortex. In the myofascial population that is resistant to change, increased Beta activity of 25 Hz is seen specifically at Cz with physical improvement not noted until this activity is decreased. In general, deviant activity in absolute power needs to be altered (normalized) for both fibromyalgia and myofascial pain before physical therapies start to work and the pain changes.

Patterns of coherence, phase lag and MTBI results are more difficult to relate to chronic pain as a large percentage of our sample is post motor vehicle accident. Clinical observations by Donaldson suggests that changes in the right hemisphere in coherence and phase lag (to a lesser extent) are associated with a decrease in chronic pain. Specific sites affected include F8, F4, C4, P4, T4 and T6. At this time it is not known if these results are causal or correlational.

Data from the MTBI is utilized in a couple of different ways. First it is used to confirm that there is a probability of membership in the closed head injury population. This is particularly important for those individuals thinking they have Alzheimer's disease or think they are imagining their cognitive problems. The severity index gives them an understanding as to the nature and extent of the injury. The actual results are not used to specifically direct treatment. It is important these results are communicated not only with the patient but with a significant other or spouse as recent research shows that within 5 years 17–48% (Arango-Lasprilla et al., 2008; Kreutzer, Marwitz, Hsu, Williams & Riddick, 2007) of individuals sustaining a closed head injury in an MVA are divorced. The MTBI results are an attempt to help the other understand what the patient is going through, is not faking or malingering and suggest resources to help them cope. Results of the cognitive functioning testing are introduced at this point to reinforce and to connect the noted cognitive dysfunctions to the findings of the QEEG.

Data produced by LORETA shows numerous areas can be affected by trauma, particularly Brodmann areas 21 (lateral temporal cortex) and 23 (posterior cingulate gyrus). This is not surprising considering their anatomical locations. However, what is not known is how trauma to these areas affects chronic pain.

The neuroplasticity model predicts that the brain will change in response to changes in stimulation, whether external to itself or to changes that occur within the brain's structure itself. Once these changes occur, as seen in chronic pain patients, it is believed that the changes have to be altered to break up the reinforcement patterns that exist to cause and maintain the pain. The QEEG serves this purpose well by showing exactly which areas and pathways have been altered.

Peripheral Nervous System Contributions

The acquisition of motor skills (i.e. learning to walk) is a demonstration of the effect of the peripheral nervous system upon the central nervous system. The continued feedback of the movement and its outcome shapes neural pathways that are retained for the rest of one's life. While clumsy at the start, as repetitions occur the movement becomes more refined and efficient. Leonard, Moritani,

Hirschfeld, and Forssberg (1990) studied the H reflex in children as they grew up from birth to age five, comparing children suffering from cerebral palsy to those with normal development milestones. He found that by age five the H reflex had been suppressed in the normal sample while still present in the cerebral palsy sample.

Grouped muscle pattern activity in children was studied by Janda and Stara (1965). They demonstrated that predictable patterns of coactivation existed in younger children throughout the body even in muscles far removed from the site of study. As the children matured this activity disappeared and was absent in normal adults. Under psychophysiological stress these patterns reappeared. The authors went on to demonstrate that these reappearing patterns can then be inhibited and disappear with biofeedback training. All of these results were reproduced by Gate (1967) replicating the effect of maturation upon motor control. Thus skill acquisition appears to be a learned behavior in which the inhibition of excessive or inappropriate muscle activity leads to the development and improvement of motor control and the desired movements (Donaldson, Nelson & Schulz, 1998).

As indicated, the neuroplastic model suggests that changes in the central nervous system are associated with repeated stimulation from the peripheral nervous system. This may be both advantageous and harmful. Travell and Simons (1983) indicate that pain arising from muscle trauma affecting the neural activity at the dorsal horn for that vertebral body is 90 times greater and lasts 15 msec. longer when compared to pain from skin abrasions. Thus pain produced from muscular dysfunction(s) can potentially be a major factor in causing neuroplastic changes.

As a tool for understanding the effects of neuroplasticity on the peripheral nervous system, surface electromyography (SEMG) procedures offer a vast wealth of information. SEMG techniques indicate (in no particular order) information about (a) muscle tonicity (hyper and hypo) (Sella, 2000), (b) force generation (Basmajian & DeLuca, 1985), (c) amount of force required to generate movement (Basmajian & DeLuca, 1985), (d) interactions with other muscles (Basmajian & DeLuca, 1985) and (e) timing of muscle interactions (Bolek, 2003) and their kinesiological properties.

Disruptions in the inhibitory activity produces muscle dysfunctions which have been associated with numerous types of chronic pain including: (a) carpal tunnel (Skubick, Clasby, Donaldson & Marshall, 1993), (b) low back pain (Sihvonen, Partanen, Hanninen & Soimaakallio (1991), (c) headaches (Donaldson, Rozell, Moran & Harlow, 2012), (d) fibromyalgia (Donaldson, Snelling, MacInnis, Sella & Mueller, 2002), (e) trigger points (Donaldson, Skubick, Clasby & Cram, 1994) and (f) cerebral palsy (Bolek, 2003). Presently, no other tool can offer such information which is so important in understanding the development of neuroplastic changes associated with chronic pain.

Each one of the conditions listed above can result in altered neurological afferent sent back to the brain which in turn alters its systems. The continued presence of the disturbed signal leads to the development of trigger points and tender points, causing and maintaining the chronic pain and altering the signals in the brain, producing neuroplastic changes.

SEMG assessment follows standardized protocols (Donaldson, 2003) in which electrodes are placed over the targeted muscles. Individuals are asked to sit or stand still and then to perform movements primary for that muscle. Data is captured at a sampling rate of 2048 samples per second for 10 muscles simultaneously as the person performs various movements primary for that muscle. Data is captured in Root Mean Square (RMS) format and analyzed for each muscle for: (a) level of activity at rest, (b) level of activity at rest as compared to the homologous partner, (c) maximum activity during unresisted movement, (d) maximum activity during unresisted movement as compared to its homologous partner, (e) activity as part of the myotatic unit and (f) as part of the kinesiological functioning of the body.

The SEMG signal provides the information that is going back to the brain. Over time the crossed extensor pathway becomes altered (increased and decreased afferent), affecting the learned inhibitory pattern leading to a change in the systems and neuroplastic based dysfunctions in both the central and peripheral systems.

Psychophysiological Stress Profiling

The activity of the autonomic nervous system (ANS) is studied through the use of psychophysiological stress profiling (PSP). The ANS functions automatically, controlling blood pressure, heart rate and rate of breathing, amongst other systems. While generally considered as an unconscious function, its activity can be brought under voluntary control through various conditioning techniques.

The main concern in studying this system is to understand how its activity may be helping to maintain and enhance the chronic pain. Generally, increased autonomic activity will enhance the activity of the peripheral system in several different ways.

Numerous physiological systems are monitored simultaneously, examining for significant deviances from established norms. The physiological systems monitored include (a) heart rate, (b) heart rate variability, (c) respiration, (d) peripheral hand temperature, (e) galvanic skin response, (f) muscle tension in the face and (g) muscle tension in the shoulders (upper trapezius). In addition, brain wave activity is monitored.

The test follows a standardized protocol. The individual is hooked up to a computer. Then a baseline of activity is recorded for 2 minutes with eyes open, followed by 2 minutes of eyes closed. Then a computerized task is performed, followed by a rest period. This procedure is repeated for 8 tasks and recovery periods. Data is examined for deviations from normal, reaction to the stressors and the ability to recover.

Each deviant measure is thought to play a role in the development of chronic pain or neuroplasticity. It is believed that the impact is significant on both the central and peripheral systems exacerbating dysfunctions or altered neural pathways. For example, increased heart rate, increased respiration and decreased peripheral hand temperature are all thought to exacerbate muscle pain and are associated with anxiety. Increased rate of breathing is thought to increase frontal Alpha. A heightened galvanic skin response prepares the body for a flight or fight response, creating a cascade of reactions peripherally and centrally. This factor is considered a major factor in numerous diseases associated with stress.

In conclusion, the ANS has a major impact upon all the systems and needs to be included in the evaluation of any chronic pain situation.

Headaches: An Example of Neuroplasticity

An example of how all these ideas culminate in creating chronic pain now follows. The individual is a middle-aged high performance executive who has suffered from chronic headaches for over 30 years. The headaches occurred daily, varying in location but more often in the temples. While diagnosed as migraines she did not respond well to migraine medications, often becoming nauseated with them, but persisted in taking them. Her history is that of abuse coupled with numerous falls off of horses, including one in which she was knocked unconscious. As it was thought that her sinuses were causing the problem, surgery to correct this problem was performed without success.

A comprehensive evaluation showed the following results:

QEEG:
absolute power—decreased (2SDs) Alpha activity throughout,
relative power—increased (2SDs) posterior Theta and central High Beta,
phase lag issues involving right hemisphere,
MTBI positive at 97.5% level with severity index of 2.33 principally affecting T4.

SEMG:
whiplash involving left sternomastoids, right cervical paraspinals, left scalenes, right lower trapezius.

PSP:
low heart rate variability,
rapid respiration,
decreased peripheral hand temperature,
facial muscle tension.

(Please note the above results represent only information relevant to this discussion. Please see Donaldson et al. (2012) for the complete details of this evaluation)

A routine course of treatment lasting six weeks was initiated. This included:

a) EEG neurotherapy
   1. utilizing LENS techniques to normalize the activity at T4, and
   2. routine EEG neurotherapy to down train the excessive High Beta activity (> 20 Hz) at Cz.
b) SEMG neuromuscular retraining techniques (Donaldson et al., 1998) were utilized to rebalance the activity of the sternomastoids, cervical paraspinals, scalenes and lower trapezius.
c) Massage therapy was concurrently utilized to deactivate the associated trigger points.
d) Heart rate variability training was also employed.

Headaches had diminished in six weeks and she was withdrawn from her migraine medications at that time.

One year follow-up showed no presence of excessive High Beta activity at Cz and a normalization of the Z scores at T4. The neck muscles remained balanced and heart rate variability training was utilized as needed. Presently, she is almost completely symptom free except when the weather changes and chinooks occur. Also improved cognitive functioning is reported.

Neuroplasticity: A Factor in the Development of Motor Control

As previously stated, neuroplasticity is an umbrella term that encompasses both synaptic plasticity and non-synaptic plasticity—it refers to changes in neural pathways and synapses which are due to changes in behavior, environment and neural processes, as well as changes resulting from bodily injury (Pascual-Leone et al., 2011).

The acquisition of motor skills (i.e. learning to walk) is a demonstration of the effect of the peripheral nervous system upon the central nervous system supporting the concept of neuroplasticity. The continued feedback of the movement and its outcome shape neural pathways that are retained for the rest of one's life.

However, various authors (Gate, 1967; Janda & Stara, 1965; Leonard et al., 1990) suggest that skill acquisition occurs when neuromuscular reflexes and patterns are inhibited.

It would appear that these reflexes and patterns are only inhibited and lie dormant as they reappear during periods of stress and disease (Janda & Stara, 1965).

The question becomes: what part of the chronic pain process does neuroplasticity play?

a) If neuroplasticity reflects changes in the system, in the above example, what part does the abnormal power at T4 play? Does it reflect an injury to the brain producing fewer cells to control that part of the brain or producing decreased ability to compensate or inhibit, or is it an incidental finding?
b) Does the appearance of increased High Beta activity at Cz reflect a loss of inhibitory control or increased compensatory activity? Janda and Stara's 1965 work would suggest the former.
c) What does the appearance of increased activity in the Theta frequencies in the posterior part of the brain indicate? Any of the above reasoning could be applied to this finding.

d) The correction of the muscle imbalances in the neck would indicate that correct inhibitory processes in the peripheral control mechanisms had been re-established. These changes are consistent with the ideas of neuroplasticity affecting the peripheral mechanisms, but can the muscle changes be an explanation for the noted changes in brain wave activity?
e) The noted changes in autonomic activity are also evident. Does the improvement in muscle control and changes in brain wave activity lead to a decreased autonomic activity, or does the opposite happen?

Conclusion

Neuroplasticity is basically a mechanism that allows for an organism to adapt to changes in the environment. While this phenomenon is occurring daily in minuscule amounts, major changes in the processing of environmental stimuli through alterations in the functioning of different parts of the brain have been shown to be possible. These changes, while generally positive in nature, can also have negative consequences as seen in the development of chronic pain.

The development of chronic pain is seen in changes occurring in the central, peripheral and autonomic nervous systems. A change in one of these systems not only affects its own functioning but the functioning of the other parts of the nervous system. Biofeedback procedures offer the health care provider an insight into these systems individually and collectively.

Too often, the health care provider is trained in understanding only one system, often ignoring the impact of the remaining unchanged systems. Perhaps this is why short-term benefits (pain reduction) are reported but long-term results show the need for continued treatment or a relapse to the chronic pain cycle. Treatment programs which utilize multi-modality techniques are now demonstrating significant long-term outcomes (Flor, Fydrichc & Turk, 1992).

Biofeedback techniques offer the health care provider a unique ability to see into the functioning of the three parts of the nervous system. No other techniques offer this ability. It is incumbent for the practitioner to be aware of the strengths and limitations of one's techniques and refer to others who have different skills.

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