Chapter 4

Treatment of Psycho-Neurogenic Non-Pathological Neuromuscular Dysfunction Utilizing Electromyometric Feedback

It has been said that human beings, like electricity, follow the path of least resistance. In general, we exhibit a natural tendency to seek the least emotionally stressful pathway through life. Due to this tendency, we may fall into a peculiar trap referred to as "***defense mechanisms***." These mechanisms take many forms: phobias, obsessions, compulsive behavior, paranoia, and somatization. ***Defenses*** take form in the unconscious part of the mind against guilt, fear, anxiety or frustrations. It would appear easier for the human mind to deal with something more concrete, like expectations, images, ruminations, self deceptions, externalized fear (phobias), compulsive behavior or physical pain, than to deal with frustration, stress, guilt or anxiety. All people experience these defense mechanisms in varying degrees. Usually they become manifest in everyday circumstances and may even have considerable value, but they can become a problem when they begin to be seen as symptoms and to dominate a major portion of our lives. Should the symptoms become dominant in the extreme, they may make it difficult to maintain social or familial interpersonal relationships, to function in an occupation, or even to maintain feelings of identity and self-esteem.

Several biofeedback modalities and techniques have proven useful in dealing with some types of symptoms of defensive mechanisms. Electromyometry has proven especially effective in the treatment of the somatizing defense mechanism symptoms that involve neuromuscular function. These include the tension ***headache, the neck and shoulder pain syndrome***, the ***temporomandibular joint (TMJ) syndrome***, the ***low back pain syndrome*** and ***torticollis***. All of these may occur because of neuromuscular imbalance created by unconscious interpretations and responses to externally or internally induced emotional stresses. Many patients who have developed ***neuromuscular somatization dysfunction*** have reported an emotionally traumatizing conflict occurring shortly before or after a traumatic muscular injury (strain, sprain, whiplash or percussion). Possibly, the supraspinal structures (the brain's structures acting in concert) conceptualize (on an unconscious level) that it is easier to deal with physical pain than emotional pain. Subsequently, these structures induce pain by over-tensing previously injured extrafusal or intrafusal muscle or muscles associated with the previous injury.

Brodal (1974) notes that the gamma motor system, which is responsible for maintaining muscle tone, is so attuned to supraspinal structure influence that muscle tone is directly affected by mood changes. It appears from observation that such changes in muscle tone are in fact ***programmed responses*** to a given set of stimuli. In other words, unconscious neuromuscular defense somatization is a function that is learned. It is a triggered neuromuscular response to environmental circumstances just as any other fine motor skill.

Actually, the defense mechanism might serve as a convenient way of avoiding abstract emotional stress except that it is a programmed neuromuscular function. As such, with reinforcement, it soon becomes as habitual as the neuromuscular programming discussed in the previous two chapters, and it inconveniently refuses to go away when no longer needed. It may even have the disadvantage of becoming a generalized response to any emotional stress, good or bad. For example, the lady who only had tension headaches when she was under pressure while typing (at work) soon finds herself having tension headaches whenever she gets a little excited about anything. At this point, the symptom is no longer useful and has begun to interfere with normal daily living and there is usually a strong desire to get rid of the dysfunction. This desire may lead to a request for therapy.

To treat the neuromuscular defense mechanism (somatization), it is first necessary to alert the supraspinal structures that there is a neuromuscular dysfunction. Usually this dysfunction takes the form of a neuromuscular imbalance between back, shoulder, and neck or jaw stabilizers on one side of the body when compared with the musculature of the opposite side. This can be determined and demonstrated for the patient with electromyometric feedback as illustrated in **Figure 16** of **Chapter 2**. The patient can be taught to correct the dysfunction by utilizing the electromyometric feedback to reprogram the supraspinal structures to change responses to emotional stimuli. Programming must be performed in the manner in which the supraspinal structures are prepared to receive it. The sequence suggested must be taught in the way that optimizes learning. The protocol discussed in **Chapter 2** still applies: (1) ***facilitation*** of weak or less active musculature, (2) ***inhibition*** of overactive musculature, (3) ***balancing*** of opposing musculature, and, (4) using regained muscle control ***functionally***. The following techniques were developed independently of one another through a trial and error process. Step by step, an explorative protocol of treatment emerged which produced predictable patient improvement. The above theme emerged as the basic protocol upon which these techniques were built.

Headache, Neck and Shoulder Pain Syndromes

The pain associated with the ***headache, neck and shoulder pain syndrome*** is usually produced be ***trigger points*** (intrafusal muscle spasm) or spasm of the extrafusal muscle housing the trigger points (Taylor, 2002). The tension headache, for example, usually involves trigger points in the ***sternocleidomastoid***, ***upper trapezius*** or ***splenius capitus*** muscles. Together, they can produce referred pain patterns which may vary from simple aching pain at the base of the skull, or on the top of the head, to pain which covers one side of the head and is accompanied by "stabbing" eye pain, dizziness, nausea and distorted vision. The severity of headache pain and the accompanying secondary symptomology may be so pronounced that the condition may be misdiagnosed as a migraine headache. Although the symptomology may resemble a migraine or vascular headache, the condition will not respond well to vasoconstriction medication. The upper trapezius and sternocleidomastoid muscles together may be entirely responsible for the migraine-like symptoms. The sternocleidomastoid trigger point produces a pain pattern that is seen on only one side of the head, and at the same time produces dizziness and nausea. The symptoms occur as a result of pressure exerted by the sternocleidomastoid

on either the Eustachian tube or the acoustic artery, as they cross near the sternocleidomastoideus insertion on the mastoid process, as the extrafusal muscle shortens, causing a differential pressure between the two middle ears. The effect is a disorientation of the vestibular balance mechanisms, resulting in dizziness and nausea. The upper trapezius produces a referred pain pattern that radiates from the base of the head over one ear and into or behind the eye. The eye pain seems to affect the extra orbital musculature on the involved side by causing it to tighten and pull the eye out of alignment with the other eye, causing double or blurred vision. This may be misinterpreted as the prodromal "aura" of the migraine headache. Thus, the trigger points housed by these two muscles combine to produce a ***migraine-like*** (pseudo-migraine) headache, but all of the symptomology may be purely the result of referred pain patterns and other related mechanisms (Taylor, 2002).

The ***tension headache*** is notorious for not responding well to medication. The physician often has had to resort to medication that knocks the patient out to give the patient relief from the pain. In extreme cases, the recuperation time may involve bed rest and heavy medication. The patient may have a future full of extreme and sudden periodic attacks. This may be avoided if the condition is properly evaluated and treated.

A ***true migraine*** is a ***vascular headache*** caused by an over distention of certain arteries in the meninges of the brain. These vessels are allowed to dilate, and the normal blood pressure exerting itself on the meninges through the vessel walls produces the throbbing pain and associated symptomology. As the dilation occurs, other vascular beds are constricted in a reciprocal neurovascular balance, including certain capillary beds in the hands. Although not always the rule, if it can be determined that the capillary beds have indeed constricted with headache onset, this response can be used as corroborating evidence of the migraine diagnosis. This vascular reciprocal relationship may be used directly as a diagnostic aid by applying hot and cold contrast baths to the hands. If this relieves the pain, even just for minutes, it is positive evidence of a vascular headache. A similar relationship exists between the capillary beds in the ***pinna of the ear*** and the meningeal arteries. This relationship, however, is based upon a coinnervation of these structures. To use it diagnostically, a quick ice massage over the pinna of the ear (three quick brushes of an ice cube over the back and front curls of each pinna) may be applied to immediately relieve the headache. If the relief lasts for several minutes it may also confirm the vascular headache diagnosis.[1] Other evidence of a ***true migraine*** will also include the "aura" effect on vision. The ***true aura*** involves figures or distorted lines floating through the visual field.

Should evidence of migraine be missing and evaluative evidence suggests that trigger points and hypertonus of key muscles may be responsible for the headache syndrome, it should be treated as a tension headache. Even if positive evidence of the true migraine does exist, an evaluative survey of possible trigger points and muscular hypertonus should be made. A vascular headache will often be accompanied by a tension headache,

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1 *The pinna of the ear is vascularly co innervated with the temporal meningeal artery and if the quick ice causes vasoconstriction of the vessels in the ear, it will cause simultaneous vasoconstriction of the temporal meningeal artery.*

with the two accentuating each other's symptoms. This ***mixed syndrome*** condition is not rare, and the therapist must be prepared to identify and treat both components appropriately if the entire condition is to be ameliorated. The vascular component should be treated with temperature training of the capillary beds in the hands and ear. The patient should be taught to voluntarily increase the surface temperature of the hand and decrease the surface temperature of the pinna of the ear. The muscle components should be treated with suitable modalities, depending on the acuteness of the condition. These modalities may include electromyometric feedback.

Most headache syndromes are not as complex as the pseudo-migraine or the mixed syndrome, and their origins can easily be identified by their characteristic pain patterns and palpation of possible trigger point sites. The same is true of the ***neck and shoulder pain syndromes***. The ***neck pain syndrome*** usually emanates from trigger points in the ***sternocleidomastoid***, ***splenius capitus***, ***upper trapezius***, ***lower trapezius***, ***posterior cervical***, ***levator scapulae*** or the ***infraspinatus*** muscles. The referred pain patterns of these trigger points are often mistaken to have arthritic or discogenic origins, but if treated with cervical collars, traction, heat packs, diathermy or ultra-high frequency sound, there is usually a failure to respond or even an increase in the intensity of pain.

The ***shoulder pain syndrome*** usually emanates from trigger points in the ***lower trapezius***, ***levator scapulae***, ***posterior cervical***, ***upper trapezius***, ***infraspinatus***, ***pectoralis major*** or ***scaleni*** muscles. The resulting referred pain patterns are often erroneously labeled as radiculitis, shoulder/hand syndrome, tendonitis, capsulitis, bursitis or osteoarthritis because of incomplete evaluation or diagnostic assumptions. Careful orthopedic evaluation can distinguish these conditions from trigger point syndromes. Should misevaluation occur and inappropriate treatment be given, little improvement or an increase in the intensity of the pain symptomology may result. [2]

Like the headache syndrome, the neck and shoulder pain syndromes must be correctly evaluated before they can be appropriately treated. Many times trigger points will be found which prove to be isolated (intrafusal spasm) without extrafusal muscle involvement (hypertonus or spasm). These should be treated with appropriate modalities (Taylor, 2002). If extrafusal muscle hypertonus or spasm can be demonstrated with or without accompanying trigger points (through electromyometry), then it may be possible to treat these syndromes, at least in part, with electromyometric feedback.

The Breathing Technique

Early in the 1970's, it was clinically popular (but inadequate) to use general relaxation techniques (frontalis or "wrist-to-wrist" relaxation) to treat ***headache, neck*** or ***shoulder pain syndromes***. They were sometimes helpful in developing body awareness, but did little to reduce the pain syndromes overall. Likewise, attempts at direct relaxation of the key offending muscles involved in the related pain syndromes (***sternocleidomastoid*** or

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2 *Medically, "muscle relaxants" are typically given to help with pain syndromes that are thought to be muscular in origin. The typical medications used are actually central nervous system depressants and have no direct effect on muscle tissue.*

upper trapezius muscles in ***headache and neck pain syndromes***) often proved fruitless. Consequently, trigger points were treated with more traditional modalities like icing and stretch, ice packing, electrical stimulation, soft tissue manipulation and isometric exercise. While these techniques were often helpful in reducing the pain intensity and episodic frequency, they proved to be poor curative agents. Until the ***Breathing Technique*** was developed, we were unable to provide a way of dealing directly with the somatization responsible for such syndromes.

The ***Breathing Technique*** developed from the work done with a woman patient in her late twenties, who had suffered from a headache syndrome for five years. She initially reported that she suffered from headaches on the left side of her head. These headaches had occurred almost every working day since beginning work for a particular person. The pain followed the referred pain pattern characteristic of the ***upper trapezius*** trigger point syndrome. We were able to establish, through palpation, that she did, in fact, have an upper trapezius trigger point on the left side. An electromyometric evaluation showed that the extrafusal left upper trapezius muscle was myoelectrically hyperactive (above five-microvolts at rest). Direct upper trapezius relaxation with electromyometric feedback had been abandoned because of previous repeated failures and on the advice of a physician who utilized hypnosis in his gynecology practice. He stated that the upper trapezius was so unconsciously controlled that it was the last surface muscle to relax in a hypnotic state. Consequently, this patient's referred pain headaches were treated with direct applications of electrical stimulation, transcutaneous (electrical) nerve stimulation and isometric exercises. There was a reduction in the number of headache episodes, but no complete resolution. This patient had an abnormal upper ***chest-breathing*** pattern, commonly called "***accessory muscle breathing***." Electromyometric evaluation confirmed that almost no diaphragm myoelectric activity was taking place but, bilaterally, her ***accessory*** and ***upper trapezius*** muscles were myoelectrically hyperactive during inspiration.

Electromyometric feedback was utilized to teach her how to use her diaphragm. After a few attempts, with one hand placed over her upper abdominal muscles, so that she could "feel" her response as well as hear it and see it on the electromyometer, she was able to demonstrate a marked increase in diaphragmatic myoelectric activity. She was then allowed to see the myoelectric activity from the accessory muscles and was encouraged to increase simultaneously myoelectric activity from the diaphragm as she reduced it from the accessory muscles, utilizing ***two electromyometers***. She was able to accomplish this in a single session. The upper trapezius myoelectric activity was rechecked and coincidentally showed a marked decrease in involuntary myoelectric activity both during rest and inspiration. After two reinforcing sessions, she reported a marked decrease in the number and intensity of the headaches. Reevaluation showed remarkably little myoelectric activity from the accessory and upper trapezius muscles during inspiration. She was then asked to count to ten out loud. As she proceeded through the sequence, the myoelectric activity rose from close to one-microvolt (mv), to above 12. She was then asked to once again count to ten out loud, but to try to keep the myoelectric activity below a given level (eight to ten mv). After several attempts, she was able to demonstrate that control. She was then asked to develop similar control over the upper trapezius

muscles during counting. After many more attempts (this took a little longer), she accomplished it. On her next visit, she reported that there had been further improvement in her headache condition. At this point, she demonstrated very good control of both the accessory and upper trapezius muscles by being able to keep myoelectric activity below six mv from either muscle during breathing and counting. She was next asked to say out loud the names of people she knew (with both favorable and unfavorable associations). It became immediately apparent from her myoelectric responses, that certain names were more somatically provoking than others. With practice, she soon learned to inhibit these myoelectric over-responses, and then began exerting similar control over the accessory and upper trapezius muscles during general conversation. It took her several sessions for her to fully control her myoelectric responses, but by the end of these sessions, she was able to report a complete cessation of her headaches. Months later she reported that aside from occasional "small" tension headaches (apparently related to too much typing), she was headache free.

Evaluation and treatment of the ***head, neck and shoulder pain syndrome*** is fairly standardized. On the initial visit, a thorough history is taken regarding the events leading up to and following the onset of the pain syndrome. A survey is then made of the possible contributory factors. Trigger points are noted, and appropriate neurological and orthopedic tests are performed and responses noted. An electromyometric evaluation is performed to establish the presence of abnormally high myoelectric activity.[3] Readings are taken during the events of breathing and talking (counting and saying the names of people, with both positive and negative connotations). If the electromyometric evaluation is negative, then a program employing other appropriate modalities is begun. If the electromyometric evaluation is positive, and if other contributory factors are demonstrated besides trigger points (osteoarthritis, tendonitis or acute extrafusal muscle spasm, etc.), then an appropriate modality program is begun including the ***Breathing Technique***. If the electromyometric evaluation is positive with or without contributory trigger points, the ***Breathing Technique*** is applied.

In the ***Breathing Technique***, the myoelectric activity from the responsible muscles is monitored. The ***upper trapezius*** is usually found to be representative of myoelectric hyperactivity in the upper back, shoulder, and posterior cervical areas, while the ***scaleni*** muscles may be representative of accessory and sternocleidomastoid muscle hyperactivity. Should the muscles be symmetrically involved (the ***upper trapezius muscles bilaterally***, for example) they are monitored simultaneously by splitting the pickup electrodes from a single lead between them. More commonly, the pickup electrodes are both used to monitor a muscle unilaterally. The unilateral placement is favored, especially for evaluations, since it allows contralateral and homolateral comparison between shoulder and neck muscles, respectively. Placements used during the training phases are usually homolateral and on the involved side (for diaphragm

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3 *Ideally the patient should not be able to see or hear the instrumentation during the evaluation. This allows an establishment of myoelectric activity levels without patient conscious or unconscious interference.*

electrode placement, refer to **Electrode Placements**). A split lead placement between the scaleni and upper trapezius muscles is sometimes used when these muscles are equally involved, homolaterally.

Once electrode placements are selected, electromyometric feedback neuromuscular reeducation is begun as outlined in the previous case study. The patient is encouraged to begin facilitation of myoelectric activity from the ***diaphragm*** (***muscle A***) on inspiration. When increased myoelectric activity has been sufficiently demonstrated (four to eight mv) the patient is encouraged to ***inhibit*** myoelectric activity from the least overactive representative muscles (***muscle B***, usually the ***scaleni*** muscles) and to keep it below a given level as she inspires. Then she is asked to ***facilitate*** myoelectric activity from ***muscle A*** on inspiration as she inhibits myoelectric activity from ***muscle B*** (***balance***). During these attempts, the patient should be instructed to take only moderately deep breaths.[4] Once the patient is able to keep the myoelectric activity from ***muscle B*** below the predetermined level (usually 6 mv) the patient is encouraged to attempt the same feat with the more hyperactive representative muscle (***muscle C***, usually the ***upper trapezius***).[5] When control of ***muscle C*** is demonstrated during breathing, ***muscle B*** is again monitored, and the patient is asked to count out loud while attempting to inhibit myoelectric responses from it (the ***diaphragm*** is no longer monitored unless the stress of the control attempts warrants using breathing control to regain lost control of the ***accessory*** or ***upper trapezius*** muscles). Once control of ***muscle B*** is demonstrated the same process is repeated with ***muscle C***. The next step is to achieve the same kind of control with each respective muscle while the patient says names of people she knows out loud. Finally, myoelectric control of ***muscle B*** and ***muscle C*** should be attempted while the patient converses with the therapist. This last step should closely resemble the procedure utilized in desensitization employed with the galvanic skin response monitor. The three talking steps constitute the ***functional phase*** of training.

In this technique, we have closely approximated the theme of ***facilitation, inhibition, balance*** and ***function*** suggested earlier. When this approach is coupled with selective toning of the involved muscles (isometric exercise) to help avoid strain that might inadvertently revive the syndrome, it has been demonstrated that the patient has an excellent chance of defeating the somatization of the ***headache, neck and shoulder pain syndromes***.

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4 *Deep breathing inadvertently encourages involvement of all shoulder and neck muscles, which may be used during inspiration.*

5 *To help the patient develop control, from a realistic point of view, the patient is encouraged to attempt to keep the overactive muscle's activity below levels that might be possible in their particular state. Some* patients begin with overactive readings of more than 20 mv, so initial legitimate expectations would be for the patient to attempt to maintain activity below 15 mv, for example. As relative control is demonstrated, the control limits should be progressively lowered until the optimum six mv level is reached.

The Temporomandibular Joint Pain Syndrome

The ***temporomandibular joint (TMJ) pain syndrome*** characteristically demonstrates pain patterns typical of trigger points resting in the ***temporalis*** or ***masseter*** muscles. It is one of the pain syndromes often mistaken as a ***tension*** or ***migraine*** headache. To help establish the possibility of the ***TMJ pain syndrome*** being present, the appropriate orthopedic tests, Acuscope and auricular acupuncture and trigger point surveys are made. Conditions not strictly belonging to the ***TMJ pain syndrome*** described here are treated with other modalities. The common TMJ pain syndrome does not typically demonstrate evidence of orthopedic problems. Trigger points may be palpated, but usually only as secondary elements of the true ***TMJ pain syndrome***. Electromyometric evaluation consistently (with few exceptions) confirms an electromyometric imbalance between the jaw muscles on opposing sides of the face to be the primary cause of the syndrome. The electromyometric differences between the two sides may vary from a few mv to over a 100 mv during rest and teeth clenching. Interestingly enough, it is sometimes found that the overactive side is not the painful side. In such cases, it is thought that the over activity being produced on one side puts a subluxing strain on the opposing side, straining involved ligaments. The under active jaw muscles may also be strained as they attempt to maintain joint integrity and cause themselves to produce trigger points. If the pain pattern is on the same side as the excessive myoelectric activity, it is postulated that the pain is produced from forceful ***joint approximation***, ***extrafusal spasm*** or accompanying ***trigger points***. Whatever the cause of the pain, it may represent a ***somatization defense mechanism***. A carefully taken history and electromyometric evaluation may help to confirm this possibility.

An evaluation of the myoelectric activity from the jaw muscles requires two electromyometers. Each machine should be set to monitor two muscles or muscle groups simultaneously: masseter versus temporalis homolaterally, and contralaterally masseter versus masseter; temporalis versus temporalis, or masseter and temporalis versus masseter and temporalis.[6] Evaluation should be based on the following observations: (1) the jaw relaxed with the mouth slightly open; (2) the jaw as relaxed as possible; (3) the jaw voluntarily opened as far as possible; (4) the jaw relaxed as much as possible with the teeth touching; (5) the teeth firmly clamped together; and, (6) the teeth together with the jaw maximally clenched. Readings should be taken first ***without*** and then ***with*** an even ***bite platform*** between the teeth on each side (two halves of a tongue depressor, for example). Ideally, the patient should not be allowed to hear, see or feel the feedback modes during the evaluation, to provide a true picture of myoelectric activity without the patient's conscious or unconscious modification of those readings.

Should an imbalance be seen between muscles or muscle groups, neuromuscular reeducation is indicated. The muscle or muscle group exhibiting the highest myoelectric

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6 A thorough evaluation will include readings from each of the muscles suggested. ***Special Note***: The treatment program described here assumes the patient to have marked consistent myoelectric imbalance during the separate events. Should a patient not demonstrate an imbalance during all events the program may be modified to efficiently meet the patient's needs by deleting unnecessary steps in the protocol.

activity is designated ***A***, and the muscle or muscle group exhibiting the lowest myoelectric activity is designated ***B***. To be consistent with the theme of treatment, the patient should be encouraged to learn to facilitate an increase in myoelectric activity from ***muscle B*** while biting down on the bite surface provided (tongue depressors). When the patient is able to generate myoelectric activity equivalent to that produced by ***muscle A*** with the biting platform, she should be asked to repeat the feat

without the bite platform (assuming that dental structure allows this movement).[7] The patient should then be asked to inhibit voluntarily myoelectric activity from ***muscle A***, first with the teeth together, then with the mouth slightly open, then finally, with the mouth open as widely as possible.[8] When the patient is able to demonstrate inhibitory control (less than six mv) of ***muscle A***, she should be asked to facilitate myoelectric voluntarily activity in ***muscle B*** as she inhibits myoelectric activity in ***muscle A*** as a balancing function. This should first be done with the bite platform between the teeth on the uninvolved side, or on both sides. The goal should be for ***muscle B*** to be able to generate at least five-times more myoelectric activity than ***muscle A*** (in the 0 to six mv range). This should be repeated without the bite platform(s) between the teeth. The last step is to have the patient inhibit excessive myoelectric activity from ***muscle A*** as she proceeds through a desensitization sequence similar to that described in the previous material on ***headache, neck and shoulder pain syndrome*** to provide a functional phase.[9]

Should the patient also suffer from a ***headache, neck or shoulder pain syndrome***, the desensitization phase should include variations in electrode placements. The pickup electrodes should be split between the prominently hyperactive muscles in each syndrome. For example, a patient who suffers from both a headache syndrome, in which the left ***upper trapezius*** was hyperactive, and a TMJ syndrome in which the left ***masseter*** was prominently hyperactive, should have both the left upper trapezius and left masseter muscles monitored together by the same electromyometer as the ***desensitization sequence*** (counting, names, and conversation) is performed.

Though success with this technique for patients who have dental structure abnormalities has sometimes been temporary (lasting for several months), it has been notably successful as a cure for patients with simple muscular imbalance and accompanying trigger points. This technique has been notable as a successful treatment for ***bruxism*** (nocturnal unconscious clenching of the jaw or grinding of the teeth).

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7 Facilitation attempts should last for 6 seconds, followed by a six-second-relaxation period to maximize the toning effect of the isometric exercise and to provide for maximal opportunity to reinforce successful attempts.

8 *Some patients have demonstrated an inability to voluntarily part the jaws very far. The patient may be taught to voluntarily inhibit myoelectric activity of the jaw muscles as the jaws are progressively separated (tongue depressors progressively taped together, one on top of another, may be used).*

9 *Should the patient present no dental structural abnormalities, it is suggested that food chewing should only take place on the weak side as a functional step. Later, chewing time should be equally divided between the two sides.*

The Low Back Pain Syndrome

The low back syndrome characteristically demonstrates referred pain patterns from the ***iliocostalis***, ***gluteus medius***, ***longissimus***, ***multifidus*** and ***gluteus minimus*** muscle ***trigger points***. Because so many conditions can cause the symptomology usually described by the patient with a ***low back pain syndrome***, a careful history is taken. Attention is paid to the events leading up to and following the injury, the mechanism of injury and the patient's response to any previously applied modalities and also the home, family and work ***emotional environment***. This is done (1) to establish if circumstances may have precipitated a ***somatization defense mechanism***, (2) to explore the possibility of any undiagnosed or misdiagnosed elements, and (3) to establish ***environmental factors*** that are contributory in sustaining the pain syndrome. All appropriate orthopedic and neurological tests are performed, as well as a thorough trigger point. An electromyometric evaluation is performed to establish if a ***muscle imbalance*** may be a factor (the electrode placements are illustrated in **Electrode Placements**).[10] The pickup electrodes are attached to the superior and inferior placement sites bilaterally.[11] The right side placement is called ***A*** and the left side placement is called ***A' (A Prime)***. The superior and middle electrode sites taken together on the right side are called placement ***B***, and those on the left side are called placement ***B' (B Prime)***. The middle and inferior electrode sites taken together on the right side are called placement ***C***, and those on the left side are called placement ***C' (C Prime)***. ***A*** and ***A'*** placements provide for a rather gross assessment of back muscle myoelectric activity. If no difference in the two is noted and the readings are ***not abnormally high***, the evaluation is said to be ***negative***, and electromyometric feedback discarded as a possible treatment modality. The evaluation should continue if an imbalance appears between ***A*** and ***A'***, or if the readings are symmetrical but excessively high, then ***B*** and ***B'*** placements are then monitored and compared as are ***C*** and ***C'***. These readings are first taken with the patient ***sitting***, and then ***standing***. All of these readings are taken to determine the location of ***myoelectric imbalance*** or over activity, as well as providing information on habitual posture and functional problems contributing to the disorder. To get true readings without patient interference, the patient must ***not*** be allowed to view or hear myoelectric responses.

When the location of ***imbalance*** or ***hyperactivity*** has been determined and the positions producing the greatest abnormal readings has been established, a program of ***electromyometric feedback neuromuscular reeducation*** is recommended. The technique recommended for a low back imbalance is peculiar when compared with the other neuromuscular reeducation techniques previously discussed. Observation has shown that one electromyometer may be utilized for the entire program (though two units

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10 The low back syndrome discussed here may have associated trigger points and other orthopedic pathological factors (osteoarthritis, facet syndrome, etc.) but will not demonstrate orthopedic structural damage (discogenic disease, for instance) resulting in nerve impingement. Instead, it demonstrates excessive myoelectric activity on one side of the back when compared with contralateral activity.

11 The remaining electrode site is used for ground electrode attachment.

have been successfully used in a manner consistent with previously discussed techniques). The patient is first asked to find a sitting position or posture that produces comparatively little myoelectric activity from the location of hyperactivity (the position of minimal activity). The patient is then directed to change positions slowly, while keeping the myoelectric activity below a ***reasonable control level*** until achieving a normal sitting posture.[12] Initially the patient (with an imbalance while sitting) is usually unable to find the position of minimal activity while in the sitting position. Having tried and failed, the patient is then directed to stand with feet three to six-inches apart and with knees straight.[13]

The patient should then attempt to find a standing position that produces myoelectric activity from the hyperactive location, and then slowly shift toward a more normal standing position while keeping the myoelectric activity below the ***reasonable control level***.[14] The patient usually finds the position of minimal activity by shifting the body weight back onto the heels (most ***low back pain syndrome*** patients unconsciously try to avoid the back pain by shifting the weight to the balls of the feet to "lean away" from the pain, which increases the lordotic curve and the potential for strain and pain), or standing with most of the body weight on one foot. When a normal standing position is achieved, while keeping the myoelectric activity below the reasonable control level, the patient is asked to once again assume the ***position of minimal activity***. Predictably, the myoelectric activity experienced in the ***position of minimal activity*** will have decreased, and a new ***reasonable control level*** can be established. This process of shifting with inhibition should continue until normal activity levels are reached (***"normal"*** is usually established by readings taken during evaluation from uninvolved muscle areas; it usually ranges from two to eight-mv when standing still).

The patient is then directed to once again sit down and attempt to find a ***position of minimal activity***. The patient is usually able to find the position of minimal activity on this second attempt and is also usually able to demonstrate control of myoelectric activity when shifting to assume a more normal sitting position. Eventually, the patient should be able to sit in a normal position with the myoelectric activity below four-mv If necessary, the patient should return to a standing position and repeat the ***shifting inhibition process*** until control is also demonstrated. The final step may include carrying out the desensitization sequence discussed in the two previous technique descriptions, but using the hyperactive back muscles as the muscles to be inhibited. When full control of myoelectric activity has been demonstrated in the sitting and standing positions and during the desensitization sequence, the pain-generated from the muscular imbalance has

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12 The reasonable control level is usually based on how low the myoelectric activity is when a position of minimal activity is established. Usually a few microvolts or above that level of activity in the position of minimal activity is established as the immediate reasonable control level.

13 Bending the knees mechanically reduces muscle activity by tilting the pelvis anteriorally and relieving the lordotic curve. Experience has shown that controlled straightening of the knees will not aid in teaching low back myoelectric inhibition.

14 Electromyometric monitoring of back myoelectric activity has proven useful in helping the patient acquire awareness of body position and has served as an aid in helping correct pain generating postures.

usually disappeared. Any residual pain must be assumed to be a product of other factors (including ***trigger points***) and must be treated appropriately. Routinely, ***isometric exercise*** is employed to tone the back musculature to help avoid strain that might reactivate the ***low back pain syndrome***.

Special Note

Although the aforementioned techniques were presented as treatment procedures suggested for pain syndromes affecting neck, shoulder, thoracic and low back areas that do not demonstrate discogenic or other neurogenic damage, it should be noted that cases involving such structural defects have been successfully treated with those techniques. A good example was a woman in her early forties who had been diagnosed as having a ruptured cervical disc and an advanced ***cervical dorsal outlet (CDO) syndrome***. The ***ruptured disc*** resulted from an awkward fall (two years prior) and the ***CDO syndrome*** had developed slowly thereafter. She was told that there was pressure on the radicular nerve roots in the left cervical area and that a progressive peripheral nerve impingement was occurring between the scalenus muscles. She began experiencing radiating pain from the left neck to the tips of all the fingers in the left hand and was also experiencing numbness throughout the left upper extremity. She had been hospitalized by an orthopedist, and a cervical laminectomy fusion was recommended to relieve the radicular nerve pinch. A neurosurgeon was consulted, and he recommended relieving the pressure on the nerve between the scaleni muscles by ***removing the first rib*** (upon which they insert). The patient refused to make any immediate decision. When the pain decreased somewhat, she returned home.

When initially evaluated for treatment, the patient demonstrated the symptoms previously mentioned and stated that they were chronic and constant. She indicated that she had been a college student studying for her Master's degree just before the accident, and for a short time afterwards, but had to stop because of increasing pain. She also mentioned that she was the mother of two teenagers, and indicated that she and her husband were members of a religious group that frowned upon higher education for women. The position taken by this group was that a woman belonged in the home or in the church.

An electromyometric evaluation of her neck and shoulders was performed with positive results. The scaleni muscles proved to be the most active when compared with the upper trapezius on the left side. Without much optimism, the ***breathing technique*** was applied. The patient readily learned to control her breathing pattern. In a single session, she gained control of the upper trapezius and the accessory muscles, and the pain and numbness disappeared from her left neck, shoulder and upper extremity entirely. Over the next several months, she was seen twice a week. She was able to complete successfully the ***Breathing Technique sequence***. Several modalities were used to treat occasional muscle strain, and a ***Galvanic Skin Response monitor (GSR)*** was used to help complete the process of ***desensitization***. ***Isometric exercise*** was used to help tone the muscles involved. Eventually, she was able to return to school and continue her education. She was seen occasionally after that when emotional trauma combined with

long periods of physical inactivity and lack of exercise (usually around final examination time) partially revived the syndrome, but those episodes were brief.

To conclude, it seems apparent that when muscle tension plays a role in effecting an orthopaedic or neurogenic pathological condition, that condition may be treatable, to a given extent, by neuromuscular reeducation utilizing electromyometric feedback.

Torticollis

Torticollis (wry neck) is a good example of a neurogenic disorder that manifests itself through neuromuscular imbalance. This disorder typically involves the ***sternocleidomastoid muscles*** bilaterally. Characteristically, one of the pair of sternocleidomastoid muscles (on the involved side) is hyperactive and comparatively stronger than its counterpart. Consequently, the patient's chin is drawn toward the weak side and elevated while the ear on the involved side is pulled down toward the shoulder. It may remain statically in this position if unopposed. If the opposing sternocleidomastoid muscle actively fights this action and a ***phasic stretch reflex*** occurs from the involved muscle, the head will be jerked back and forth in ***"spastic"*** maneuvers. Sometimes the ***upper trapezius muscles*** become involved, adding complexity and eccentricities to motion pattern or head position. Unless ***cerebral damage*** has precipitated this neuromuscular dysfunction, it is ***another expression of somatization***. Although neck muscle strain with its accompanying pain is often responsible for the precipitation of the condition, after the acute stage, it is often relatively painless. Pain may later develop, however, from muscle strain or spasm, should the patient consciously attempt to stabilize or change the head position. ***Muscle spasm*** and joint pain may also result from osteoarthritic joint changes that may occur from prolonged violent cervical joint rotation and unequal pressure into the vertebral joints. These secondary pain problems complicate the rehabilitation program by limiting the effectiveness of the ***electromyometric feedback neuromuscular reeducation*** program.[15] Successful treatment will depend on the amelioration of these secondary symptoms, as well as neuromuscular reeducation, since the pain involved may provoke ***patient anxiety*** that tends to increase the force and violence of the muscle contractions.

The initial evaluation should include the taking of the ***history of events*** leading up to the onset of the disorder to help differentiate between the ***pathoneurogenic*** and ***psychoneurogenic*** nature of the syndrome. An electromyometric survey should then be taken of the myoelectric activity from each sternocleidomastoid and upper trapezius muscles bilaterally (for electrode placements see **Electrode Placements**), ***without*** allowing the patient to see or hear the feedback modes, to prevent conscious or unconscious interference with myoelectric responses. Readings should be taken from each muscle (upper trapezius and sternocleidomastoid, bilaterally) during each of the following eleven events. In the ***first event***, the patient should be sitting in a 45^{o} reclining position with the neck and head in a ***relaxed midline position*** (if possible). For the ***second event***, the patient should be brought to a ***full upright sitting position*** with the head

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15 *Pain syndromes may spontaneously appear as an apparent defense against therapeutic intervention.*

in midline. For the ***third event***, the patient should ***stand erect*** with the head in midline. For the ***fourth event***, the patient should be sitting ***and reclined to 45^{o}*** with the head in midline; the patient should be asked to ***lift the head and attempt to put her chin on her chest*** while the readings are taken. For the ***fifth and sixth events***, the patient should be sitting and ***reclined to 45^{o}*** and asked to turn the head ***forcibly toward each of the shoulders*** in turn while the readings are taken. For the ***seventh and eighth events***, the patient should be sitting and ***reclining to 45^{o}*** and asked to turn the ***head toward the shoulder*** and to lift the head so that the head nearly ***touches the shoulder*** while the readings are taken. This should then be repeated for both shoulders. For the ***ninth, tenth, and eleventh events***, the patient should be sitting and ***reclined to 45^{o}*** with the head in approximate ***midline*** while ***counting out loud*** from one to ten, ***reciting names*** of people of positive and negative personal acquaintance and ***conversing*** on topics of concern while readings are being taken during each of these events.

The ***neuromuscular reeducation protocol*** should begin only after the ***overactive muscle(s)*** (***muscle A***) and the ***under active*** or weakened ***muscle(s)*** (***muscle B***) have been identified, and the events which have demonstrated the greatest adverse responses from those muscles has been noted (for re-evaluation purposes). Initially, the patient should be placed in a ***45^{o} sitting position*** and asked to facilitate voluntarily myoelectric activity from muscle B while turning the head toward the involved side to the end of range and trying to touch the involved shoulder with the chin. The muscle contraction should be ***held for six-seconds*** and then ***relaxed for six-seconds***. During the ***relaxation phase***, the patient should attempt to keep the head turned passively toward the involved side. This exercise should be repeated until the electromyometric readings approximate those elicited from ***muscle A***, established during the initial evaluation. This often requires several sessions. The work with the electromyometer should be supplemented by a home program of shoulder and neck isometric exercises, and should be performed with the head turned toward the involved side while in an upright sitting or reclining position of 45^{o}.

The next step in the program is to have the patient remain sitting in a 45^{o} reclining position with the head turned toward the involved side to the end of range. The patient should be directed to attempt to inhibit the myoelectric activity in ***muscle A***. After ***muscle A*** has "quieted", the patient should attempt to ***balance muscles A*** and ***B*** by increasing myoelectric activity from ***muscle B*** while attempting to ***inhibit*** myoelectric activity from ***muscle A*** at the level achieved during relaxation (this becoming the reasonable control level). Each attempt should be six-seconds long, followed by a six-second-relaxation period. The established goal is for ***muscle B*** to exhibit five-times the myoelectric activity of ***muscle A***. When this goal is reached, the patient should repeat the process with the head turned toward the involved shoulder to ***50% of full range of motion***. When balanced control has been demonstrated to the degree previously indicated, the patient should be directed to repeat this performance with the head in midline. The patient is then brought up to a full ***sitting position*** and the identical sequence of events described above is repeated. The patient is then asked to repeat the same sequence while ***standing***. Finally, to help make the gained control functional, the patient is required to stand and perform delicate hand tasks while keeping the muscles

equally balanced in the 0-12 mv range (ideally below six-mv). The patient should also be guided through the desensitization sequence suggested for the treatment of the headache, neck, and shoulder pain syndrome, while myoelectric activity from ***muscle A*** is being monitored and controlled. A ***GSR*** may also be helpful in fully ***desensitizing*** the patient autonomically to emotional stimuli concurrently with neuromuscular reeducation.

The Post Immobilization Syndrome

A common orthopaedic problem treated by the physical or occupational therapist is the ***Post Immobilization Syndrome*** of joints and involved muscles after extended periods (from weeks to months) of ***joint immobility*** in casts, splints or as the result of other incidental joint disuse (prolonged sling use or the kind of prolonged immobility observed in comatose or neurologically injured patients). The ***Post Immobilization Syndrome*** usually results in the patient being ***unable*** to lengthen or shorten the involved muscles to allow ***normal range of motion*** of the joint. Muscle contractures resulting from complete muscle atrophy are ***not*** included in this discussion because of the different nature of their etiology and treatment.

When joints are immobilized for extended periods the muscles involved are only allowed to shorten or lengthen through a small part of normal range. Consequently, the supraspinal structures accommodate tonically by changing the involved muscle length ranges allowed by the immobilization. Like all muscle functions that are repeated over an extended period of time, this activity is gradually turned into a neuromuscular program (developed in ***Computer D***) to form a ***new habit***. The longer the period of joint immobilization, the more reinforced the habit becomes, making it increasingly difficult to unlearn the habit when no longer needed.

The problem of joint range ***limitation*** is complicated by muscle atrophy and loss of muscle strength from the enforced muscle inactivity, making rehabilitation even more complicated.

Traditionally, the treatment of the lost range of motion and weakness that occurs as a result of prolonged immobilization has involved ***manual stretching***, ***isotonic exercise*** (usually progressively resistive), and sometimes exercise techniques that promote changes in muscle length as well strength (***Hold-relax and Contract-relax Proprioceptive Neuromuscular Facilitation techniques***). These techniques are often painful for the patient and frustrating for the therapist, since they usually depended on the patient being able to force change in muscle length (both intrafusal and extrafusal muscle fiber length) while ignoring the extreme pain involved. These techniques also have the disadvantage of ***stressing the soft tissues*** in the joint, increasing soft tissue swelling and acute and chronic pain.

A much more efficient technique for treating these problems has developed out of the use of electromyometry in the treatment of the spastic post CVA syndrome, which has as one of its goals the resetting of tonically set musculature. In the ***Post Immobilization***

Syndrome, however, there is the advantage of having an intact, undamaged nervous system to deal with, making the task potentially easier.

Evaluation

The first step in the treatment of any disorder should be complete and appropriate evaluation. To evaluate the ***Post Immobilization Syndrome***, initially, the voluntary and involuntary range of motion of the involved joints should be measured by goniometry followed by appropriate gross muscle testing. Then a survey is performed of electromyometric activity from all the involved muscles during each of the joint notions (***flexion***, ***extension***, ***internal*** and ***external rotation*** or ***abduction*** and ***adduction***). For example, should the knee be the involved joint, simultaneous electromyometric readings should be taken from the ***rectus femoris*** (quadriceps group) and ***hamstring muscles*** during voluntary extension as well as voluntary flexion. Electromyometric readings should also be taken from the same opposing muscles on the ***contralateral uninvolved side***, during all of the joint motions. These latter readings serve as a measure of normalcy for that patient and can serve as a comparative measure of improvement, both in terms of the percentage of normal muscle contraction and in terms of acceptable inhibition levels for ***antagonistic muscle activity***. All readings should be taken while the patient performs the contractions at the ***end of each range***, in a ***neutral range***, and ***with and without resistance***.

Treatment

The ***first step*** in the treatment process is to have the patient attempt to increase (***facilitate***) electromyometric activity in each of the involved muscles in turn, within a comfortable range of motion, first against resistance and then without resistance. When the patient can demonstrate an increase in electromyometric activity well above that demonstrated initially (the amount to be determined by the therapist), the patient should then shorten one of the involved muscles to the end of range and attempt to decrease the electromyometric activity from the stretched muscle to a relaxation level. When the stretched muscle is ***inhibited***, to the extent that its electromyometric activity approximates the relaxation level established for its counterpart on the contralateral side, the patient should attempt to ***voluntarily contract*** the shortened muscle isometrically against resistance while continuing to ***inhibit*** the stretched muscle. When the agonist exhibits at least ***twice*** the electromyometric activity of the antagonist, the patient should then be asked to ***contract the agonist muscle*** at the end of range ***without resistance***, thereby promoting an increase in agonist shortening and antagonist lengthening and possibly a range of motion increase. When an increase in range of notion has been accomplished the patient should again be asked to contract the agonist isometrically ***against resistance*** and repeat the sequence. Each contraction should be held for six-seconds followed by a six-second-relaxation period. After repeating the sequence several times the agonist and antagonist should reverse roles and the aforementioned process repeated at the opposite end of the range of notion.

As the ranges of notion approach normal extremes, ***isotonic exercise*** of the involved muscles (weight lifting, for instance) may be added to the program to foster functional adjustment of the neuromuscular system as well as to help increase muscle strength and muscle mass.

Special Considerations

Soft tissue swelling that often accompanies the conditions that lead to the need for immobilization is often still present when the immobilization is relieved. Ice packing and ***electrical stimulation*** may be helpful in reducing such swelling (Taylor, 2002).

Acute and chronic pain often accompany such conditions and the modalities used for the treatment of the acute pain elements of the trigger point referred pain syndrome including ice packing, electrical stimulation, soft tissue manipulation and phonophoresis of an effective anti-inflammatory are often helpful in its reduction (Taylor, 2002).