TREATMENT OF THE HEMIPLEGIC SHOULDER THROUGH BIOFEEDBACK: A CASE STUDY

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Abstract

Motor learning requires feedback. When a person is acquiring a new skill, or modifying a previously acquired skill due to physical dysfunction, sensory feedback optimizes motor learning.

Electromyography is a discipline that focuses on clinical and neurophysiological evaluation of neuromuscular pathology, and on certain aspects of CNS pathology (acquired or traumatic brain injury, etc.).

Currently, use of biofeedback devices, amplifying the signal produced at the motor endplates, has allowed the introduction of this therapeutic tool to treatment of patients with acquired brain injury. Biofeedback can provide reinforcement of motor control improvements acquired through physiotherapy sessions, and help with development of specific sensorimotor skills, not only analytically, but also during occupational tasks. Availability of portable devices that are easy to use has allowed widespread application of biofeedback to functional improvement in activities of daily living.

Key words

Biofeedback, superficial electromyography, operant conditioning, mental imagery, brain injury, baseline, threshold value, dispersion, tonic activity, E.P.P., E.M.D., activities of daily living.

INTRODUCTION

Electromyographic biofeedback systems are devices that amplify a physiological signal. In this particular case, the amplified signal represents muscle activity, presented in a form that is easy for the patient to understand, i.e. through graphs, animation, sounds, etc. This facilitates the subject to voluntarily control muscle activity by providing immediate information on the performance of a specific task.

The principal theories proposed to describe the effects of biofeedback training are based on two fundamental models:

a) Instrumental or operant learning model based on the importance of reinforcement and motivation.

b) Signal processing model based on the importance of motor image development at the cortical level.

BASIS OF BIOFEEDBACK

Servomechanism model

Servomechanisms are control systems that allow regulation of function, in this case, the muscular system. These systems are self-adapting; in other words, they change according to variations in the environment. Since they are open systems, they interact with the environment by modifying function according to changes introduced in the system. Therefore, we can say that biofeedback systems consider the nervous system of the patient to be a highly complex, open servomechanism, in which the electronic device provides the patient with information to generate feedback (fig. 1).

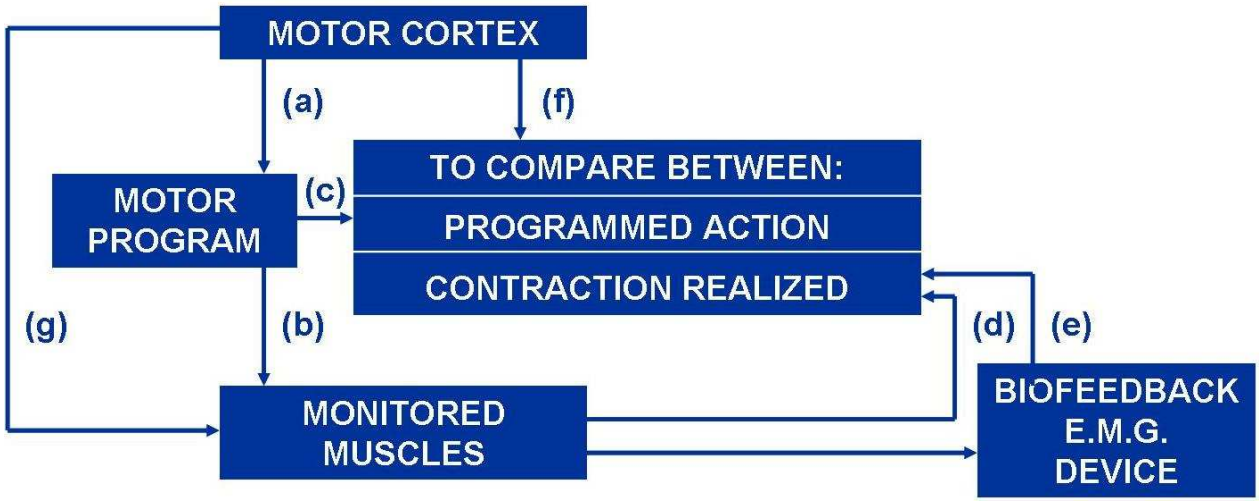


Fig. 1. Muscle contraction control with biofeedback and E.M.G. The motor cortex makes a decision (a) which is sent to the motor program, where the order is carried out (b). In the centre, the actual contraction [(d) and (e)] is compared to the programmed contraction (c), and the differences are sent to the motor cortex (f), where pertinent corrections are made.

Processing the feedback signal

Two types of feedback will be taken into consideration:

a) Positive feedback: Output (action performed) produces an effect of increasing input (information source), producing an acceleration effect that disrupts the homeostatic system.

b) Negative or inhibitory feedback: Output causes a decrease in input, restoring equilibrium.

Proprioception is an essential component of sensory feedback, along with visual, auditory and tactile input. Therefore, any deficit in the flow or organization of afferent sensory input (proprioceptive or exteroceptive) will produce an alteration in motor pattern performance.

Biofeedback and operant conditioning

Operant conditioning is the basis of trial and error learning. Any reward can be used to immediately reinforce any response emitted.

Biofeedback is sufficiently similar to operant conditioning in its methodology, to the point in which it can be considered an extended version of it. Physiological activity (in this case the muscle contraction) is delivered to a monitoring device that transmits the measured variable and returns it (feedback) in such a way that facilitates control of the given physiological variable (fig. 2).

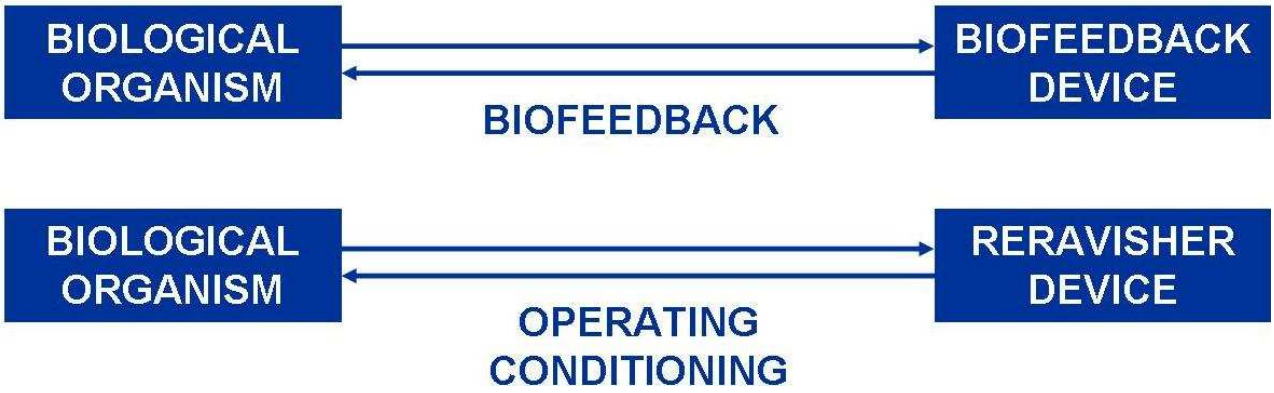


Fig. 2 Similarity between biofeedback and operant conditioning

Biofeedback requires an exteroceptive stimulus to accompany motor activity. This stimulus can be sound, light, graph, animation on a computer screen, etc.

Operant conditioning techniques in biofeedback

The basic principles of conditioning in biofeedback are:

a) Reinforcement: Defined as all action that increases (positive) or decreases (negative or inhibitory) the response frequency. In biofeedback devices, feedback provided by the patient acts as secondary reinforcement, since its presentation (light, sound, graphs on a computer screen, etc.) has little intrinsic value. Value is added when the patient establishes the feedback connection with improvement in his/her symptoms. Therefore, it is necessary, upon initiation of therapy, to create this connection, motivating the patient through encouragement each time he/she improves bioinformative performance.

b) Reinforcement programs: To acquire and maintain performance control, it is necessary to develop a structured effort distribution system. Initially, continuous feedback will be applied only when the proposed goal is achieved. Once a patient acquires performance control, intermittent reinforcement is applied, i.e. feedback is produced when the patient has emitted several responses, not after each one. Gradually more responses will be required to generate a feedback response. The goal is to achieve permanent behavior modification without use of the biofeedback device.

c) Modeled: Consists of successive response stimuli that become more similar to the desired outcome. Thresholds are established for the patient to try to achieve.

d) Generalization: logical extension of the modeled program; consists of stimulus transfer to activities different from the trained activity. Most frequently the patient extends behavioural control from the treatment room to activities of daily living, first through use of a portable biofeedback device, and finally without it. Another option is that the change in activity in the working muscle be extended to nearby muscles. In this case global function improves significantly.

Utilization of mental imagery

To achieve modification of neurological impairment and to improve the patient's symptoms, it is necessary to stimulate prior brain activation. In other words, the patient is asked to try to imagine the activity to be performed. This process preactivates nearly all cerebral areas that will intervene during the actual performance of the activity. In fact, this previous step generates changes in electromyographic activity.

The process of creating a mental image of the activity involves three fundamental processes:

a) Creation of a visual image: we ask the patient to imagine a situation (e.g. lying on the beach).

b) Creation of a somesthetic image: we ask the patient to imagine sensations that are produced in the given situation (e.g. imagine the heat from the sun at the beach).

c) Creation of a motor image: we ask the patient to imagine a movement (e.g. stretch your arms).

According to the type of problem, we use different strategies with the patient:

a) Imagine a movement of a specific joint in the affected area.

b) First imagine a movement on the unaffected side.

c) Imagine sensations on the affected side.

d) Through visual-somesthetic (virtual or pure motor imagery) transformation (fig. 3), the patient is asked to imagine a complex gesture as presented in the photograph (production) or to describe that gesture (decodification).

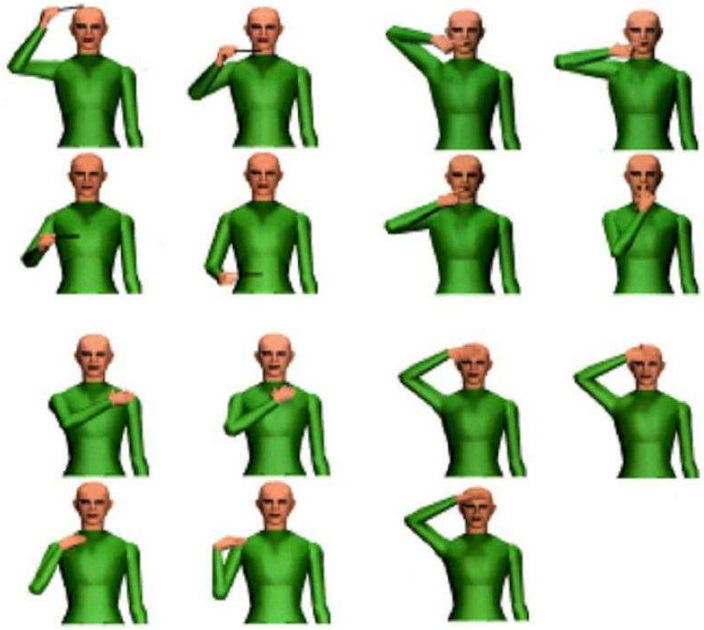


Fig. 3 Transformations

APPLICATION PRINCIPLES IN HEMIPLEGIA

Hemiplegia is the alteration of motor response to the environment, caused by a brain lesion characterized by dysfunction in tone on one side of the body, thus eliminating or reducing the ability to perform normal movement.

Patients present different deficits depending on the degree of neurological damage. Motor dysfunction appears in different areas and to varying degrees. Moreover, if the left cerebral hemisphere is affected, this commonly produces language deficits and, if more extensive, cognitive deficits (attention, memory, connection to surroundings, etc.). These factors make use of electromyographic biofeedback difficult.

Three types of alterations generally occur with acquired brain damage: sensory deficit, muscle hypotonia and muscle hypertonia.

Sensory deficit

Diminished sensory feedback produces, primarily, a reduction in the return of information to the muscular system (fig. 4).

Fig.4 Diagram of diminished transmission capacity of sensory information

Electromyographic biofeedback treatment consists of complementing the altered sensory channel by increasing the diminished capacity exteroceptively (fig. 5).

Fig. 5 Diagram of treatment of diminished sensory information

Muscle hypotonia

The cause of hypotonia is primarily due to a decrease or inactivity in sensory channel feedback, leading to a reduction in motor activity (fig. 6).

Fig.6 Diagram of muscle hypotonia

Through electromyographic biofeedback, the subject detects motor activity by observing a signal. By reinforcing sensory activity, motor activity also increases (fig. 7).

Fig.7 Diagram of treatment of muscle hypotonia

Muscle hypertonia

Muscle hypertonia, including its maximum state (spasticity-rigidity), produces hyperactivity in motor and sensory tracts, resulting in the following vicious cycle (fig. 8):

Fig. 8 Diagram of muscle hypertonia

Electromyographic biofeedback can influence sensory activity by way of negative feedback. The patient becomes less reactive to stimuli, muscle hyperactivity is reduced, and neuromuscular control improves (fig. 9).

Fig. 9 Diagram of treatment of muscle hypertonia

In relation to treatment with electromyographic biofeedback, we will focus on hypotonia and hypertonia. Hypotonia is expressed as a reduction in the use of the neuromuscular system, while hypertonia is expressed as muscle hyperactivity at rest or in relation to difficulty with coordinated movement.

The goal of biofeedback is to detect residual function and show the patient how to recognize and develop that function. In the most common cases, in which the primary problem is hypertonia, the goal of treatment is to reduce hypertonia and, in turn, facilitate movement.

CASE STUDY

Patient diagnosed with left cerebrovascular accident of unknown etiology, resulting in right hemiparesis. The episode occurred on February 7, 2009.

The following activity of daily living (ADL) was evaluated: patient reaching for a glass of water and bringing it toward the mouth to drink, then returning the glass to the table. The activity is performed in sitting, with the glass 1/3 full to allow for the addition of weight. The glass is placed 60 cm front the patient's right upper extremity.

The duration of the activity is approximately 18 seconds with the following breakdown: phase 1, reach and grasp the glass (approximately 5 seconds); phase 2, bring the glass to the lips (approximately 8 seconds); phase 3, return the glass to the table (approximately 5 seconds).

In the visual evaluation, the patient is observed to advance the right arm through the following range of motion: shoulder, approximately 50º abduction and 90º internal rotation; elbow, approximately 90º flexion, in pronation; wrist, slight extension and radial deviation; fingers, semi-flexed with slight ulnar deviation. At the level of the shoulder, the upper trapezius muscle is observed to activate immediately, with shoulder elevation and upward rotation of the scapula, without stabilization by the lower trapezius muscle fibres. Due to weakness of the anterior deltoid muscle (insufficient to elevate the arm), the patient demonstrates compensatory motion of left lateral flexion of the trunk and head, limiting glenohumeral motion necessary to perform this activity efficiently.

The first phase (reach and elevation of the glass approximately 10 cm above the table, first 4-5 seconds) is selected for evaluation and treatment since this is the phase in which the most dramatic substitutions take place (fig 10).

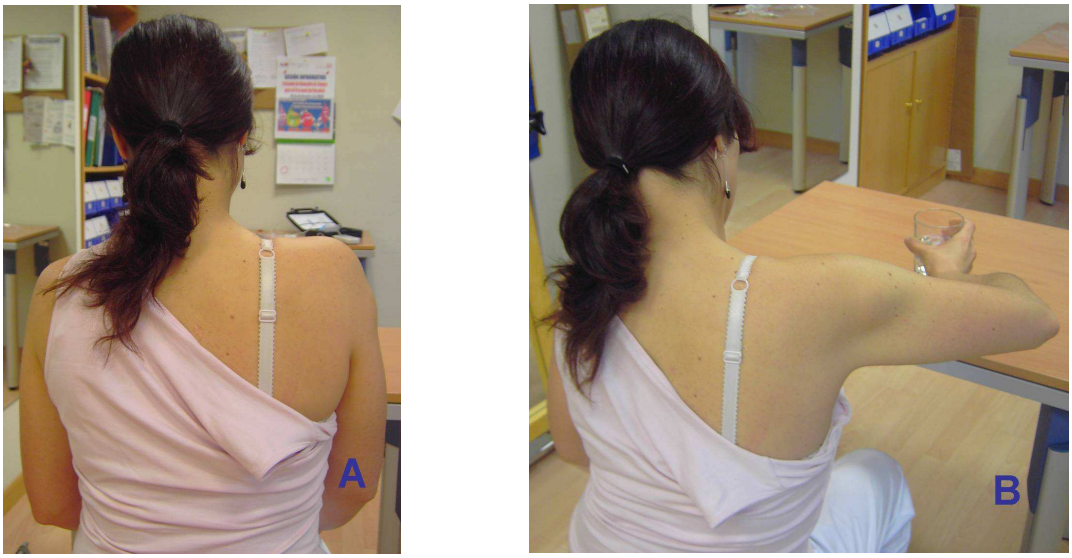


Fig. 10 Sequence of start (A) and end (B) of the first phase

Initial electormyographic evaluation

To conduct this present study, the electromyographic biofeedback device "Neurotrac ETS" was used, along with its corresponding software (version 3.01). The electrodes in fig. 11, along with their corresponding adapters, were used to perform the electromyographic evaluation, while during treatment sessions gel electrodes were used, cut to approximately 2.5 cm x 2.5 cm.

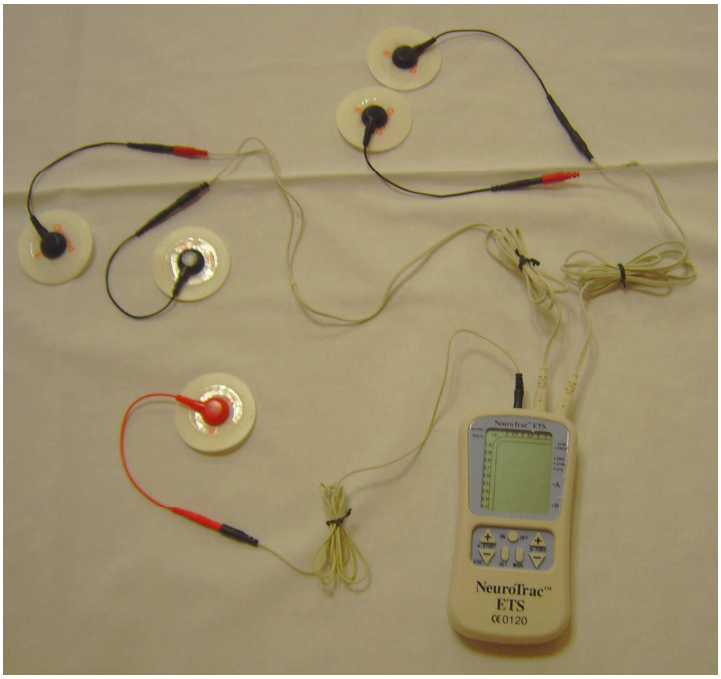


Fig. 11 Neurotrac ETS and electrodes with adapters

We chose to monitor EMG activity of the right arm muscles involved in the first phase of activity: upper trapezius (excessively active from initiation) on channel B and lower trapezius (insufficient to stabilize the scapula) on channel A. The electrodes are placed according to criteria established by S.E.N.I.A.M. (Surface Electromyography for the Non-Invasive Assessment of Muscles): as close as possible to each other, in a line connecting T8 with the trigonum spinea (lower trapezius) and C7 with the acromion (upper trapezius); the reference electrode is placed on the left clavicle (fig. 12).

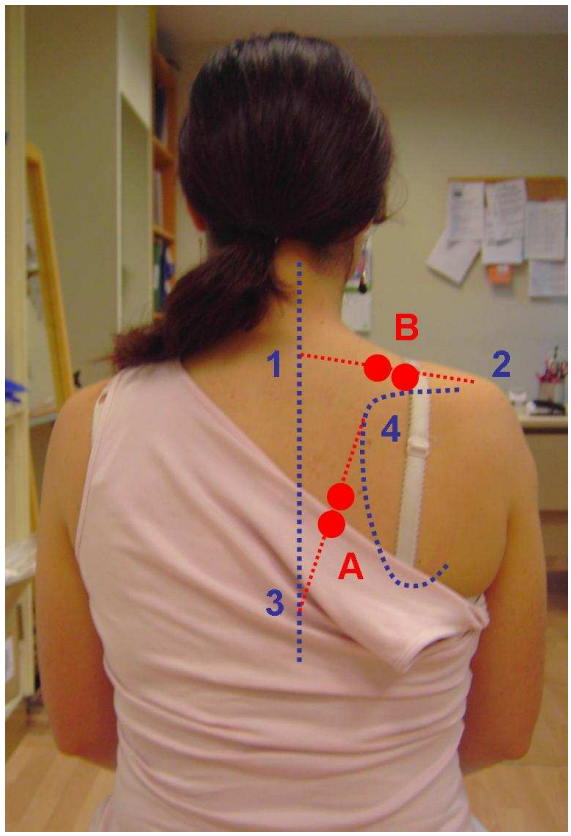


Fig. 12 Application of electrodes: lower trapezius (A) and upper trapezius (B)
1 = C7, 2 = acromion, 3 = T8, 4 = trigonum spinea

The following parameters were analyzed (fig. 13):

a) Average value at baseline: Represents the average value of activity, in µV, of the muscle at rest; in a healthy muscle it is approximately -10 µV.

b) Average threshold value: Represents average value of the best quality contraction intensities.

c) Dispersion: Represents the appearance of oscillations during contraction intensity. An increase indicates muscle fatigue and the need to stop the activity. To calculate dispersion, the maximum and minimum peaks of the best quality contraction are measured (disparity of dispersion is not excessive).

d) Phasic activity: Appears upon performing a sudden and intense contraction - spiked curve of approximately 1 second duration in a healthy muscle.

e) Tonic activity: Appears in the majority of activities of daily living (stable plateau curve).

f) Agonist-antagonist correlation: Represents the proportion between contraction intensity of a muscle and the activity of its antagonist. Normal value is 3, meaning that the intensity of the agonist contraction is three times greater than that of the antagonist.

g) End Plate Potential (E.P.P.): The point of contraction initiation that must coincide with the moment in which the command to perform an activity is given. In patients with neurological impairment, this moment is usually delayed, resulting in delayed onset of muscle contraction.

h) Electro-Mechanical Delay (E.M.D.): The velocity in which the muscle activates and deactivates. This velocity is normally very high, resulting in vertical ascending and descending curves.

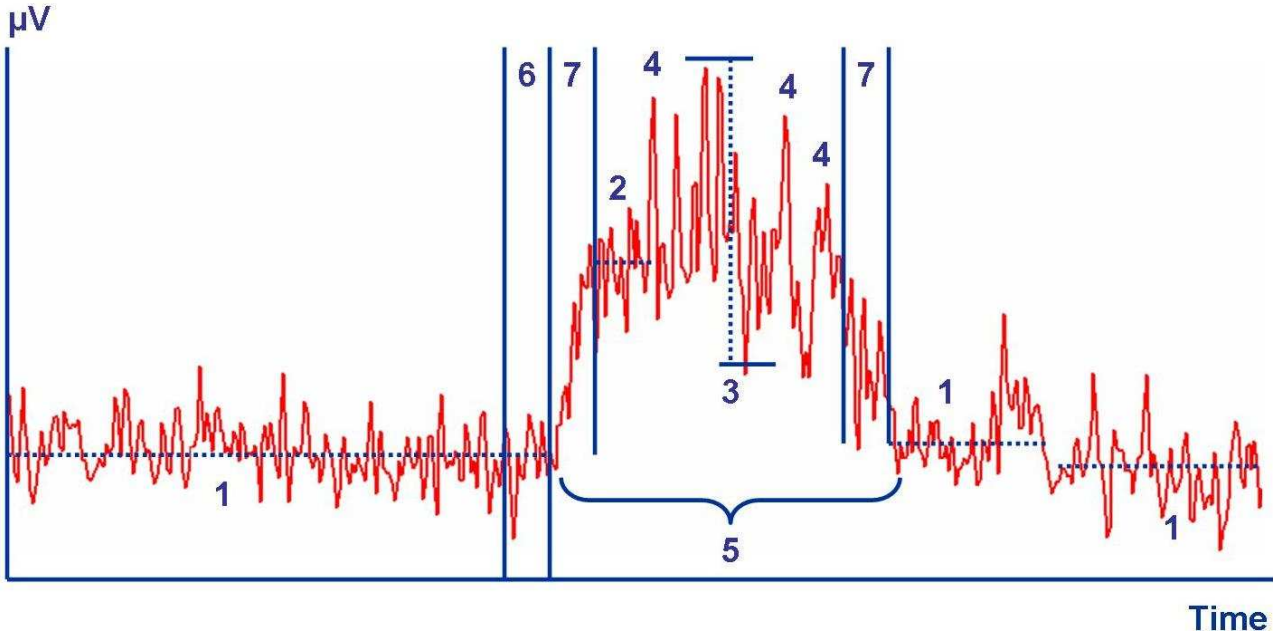
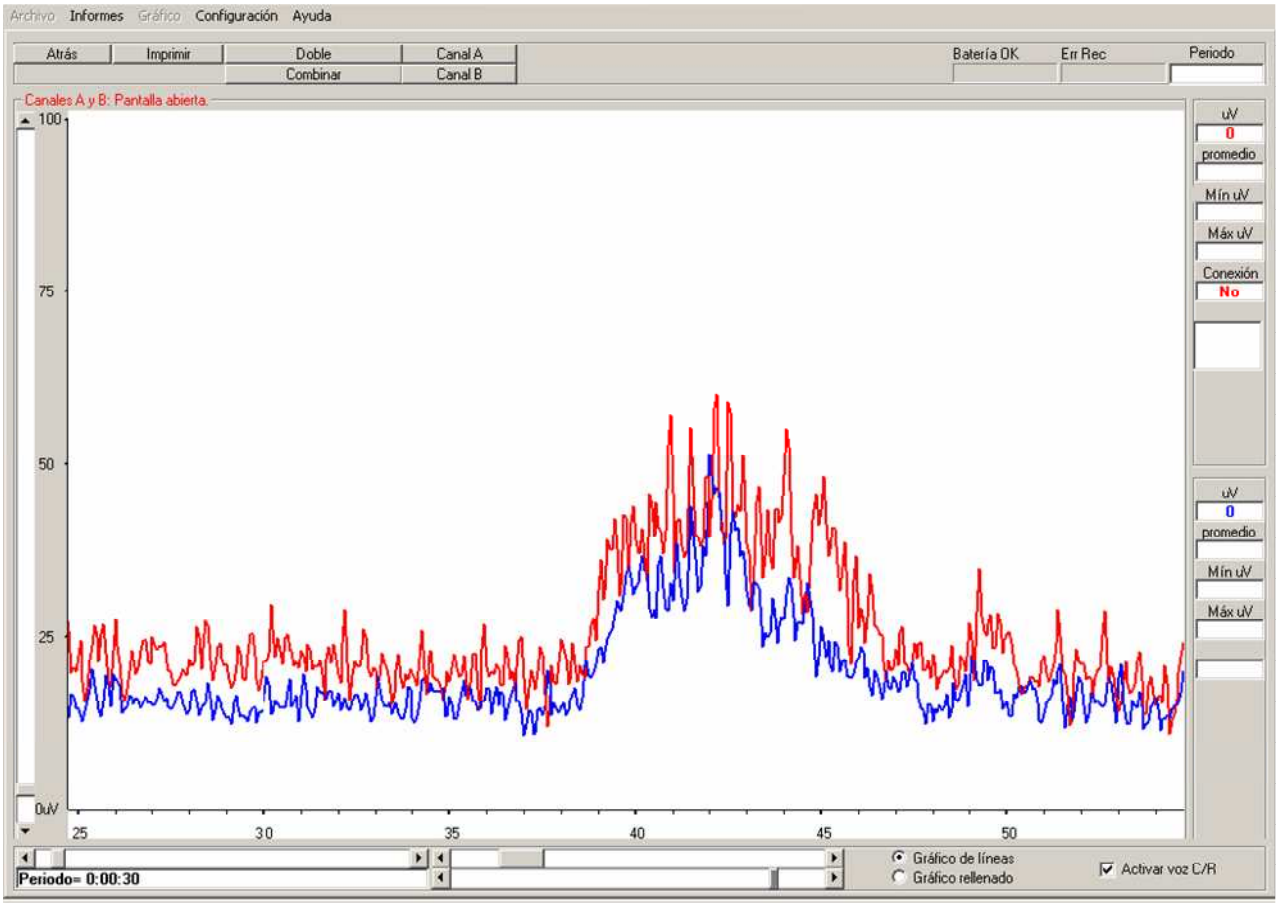


Fig. 13 Parameters analyzed: 1 baseline, 2 best quality threshold value, 3 dispersion, 4 phasic peaks, 5 tonic activity, 6 E.P.P., 7 E.M.D.

During the test, the patient is asked to breathe deeply. Normally, this should not excessively modify the reading. Next, the patient is asked to perform the activity several times until a significant dispersion is observed. This indicates muscle fatigue. With this test we determine, aside from the previously mentioned parameters, the optimal number of repetitions without dispersion, as well as the optimal rest time to restore baseline.

In the resulting test, as at baseline, no alterations are observed with respiratory synergy, and E.P.P. duration is minimal in all repetitions. Average baseline value of the lower trapezius muscle is 24 µV with dispersion of 20 µV (83% of average threshold value); average baseline value of the upper trapezius muscle is 15 µV with dispersion of 18 µV (120% of average threshold value). Tonic phase E.M.D. is initially 1.5 seconds, increasing at the end of the activity, with difficulties in relaxing both muscles. In the lower trapezius, the best quality tonic phase lasts 4 seconds, with subsequent addition of dispersion, average value increases from 39 to 43 µV as muscle contraction increases, with an average value of 41mV and dispersion of 29 µV (70% of average threshold). In the upper trapezius, dispersion is generally greater throughout the activity, with phasic peaks in the middle period, followed by decreasing activity due to fatigue, during the first 4 seconds the average value is 34 µV and the dispersion is 28 µV (82% of average threshold). The correlation factor between both muscles during the activity is 1.2. After the third repetition, dispersion increases in both muscles due to fatigue (with sharp phasic peaks in upper trapezius). Additionally, increases in E.M.D. are observed to initiate the activity, as well as to complete it and reduce the resting baseline values, with increasing problems in the final phase. Average threshold value remains more or less constant. Interphase baseline decreases progressively, from 24 µV to 12 µV in lower trapezius, and from 15 µV to 11 µV in upper trapezius, with a correlation factor that decreases to nearly 1 (fig. 14).



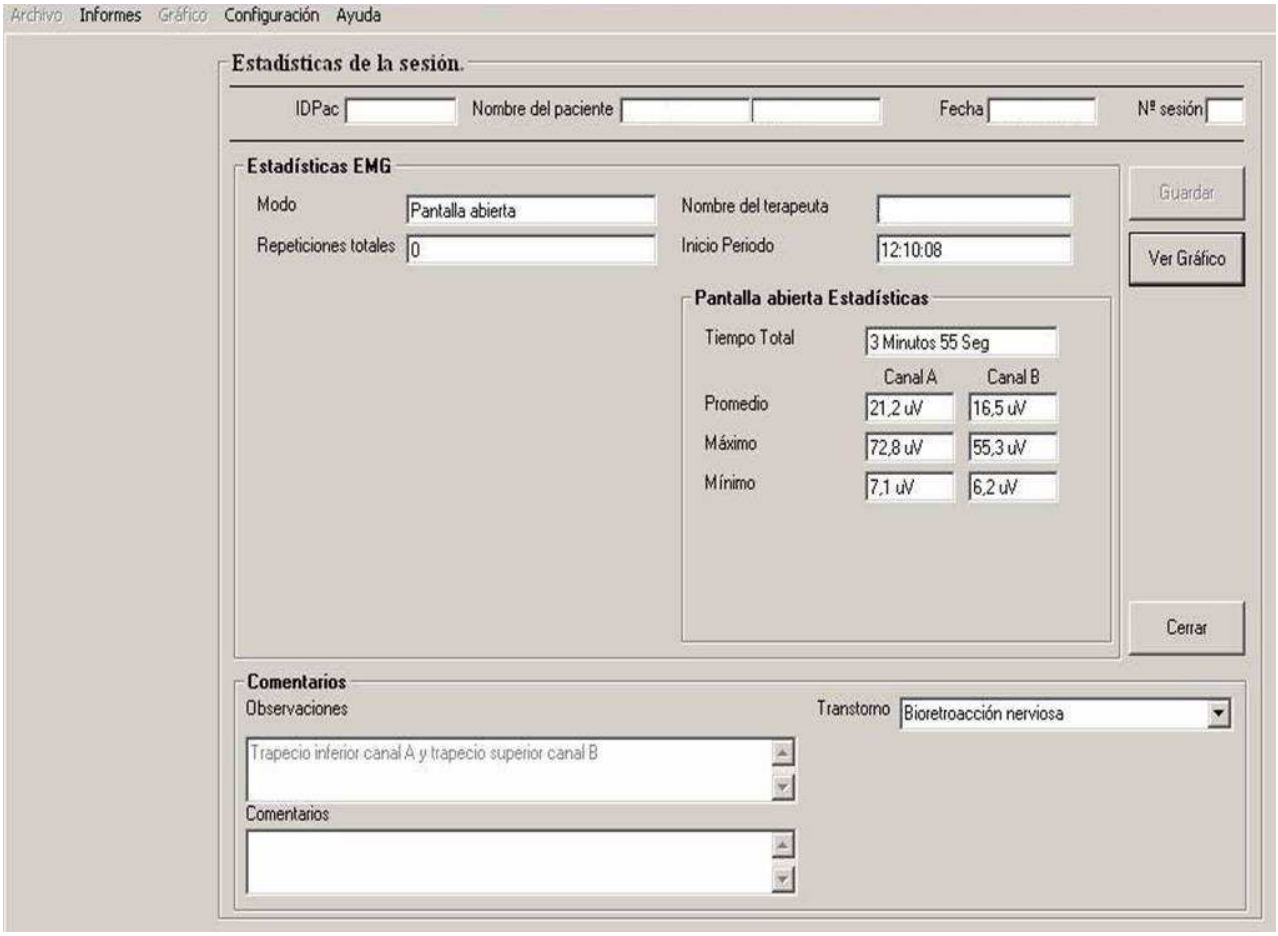


Fig. 14 E.M.G. values of upper trapezius (channel B blue) and lower trapezius (channel A red) Graph and statistics of the treatment session

Treatment Method

The initial focus will be on improving scapular stability during the activity. This requires inhibitory control of upper trapezius (the reason for monitoring this muscle on channel B) and improved activity of lower trapezius (monitored on channel A). The goal is to improve trunk alignment and facilitate deltoid muscle activation during the activity.

To achieve this objective, the subject will be trained in the first phase of the described activity with E.M.G. control of the upper and lower trapezii. In the upper trapezius, audible inhibitory feedback will be set at a threshold of 32 µV. Given the correlation factor of 1.2, upper trapezius will be measured in E.T.S. mode, with activation of the STIM phase at a threshold of 41 µV 1s delay and 3s activation. Activation time for both muscles will be set at 4s (the period of reaching and sustaining the glass above the table). The resting phase consists of the time to return the glass to the table (5s), and the interphase period (40s). Therefore, the total time of the resting phase is 45s. Two repetitions will be performed since, as previously described, fatigue causes rapid reduction in the quality of performance.

Programmed parameters of STIM mode for the lower trapezius (channel A) are as follows:

- RATE 40 Hz (mixed tonic fibers).
- WDTH 450 μs pulse width.
- WORK 3s (duration of the activity “pick up the glass and hold it”).
- REST 45s (5s to return the glass to the table and 40s rest).
- MINS 002, to allow time for 2 repetitions.
- RAMP 9.9 since it is a functional activity requiring a relatively long period of time to perform.
- OFF CHB since we will not stimulate upper trapezius.

Programmed parameters of ETS mode are as follows:

- VOL 10 to summate auditory feedback.
- WORK 4s (estimated duration of the activity “pick up the glass and hold it”).
- REST 45s (compatible with STIM mode).
- TRLS 02, i.e., two repetitions, as in SITM mode.
- ABV FDBK to activate electrostimulation in lower trapezius upon surpassing the threshold of 41 μV.
- ON SDAT to work with the computer in the mode “Controlled by Neurotrac”.
- OFF AUTO, since threshold is adjusted manually; once the patient is trained, ON AUTO mode would be used to practice at home.
- ELDT 01 to activate STIM mode in the lower trapezius as soon as the 41 μV threshold is reached (minimum delay time is 1s).
- NRW FLTA, narrowband in channel A.
- ON CHB to activate channel B, upper trapezius.
- NRW FLTB, narrowband in channel B.
- ON FBIH, to activate inhibitory feedback in upper trapezius (channel B) since the objective is to maintain low tone during the performance of the activity.
- CBTH 032 μV, inhibitory threshold in channel B, to maintain low activity in upper trapezius.
- THRS 41 μV, activation threshold of the STIM phase in the lower trapezius (channel A).

In the ETS mode, the threshold value needed to activate STIM mode is modified manually in each session, to adjust to the activity presented by the patient. For home use, adjustments are made in automatic mode so that the “Neurotrac” itself adjusts the threshold according to the variations produced in the E.M.G. reading (Fig. 15). With respect to electrostimulation intensity, value is adjusted manually at the start of each session. (a minimum of 37-40 mA is necessary to provoke a frank contraction of the lower trapezius).

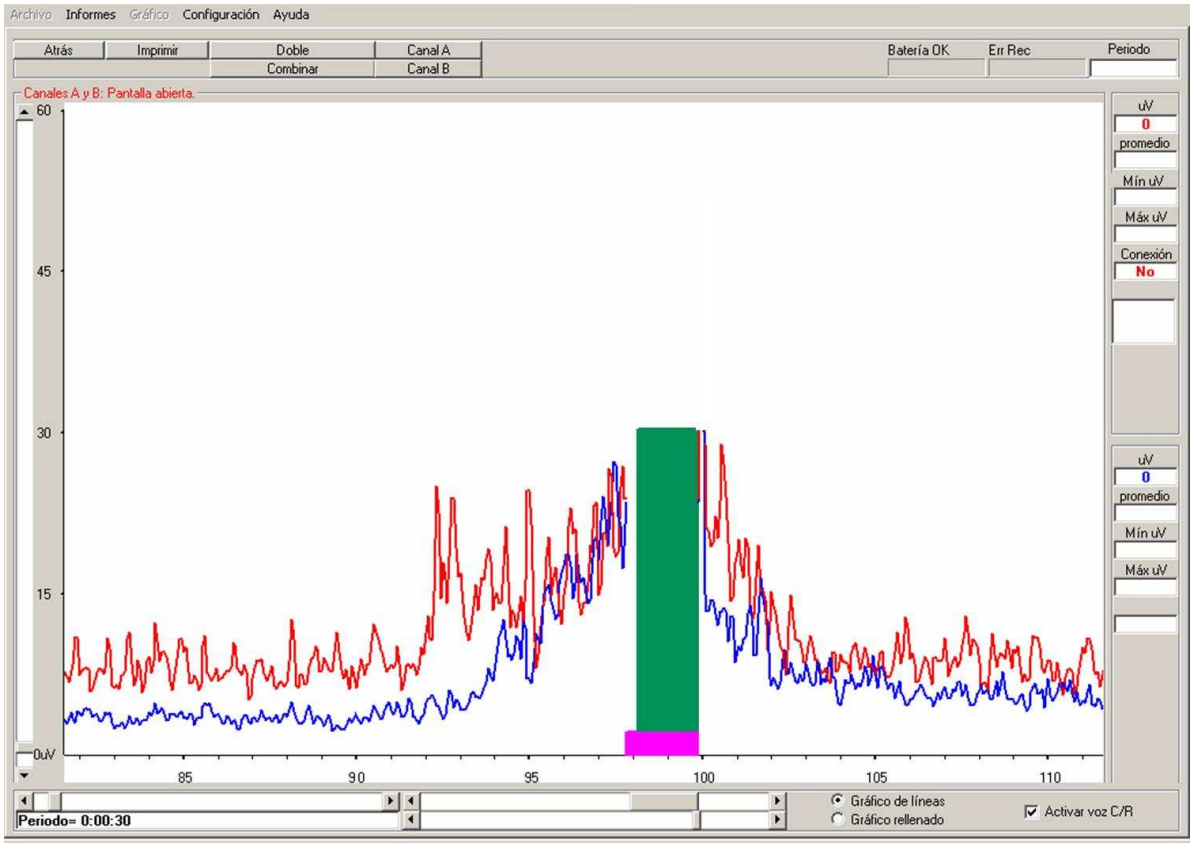


Fig. 15 Example of ETS mode

Prior to a B.F.B. session, descending and posterior movements of the right shoulder will be trained. This activity requires precise activation of the lower trapezius and low tone in the upper trapezius. To achieve this, the PNF pattern of scapular posterior depression will be used (fig. 16). The technique begins with free rhythmic initiation (one set of 3 repetitions), followed by a set of 3 repetitions with resistance, and ends with the activity of "pick up the glass from the table" (3 repetitions) with prior lower trapezius activation. The scapula must be maintained in posterior depression throughout the activity.

Fig. 16 PNF pattern of scapular posterior depression
1 initial position, 2 ending position

The treatment plan will consist of two B.F.B. sessions, preceded by the training described in the previous paragraph, with resting time of 10 minutes between each session. The treatment will be applied over a 4 day period.

Final electromyographic evaluation

In the final evaluation, as at baseline, no alterations are observed in respiratory synergy and E.P.P. duration. Average baseline value of lower trapezius, decreased to only 7 µV, and dispersion to 7 µV (100% of average threshold value). Average baseline value of upper trapezius also decreased to only 3 µV, with dispersion of only 2 µV (66% above average threshold value). E.M.D. decreased to less than 1s at the onset of the activity, although it lengthened at the end of the activity, with difficulty in relaxing both muscles. In lower trapezius, the best quality tonic phase was observed to last 2-3s. With subsequent increase in dispersion, average value decreased 21 µV and dispersion to 9 µV (42% of average threshold value). In upper trapezius, dispersion throughout the activity is generally higher, with persisting phasic peaks in the middle period. However, upper trapezius did not fatigue and maintained activation throughout the activity. During the first phase, its value was only 17 µV and dispersion 8 µV (47% of average threshold value). Correlation factor between both muscles remained at 1.2. In subsequent repetitions, values remained constant, with an increase in dispersion E.M.D. in the fourth repetition (Fig. 17).

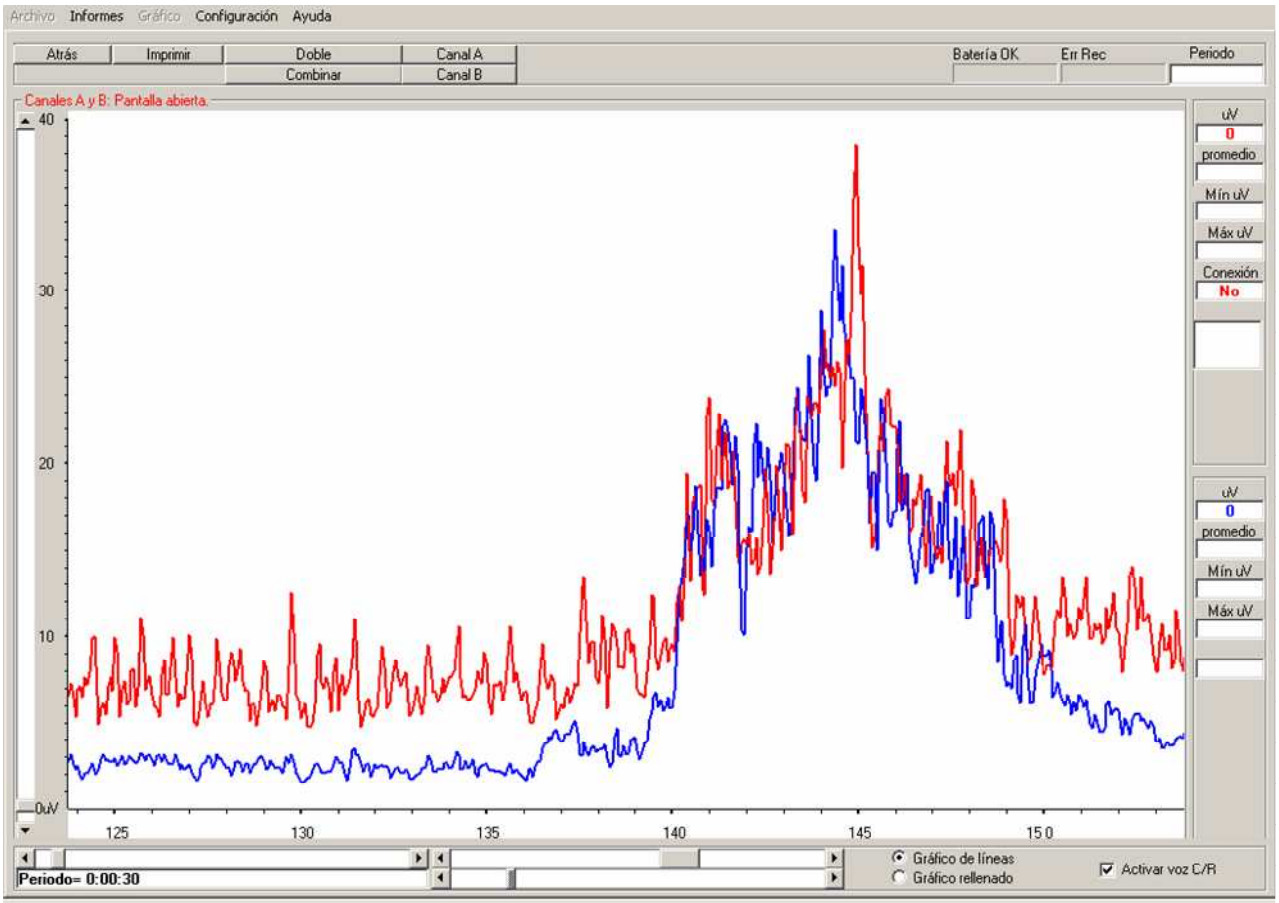


Fig. 17 E.M.G. values of upper trapezius (channel B blue) and lower trapezius (channel A red) Graph and statistics of the session

RESULTS

Upon analysis of the E.M.G. reading, it can be concluded that in only four treatment sessions, significant improvement of muscle activation occurred during the activity of “pick up to glass from the table”.

In general, activation of both muscles during the activity decreased 50%, while at rest activation this reduction reached 71% in lower trapezius and 80% in upper trapezius. At the same time, dispersion decreased in intensity 70% for both muscles during the activity; while at rest the reduction was 65% in lower trapezius and up to 90% in upper trapezius. However, in percentage values, dispersion did not suffer large modifications. The reading is similar in both the initial and final phases of the activity. Nevertheless, dispersion decreased 30% in both muscles during the activity and, although lower trapezius increased discretely at rest (probably secondary to stimulation), upper trapezius decreased 40%. Also, E.M.D. activation value decreased from 1.5s to less than 1s, while correlation factor remained constant at 1.2 (Fig. 18).

LOWER TRAPEZIUS						
	INITIAL	DISPERSION			FINAL	DISPERSION	
B. LINE	24	20	83%		7	7	100%
THRS.	41	29	70%		21	9	42%

UPPER TRAPEZIUS						
	INITIAL	DISPERSION			FINAL	DISPERSION	
B. LINE	15	18	120%		3	2	66%
THRS.	34	28	82%		17	8	47%

E.M.D.	
INITIAL	FINAL
1,5	1

CORRELATION F.	
INITIAL	FINAL
1,2	1,2

Fig. 18 Results

CONCLUSION

As previously mentioned, only four sessions were needed to reduce muscle activity intensity and, as a result, the muscle energy expenditure during the activity. At the same time, baseline muscle tone decreased in both muscles, especially significant in upper trapezius. All this demonstrates that using E.M.G. biofeedback rapidly reduces the level of muscle activity, particularly in upper trapezius, which initially interfered with the activity. Moreover, E.M.D. reduction also demonstrates faster recruitment of motor units and improved response speed.

On the other hand, even though it decreased, dispersion, in percentage values, remains high. Correlation factor also did not increase, which would be preferred. This indicates that more treatment sessions would be necessary to modify these parameters.

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