Rehabilitation Strategies for Facial Nerve Injuries

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ABSTRACT

Many treatment techniques, including exercise, electrical stimulation, biofeedback, and neuromuscular retraining, have been described for the treatment of patients with facial paresis. The degree of nerve injury determines the recovery of the facial muscles. Patients with a Sunderland third-degree injury benefit most from therapy to maximize facial nerve function. Following a facial nerve palsy, many patients present with facial muscle weakness in addition to aberrant synkinetic movements. Therefore therapy must be directed toward control of voluntary movement and decreasing synkinesis. Neuromuscular reeducation involves selective muscle control to decrease synkinesis and increase muscle excursion. Muscle reeducation using surface electromyographic (EMG) biofeedback and home exercises has been shown to be efficacious in the treatment of facial palsies. Neuromuscular retraining can be beneficial in maximizing facial recovery by initially decreasing aberrant synkinetic muscle activity and then increasing voluntary movement and excursion.

KEYWORDS: Facial nerve injury, rehabilitation, neuromuscular reeducation

Injury to the facial nerve may result in motor dysfunction that can have profound social and psychological consequences.^{1,2} Initially, patients present with a unilateral flaccid facial appearance and the majority of patients have recovery of facial nerve function. However, the patients with partial recovery following a facial nerve palsy present with a broad spectrum of motor recovery.

With the loss of facial nerve function, the muscles of the face undergo degeneration and there are subsequent cortical changes with the loss of neural input. With reinnervation of the facial muscles, motor function returns; however, the cortical mapping may not return to the same mapping as before injury.^{3–6} Neuromuscular reeducation is necessary to recruit the appropriate muscles and to ensure satisfactory cortical mapping. Passive therapy programs that include only electrical muscle stimulation and mass facial movements do little to ensure appropriate input to the motor cortex. A rehabilitation program that incorporates the goals of selective muscle control with appropriate cortical input is the most successful in maximizing facial recovery.^{1,7–18}

EVALUATION

Numerous facial grading scales and measurement systems^{13,19–27} have been developed for the evaluation of facial nerve paresis and are described in detail in an accompanying article in this issue. Many of the measurement systems provide detailed analysis of facial nerve function; however, they require additional equipment and time for analysis. Although it may be possible to incorporate these measures in research outcome measures, it is often difficult to use them in an outpatient therapy clinic because of the increased time and

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expensive equipment needed for evaluation. Observational grading systems are more adaptable and accepted in the clinical therapy setting.

The House-Brackmann facial grading system was developed to include both subjective and numeric scales.^{21,22,27} This facial grading system is widely used, although some authors have questioned the sensitivity of the scale to monitor change over time.^{24,28} Burres and Fisch introduced a linear measurement index involving measurement of five facial expressions.²⁰ Although this measurement system is quantitative, it can be tedious and it measures only excursion of the voluntary movement and does not take into account the problem of synkinesis.

The Sunnybrook Facial Nerve Grading System provides an observational rating system to evaluate facial nerve function including resting position, voluntary movement, and synkinesis.²⁴ It has been shown to be a valid and reliable grading system.^{24,29}

To monitor change in facial nerve function, a grading system such as the Sunnybrook Facial Nerve Grading System may be used. Although the grading is sensitive to change, it does not provide specific areas of improvement or movements that continue to impair function. Specific grading of the individual facial muscles with a grading for synkinesis is also necessary to monitor change with therapy.

NONOPERATIVE TREATMENT

Many techniques, including exercise, electrical stimulation, biofeedback and motor reeducation, have been described for the treatment of patients with facial paresis.^{7–12,14–16,18,30} The initial presentation of patients with a complete facial nerve injury is a flaccid facial palsy with no muscle tone, no eye closure, an absent nasolabial fold, and a mouth droop. There is no volitional facial movement with maximal effort. Functionally, this may result in eating, drinking, and speaking difficulties. Depending on the etiology, the onset of facial palsy may be sudden or over the course of several hours.

Recovery following a facial nerve injury depends on the degree of nerve injury. Because a Sunderland firstdegree nerve injury involves axonal demyelination, patients have complete recovery of facial nerve function when the nerve undergoes remyelination.³¹ This recovery may occur as early as 4 weeks but may take as long as 12 weeks following injury. Patients with a Sunderland second- or third-degree nerve injury undergo axonal degeneration and recovery takes longer because nerve regeneration. Recovery with a second-degree injury is complete, although the time for recovery is longer than with a first-degree injury. With a third-degree nerve injury, the endoneurial tubes are not intact and therefore the regenerating axons may not reconnect with the original target muscle. It is with these patients that facial movement recovery is not complete and that neuromuscular reeducation is most beneficial to maximize recovery.

Complete recovery following Bell's palsy has been reported to range from 70 to 96%. 32,33 However, depending on the degree of nerve injury, recovery from a facial nerve injury may span a broad spectrum ranging from no residual deficit to profound muscle weakness and synkinesis. Although facial asymmetry is evident, in the majority of patients with facial nerve dysfunction, weakness of the facial muscles may be only part of the recovery pattern and often the major problem is synkinesis. Synkinesis is defined as an unintentional movement that accompanies a volitional movement. Therefore, patients with synkinesis cannot selectively recruit a specific muscle without an aberrant movement occurring at the same time. If the muscle is antagonistic to the intended motion, decreased excursion occurs in the intended movement. Patients with synkinesis often report eye narrowing or eye closure with mouth movements, for example, recruitment of the orbicularis oris or risorius muscle causing aberrant movement in the orbicularis oculi.

Selection of Patients

Patients presenting with mild to severe facial paresis or weakness may be appropriate for nonoperative treatment. Patients who present with a flaccid facial palsy require education regarding eye care. Because therapy does not accelerate nerve regeneration or muscle reinnervation, there is no need for active physical therapy at this time. Patients are often referred for electrical muscle stimulation. In a denervated muscle, direct current or Galvanic stimulation is necessary to evoke a muscle contraction. The theory behind the use of Galvanic stimulation is to reduce muscle atrophy until muscle reinnervation has occurred. However, there is no evidence such as randomized control trials to support the hypothesis that that electrical muscle stimulation is efficacious in muscle recovery. Once muscle reinnervation has been established, electrical stimulation using an alternating current may be used. However, it is our belief that when muscle reinnervation has occurred, passive stimulation of the muscle does not assist in the neuromuscular retraining that is necessary to maximize facial recovery. With evidence of muscle reinnervation, patients should begin motor retraining to improve motor control and facial muscle movement.

For neuromuscular retraining, patients must have facial muscle reinnervation, as determined by clinical evaluation or electromyography (EMG). The etiology of the facial palsy does not influence the decision for nonoperative treatment. Once muscle reinnervation has occurred, a common problem causing asymmetry is synkinesis and selective muscle recruitment. Although some muscles may be weak or appear weak because of restricted excursion, exercises that encourage mass facial movements and muscle stimulation result in mass action of the facial muscles and not muscle control. This does not increase selective muscle recruitment or decrease synkinesis and therefore does not result in an optimal outcome. Therefore, it is our recommendation that muscle stimulation not be used in patients with facial palsy.

Neuromuscular Reeducation

Neuromuscular reeducation involves selective muscle control to increase muscle excursion and decrease synkinesis. Muscle reeducation using surface EMG biofeedback and a program of home exercises has been shown to be efficacious in the treatment of patients with facial palsies.^{7–12,14–18}

Nakamura et al.¹⁵ reported on 27 patients with complete facial palsy who were randomly divided into either a treatment or a control group. The treatment group patients received retraining exercises to minimize synkinetic eye closure with mouth movements. After 10 months of retraining, there was a significant improvement in synkinetic eye closure with mouth movements.¹⁵ Cronin and Steenerson¹⁴ retrospectively reviewed the charts of 24 patients with facial palsy who were treated with neuromuscular retraining. These patients were compared with six patients who declined facial retraining and received no treatment. The average onset of symptoms was 32 months prior to receiving any facial retraining. Using surface EMG electrodes, the patients were instructed in facial retraining exercises to gain selective muscle control and to decrease synkinesis. The authors concluded that neuromuscular facial retraining was efficacious in improving facial movements. Ross et al.¹⁶ evaluated the efficacy of feedback training in patients with facial nerve paresis using a prospective randomized control trial. All patients (n=25) had long-standing facial nerve paresis and were randomly assigned to mirror exercises or mirror exercises and EMG biofeedback (treatment duration was 1 year). Patients (n=7) who lived a far distance from the treatment facility were the control subjects. After 1 year, there were statistically significant improvements in the symmetry of voluntary facial movement in the patients who had mirror exercises and mirror exercises with EMG biofeedback. Therefore, the evidence supports feedback neuromuscular retraining with a home exercise program as an efficacious treatment for patients following facial nerve injury even with longstanding paresis.

Treatment of Patients

At initial presentation with a facial palsy, patients are instructed in eye care and initial exercises may be directed toward mass facial movements. These exercises may include maximal effort facial movements and cocontractions of the entire face. However, with evidence of synkinesis, mass movements should be discontinued because this increases the amount of synkinesis and muscle tone. Neuromuscular retraining, however, should begin to increase selective voluntary muscle movement.

Patients with facial nerve paresis often have facial movement goals to improve an uneven smile or to decrease eye closure with mouth movements. These goals are difficult to achieve without control of the synkinesis, and therefore an initial primary goal of treatment must be to decrease the synkinesis. To ensure that the patient has a good understanding of the problem and treatment strategies, education of the patient must begin with facial anatomy (facial nerve branching, muscles), nerve injury, and recovery. A good understanding of the basic anatomy and physiology helps the patient to understand the strategies for neuromuscular retraining.

Relaxation training is needed to increase the patient's awareness of the increased facial tone and small stimuli necessary to increase muscle activity.⁷ Patients often sit in head-forward slouching postures with the head tilted toward the involved side of facial paresis. Postural correction should begin to increase awareness of the head position and its effect on facial tone. Patients should also be instructed to avoid gum chewing or excessive chewing on the affected side because stimulation with mass facial movements increases facial tone and thus increases synkinesis. Balliet et al.⁸ also recommend that patients avoid any stimulants such as caffeinated drinks and cigarettes. Bilateral light face-tapping exercises are used to increase sensory stimulation and blood flow to the affected side.^{7,8}

Education is important to instruct the patient on strategies to decrease resting tone. Although increased resting tone is often a problem on the affected side of the face, hyperactivity of the unaffected side may be evident in some patients. Face tapping and light massage may help to increase muscle relaxation.⁷ Surface EMG monitoring can be useful in helping patients to relax the facial muscles and decrease resting muscle tone.^{7,16} A fourchannel EMG unit is used to compare the muscle activity of the affected and unaffected sides of the face.

The initial goal in facial neuromuscular retraining is to eliminate synkinesis and then progress to isolated voluntary movements.^{7,9,16,18} Some patients find it is extremely difficult to eliminate the synkinesis, and these patients would not progress to voluntary movements. VanSwearingen and Brach recommend that it may be more beneficial to have these patients increase the voluntary movement and allow the synkinesis to occur.¹⁸ As the movement increases, the patient can attempt to decrease the synkinetic movements. The decision on timing to begin isolated voluntary movements should be based on individual assessment of the patient and dependent on the patient's response to retraining.

To regain motor control of the facial muscles, it is necessary to regain small controlled movements and progress with increased effort when muscle control is regained.^{7,8} Isolated voluntary movements should begin on the unaffected side of the face. Initially, it is difficult to perform small movements on the unaffected side and therefore mirror exercises or biofeedback training is essential to learn this movement. With success of small facial movements on the unaffected side, the movement is initiated on the affected side. The isolated movement is performed to the extent that there is no simultaneous synkinetic movement and the movement appears symmetrical with the contralateral side. Slow onset of muscle activity is encouraged because fast facial movements promote improper muscle recruitment and perpetuate synkinesis.

EMG biofeedback can be used to assist the patient in initiating the voluntary movement and to control synkinesis.^{7–9,16} Similarly, observation in a mirror of the movement on the unaffected side can provide visual cues to reproduce the movement on the affected side. Because the reinnervated muscle fatigues quickly, patients are able to perform only a few trials and then a rest is required before resuming the exercise. EMG biofeedback training can also be useful in reducing synkinesis with voluntary movement. One channel is placed on the muscle to increase voluntary control, and the second channel is placed on the area of synkinesis. For example, one channel may be placed on the orbicularis oris to increase active motion and the second channel may be placed on the risorius muscle or orbicularis oculi to control aberrant synkinetic muscle activity. Using this method, the patient can observe the active movement and the synkinetic muscle activity. With a four-channel EMG biofeedback unit, the other two channels can be placed on the contralateral unaffected muscles to observe the desired action. Through visual feedback, the patient can learn to increase the voluntary movement while minimizing the unwanted synkinetic muscle activity. As more control is gained over the synkinesis, increased effort and excursion of the voluntary movement can be achieved.

Although biofeedback is a useful tool for retraining, it is limited in home use because many patients do not have access to a biofeedback unit at home. Therefore, it is important to instruct the patient in a home exercise program using visual feedback with a mirror.^{7,16} Using mirror exercises, the patient can continue with the retraining exercises more frequently at home. Initial retraining exercises should begin with muscles that are easier to isolate and not directly connected to the synkinetic muscle, that is, retraining orbicularis oris and decreasing synkinetic activity in the orbicularis oculi. As the patient is able to recruit the appropriate muscle correctly, more muscles and actions can be incorporated in the home program. Initial retraining is best initiated with EMG biofeedback training because the patient receives accurate and immediate feedback to ensure that the appropriate muscles are being recruited. With assurance that the patient is performing the exercise correctly, exercises with mirror feedback may commence.

Specific exercises to regain eye closure may begin early in the rehabilitation program and do not require EMG biofeedback training.⁷ Target training using a handheld mirror is useful to improve eye closure. The patient is instructed to gaze down into a mirror below the face and attempt to close the eye. The mirror is used to maintain the eye in a downward position to avoid Bell's reflex and to improve the activity of the orbicularis oculi for eye closure. With improvement of eye closure, the target is moved to a more horizontal position.

The initial improvements with therapy occur in the first few months as the patient understands the actions that stimulate and perpetuate the synkinesis and learns to decrease the resting muscle tone. The improvements in selective muscle recruitment and increased excursion require more complex reeducation and progress at a slower rate. With another 6 to 9 months devoted to neuromuscular retraining and an appropriate home exercise program, patients may expect to enhance facial movement and to achieve better facial symmetry.

CONCLUSION

The psychosocial effects of a facial nerve injury can be profound, and even small facial nerve dysfunction can have a dramatic impact on a patient. Therefore it is critical to ensure that facial nerve recovery is maximized and that facial symmetry is achieved for the patient's optimal satisfaction. Neuromuscular reeducation with a comprehensive home exercise program can improve function of the facial muscles by encouraging selective muscle recruitment and decreasing synkinesis.

REFERENCES

- Brach JS, Van Swearingen JM, Delitto A, Johnson PC. Impairment and disability in patients with facial neuromuscular dysfunction. Otolaryngol Head Neck Surg 1997;117: 315–321
- Jugenburg M, Hubley P, Yandell H, Manktelow RT, Zuker RM. Self-esteem in children with facial paralysis: a review of measures. Can J Plast Surg 2001;9:143–146
- Bach-y-Rita P. Brain plasticity as a basis for recovery of function in humans. Neuropsychologia 1990;28:547–554
- Malessy MJ, Thomeer RT, van Dijk JG. Changing central nervous system control following intercostal nerve transfer. J Neurosurg 1998;89:568–574
- Malessy MJ, van der Kamp W, Thomeer RT, van Dijk JG. Cortical excitability of the biceps muscle after intercostal-tomusculocutaneous nerve transfer. Neurosurgery 1998;42: 787–794

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- Merzenich MM, Jenkins WM. Reorganization of cortical representations of the hand following alterations of skin inputs induced by nerve injury, skin island transfers and experience. J Hand Ther 1993;6:89–104
- Balliet R. Facial paralysis and other neuromuscular dysfunctions of the peripheral nervous system. In: Payton OD, ed. Manual of Physical Therapy. New York: Churchill Livingstone; 1989:175–213
- Balliet R, Shinn JB, Bach-y-Rita P. Facial paralysis rehabilitation: retraining selective muscle control. Int Rehabil Med 1982;4:67–74
- Brach JS, Van Swearingen JM, Lennert J, Johnson PC. Facial neuromuscular retraining for oral synkinesis. Plast Reconstr Surg 1997;99:1922–1931
- Brach JS, Van Swearingen JM. Physical therapy for facial paralysis: a tailored treatment approach. Phys Ther 1999;79: 397–404
- Brown DM, Nahai F, Wolf S, Basmajian JV. Electromyographic biofeedback in the reeducation of facial palsy. Am J Phys Med 1978;57:183–190
- Brudny J. Biofeedback in facial paralysis: electromyographic rehabilitation. In: Rubin L, ed. The Paralyzed Face. St. Louis: Mosby; 1991:247–264
- Brudny J, Hammerschlag PE, Cohen NL, Ransohoff J. Electromyographic rehabilitation of facial function and introduction of a facial paralysis grading scale for hypoglossal-facial nerve anastomosis. Laryngoscope 1988;98:405–410
- Cronin GW, Steenerson RL. The effectiveness of neuromuscular facial retraining combined with electromyography in facial paralysis rehabilitation. Otolaryngol Head Neck Surg 2003;128:534–538
- Nakamura K, Toda N, Sakamaki K, Kashima K, Takeda N. Biofeedback rehabilitation for prevention of synkinesis after facial palsy. Otolaryngol Head Neck Surg 2003;128:539–543
- Ross B, Nedzelski JM, McLean JA. Efficacy of feedback training in long-standing facial nerve paresis. Laryngoscope 1991;101:744–750
- Segal B, Zompa I, Danys I, et al. Symmetry and synkinesis during rehabilitation of unilateral facial paralysis. J Otolaryngol 1995;24:143–148
- VanSwearingen JM, Brach JS. Changes in facial movement and synkinesis with facial neuromuscular reeducation. Plast Reconstr Surg 2003;111:2370–2375

- Adour KK, Swanson PJ Jr. Facial paralysis in 403 consecutive patients: emphasis on treatment response in patients with Bell's palsy. Trans Am Acad Ophthalmol 1971;75:1284– 1301
- Burres SA, Fisch U. The comparison of facial grading systems. Arch Otolaryngol Head Neck Surg 1986;112:755– 758
- House JW. Facial nerve grading systems. Laryngoscope 1983; 93:1056–1069
- House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg 1985;93:146–147
- May M. Facial paralysis, peripheral type: a proposed method of reporting (emphasis on diagnosis and prognosis, as well as electrical and chorda tympani nerve testing). Laryngoscope 1970;80:331–390
- Ross BG, Fradet G, Nedzelski JM. Development of a sensitive clinical facial grading system. Otolaryngol Head Neck Surg 1996;114:380–386
- Neely JG, Cheung JY, Wood M, Byers J, Rogerson A. Computerised quantitative dynamic analysis of facial motion in the paralysed and synkinetic face. Am J Otol 1992;13:97– 107
- Neely JG, Joaquin AH, Kohn LA, Cheung JY. Quantitative assessment of the variation within grades of facial paralysis. Laryngoscope 1996;106:438–442
- Brackmann DE, Barrs DM. Assessing recovery of facial function following acoustic neuroma surgery. Otolaryngol Head Neck Surg 1984;92:88–93
- Chee GH, Nedzelski JM. Facial nerve grading systems. Facial Plast Surg 2000;16:315–324
- Kayhan FT, Zurakowski D, Rauch SD. Toronto facial grading system: interobserver reliability. Otolaryngol Head Neck Surg 2000;122:212–215
- Farragher D, Kidd GL, Tallis R. Eutrophic electrical stimulation for Bell's palsy. Clin Rehabil 1987;1:265–271
- Sunderland S. Nerve and Nerve Injuries. Edinburgh: Churchill Livingstone; 1978
- Axelsson S, Lindberg S, Stjernquist-Desatnik A. Outcome of treatment with valacyclovir and prednisone in patients with Bell's palsy. Ann Otol Rhinol Laryngol 2003;112:197–201
- Hato N, Matsumoto S, Kisaki H, et al. Efficacy of early treatment of Bell's palsy with oral acyclovir and prednisone. Otol Neurotol 2003;24:948–951