

Electrical Stimulation as a Therapeutic Option to Improve Eyelid Function in Chronic Facial Nerve Disorders

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PURPOSE. To establish whether it is possible to improve orbicularis oculi muscle function in the eyelids of patients with a chronic seventh cranial nerve palsy by using transcutaneous electrical stimulation to the point at which electrical stimulation induces a functional blink.

METHODS. Ten subjects (one woman, nine men) aged 36 to 76 with chronic, moderate to severe facial nerve palsy were recruited into the study. Voluntary and spontaneous eyelid movements were assessed, using an optical measuring system, before, during, and after a 3-month treatment period. Voluntary and spontaneous lid velocities were also measured and compared with eyelid kinematic data in normal subjects (12 women, 18 men; age range, 22-56 years).

RESULTS. Therapeutic electrical stimulation applied over 3 months produced improvement in eyelid movement (>2 mm) in 8 of 10 patients during voluntary eyelid closure. However, there was no significant improvement recorded in spontaneous blink amplitudes or peak downward-phase velocity of the upper eyelid. This regimen of stimulation failed to recover function well enough that a functional blink could be induced in the paretic eyelid by electrical stimulation.

CONCLUSIONS. Electrical stimulation using transcutaneous electrical nerve stimulators units can improve voluntary eye closure, apparently because of a reduction in stiffness of eyelid mechanics, rather than an improvement of muscle function. Investigation of alternative stimulation regimens is warranted. (*Invest Ophthalmol Vis Sci.* 1999;40:547-554)

Seventh cranial nerve paralysis arises from a variety of causes involving upper or lower motor neuron lesions. The prognosis depends on the type of lesion. Lower motor neuron problems arising from viral disorders or Bell's palsy can show spontaneous recovery, although there may be some residual weakness. A lower motor neuron lesion arising from surgery or head injury can result in chronic paralysis.

Seventh nerve damage affects all the muscles of facial expression. Clinically, the most important consequence is paralysis of the upper eyelid on the affected side. This results in deficient wetting of the ocular surface, leading to corneal drying and ulceration, which could impair vision. Corneal protection measures range from lubricating ointments, (which can themselves blur vision) to a variety of surgical procedures, such as tarsorrhaphy and lid-tightening. Procedures intended to improve dynamic eyelid function include nerve crossover and muscle graft,¹ whereas eyelid closure can be improved using gold weights.²

Temporary restoration of eyelid function using electrical stimulation has been shown in animal models. Electrical stimulation applied by implanted electrodes was used to maintain functional eyelid movement for 70 days after denervation in dogs.³ It has also been shown that a symmetrical blink can be achieved in dogs with a unilateral facial paralysis by triggering a stimulator with the electromyograph signal from the unaffected side.⁴ This experiment was performed 3 months after denervation, but no indication was given of the long-term effectiveness. An implanted stimulator was used in rabbits for up to 5 months after denervation to maintain eyelid function.⁵ These studies showed that functional electrical stimulation by implanted electrodes can induce a blink in acute peripheral denervation. However, they do not indicate whether electrical stimulation can restore eyelid movement in cases of chronic paralysis when the effects of muscle atrophy must be overcome to produce functional movement.

Many muscle fiber properties are sustained and regulated by neural activity, leading to suggestions of a trophic or nourishing effect of the nerve on the muscle fiber.⁶ Fiber properties are also maintained by activity. Inactivity in normally innervated muscle leads to atrophic changes that can be reversed by electrical stimulation therapy.⁷ Chronic peripheral denervation results in atrophy of muscle fibers and changes to the resting fiber membrane potential, to acetylcholine sensitivity, and to the structure of the contractile apparatus.⁸ Some of these effects occur almost immediately, whereas others occur slowly over several weeks, and even months. The changes due to

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peripheral denervation are more difficult to reverse, and the application of electrical stimulation therapy has produced conflicting results.^{9,10} Comparisons between studies are often difficult to make because of the different animal models and stimulation protocols used.¹⁰

There is evidence to suggest that denervated facial muscles may respond to electrical stimulation. Facial muscle fibers have been observed 20 to 30 years after denervation.⁵ The facial muscles of expression are predominantly fast twitch¹¹ and should respond more readily to electrical stimulation than denervated slow-twitch muscle.¹² Also, electrical stimulation has a greater effect on small than on large muscle groups.¹³ Therapeutic electrical stimulation has already been used with some success in Bell's palsy,^{14,15} although 84% of patients with Bell's palsy recover spontaneously without treatment.¹⁶

Our study was designed to investigate the effects of transcutaneous therapeutic electrical stimulation on eyelid function in patients with a chronic lower motor neuron lesion. If such a regimen can cause functional movement, it may be possible to design an implantable system to stimulate blink movements in affected eyelids.

METHODS

Subject Recruitment

Ten patients (9 men, 1 woman) with chronic palsy in the seventh cranial nerve were recruited into the study. Their mean age was 57 years (range, 36–76 years). The small number of subjects available made a prospective study difficult. However, selection of subjects with a chronic condition (defined as lasting at least 12 months with no significant recovery) reduced the possibility that the improvements made during the treatment period occurred by chance. Eight patients had had acoustic neuroma resection with grade IV facial palsy (grades are according to the scale of House and Brackmann¹⁷) or worse. One patient had facial palsy grade III as a result of a traffic accident. One subject had grade IV facial palsy associated with Ramsay Hunt's syndrome. Two subjects had undergone tarsorrhaphy. The mean time from nerve injury to treatment in nine patients was 24 months (range, 12–48 months). The 10th patient had undergone denervation for 144 months. Ethics committee approval was obtained to measure the responses of volunteers with facial palsy to courses of treatment with standard transcutaneous electrical nerve stimulator units. Informed written consent was obtained, in accordance with the tenets of the Declaration of Helsinki.

To establish normal data for the eyelid assessment methods, 30 normal volunteers (12 women, 18 men) with a mean age of 31 years (range, 22–56 years) were recruited from within our department. Although the mean age and range of normal subjects in this study are lower than those of the patient population, age-related changes to the blink main sequence (see later discussion) have been found to be significant only in people more than 80 years of age.¹⁸

Measurement of Eyelid Kinematics

In normal blinks, there is an approximately linear relationship between the V_{max} of the eyelid and the peak blink amplitude.^{19,20} This relationship, commonly termed the main sequence, is widely used to characterize eyelid function. We used a line-scan imaging system for the measurement of the

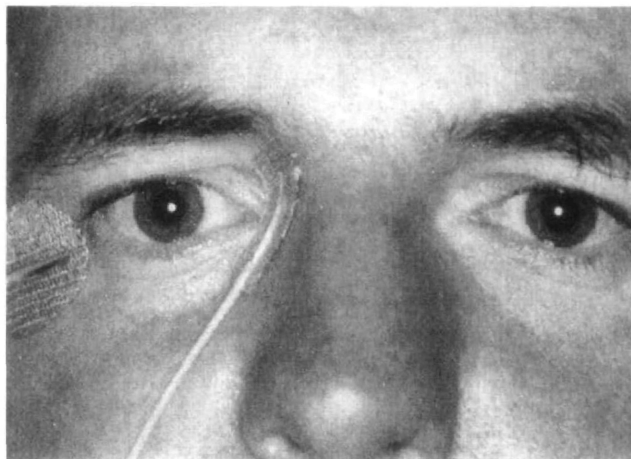


FIGURE 1. The position of the stimulating electrodes on the medial and lateral canthi.

main sequence in normal subjects and in those with palsy. The system acquires a sequence of parasagittal reflectivity profiles of the center of the eye from which the eyelid margins can be identified. The profiles are then analyzed using software that locates and tracks the eyelid margin automatically through the sequence, providing displacement versus time data. These data are used to derive the variation of velocity with time. The system has a temporal resolution of 5 msec and a spatial resolution of 0.2 mm. The method is noninvasive and is easy to use in a clinical environment.²¹

During data acquisition, the subject's head was stabilized using a chin-head rest that was adjusted to center the eye in the field of the imaging system. Subjects were asked to fixate their gaze onto the center of the lens to ensure reproducibility of the starting position of the eyelid. To minimize variability in blink main-sequence measurements from environmental factors and subject fatigue, recordings were made under fixed illumination conditions and at approximately the same time of day. Temperature and humidity were not controlled but were relatively constant.

Electrical Stimulation

The stimulation regimen was implemented using transcutaneous electrical nerve stimulators (model 120Z; ITO, Tokyo, Japan). The stimulators had a constant voltage output with a compensated monophasic pulse shape. The pulse frequency could be varied between 2 Hz and 200 Hz and the pulse length between 50 μ sec and 200 μ sec. A thumbwheel with a scale of 1 to 10 controlled the peak voltage level.

Patients were instructed to use the electrical stimulator for approximately 1 hour daily. The pulse frequency and width were set to 10 Hz and 200 μ sec, respectively, consistent with values used in previous studies using therapeutic electrical stimulation on seventh nerve palsies.^{14,15} The duty cycle of the treatment was 2 seconds on and 1 second off. One-inch diameter reusable electrodes (PAL; Nidd Valley Microproducts, Knaresborough, UK) were placed as near as possible to the medial and lateral canthi of the eye. In some cases it was necessary to trim the electrodes to fit. The position of the electrodes is shown in Figure 1. The level of skin sensation around the electrode site was assessed to minimize the possibility of adverse skin reactions during the course of the stim-

TABLE 1. Upper Eyelid Displacement Initially and after 3 Months' Treatment during Voluntary Closure

Patient	Age (y)	Palsy Duration (mo)	Grade*	Pretreatment Displacement (mm)	Posttreatment Displacement (mm)	Difference (mm)	P†
1	74	30	III	2.6 ± 0.1	4.3 ± 0.4	1.7	0.018
2	74	24	IV	4.2 ± 0.1	6.6 ± 0.1	1.4	0.005
3	30	12	III	0.5 ± 0.02	4.6 ± 0.03	4.1	0.002
4	67	26	III	3.1 ± 0.4	4.5 ± 2.6	1.4	0.131
5	73	14	IV	2.7 ± 0.05	6.1 ± 0.05	3.4	0.002
6	63	20	V	1.4 ± 0.1	3.38 ± 0.08	2.0	0.003
7	48	144	IV	4.7 ± 0.45	4.7 ± 0.05	0.11	0.442

* According to House Brackmann.¹⁷

† By *t*-test paired for means.

ulation. Patients were instructed to use a level of stimulation consistent with comfort. This was normally in the range of 3 to 4 on the thumbwheel, corresponding to a peak voltage of approximately 25 V. They were asked to record the level of stimulation used and the length of time stimulation was applied.

Method of Assessment

Patients were assessed at the start of their treatment and subsequently at four weekly intervals for 3 months. Eyelid movements were recorded using the line-scan system for spontaneous blinks and voluntary eye closure. For the latter, patients were asked to close the eye as much as possible while maintaining facial symmetry. Each recording was characterized by measuring the peak displacement and V_{max} . For each assessment, an average of three maximum eyelid displacements and associated peak velocities were used. Responses to electrical stimulation were also assessed. A measure of lagophthalmus was made using a ruler to measure the residual palpebral fissure on forced closure.

Facial movements were recorded on videotape before and during treatment. These were examined qualitatively for signs of increased synkinetic movements, although no evidence was seen in any patient during the treatment period. Facial movements were also assessed qualitatively by comparing movements between the normal and affected sides and grading the response on the affected side as a percentage of movement on the normal side. Specific movements made in the comparison included raising the eyebrows, smiling, puffing the cheeks, and baring the teeth. Assessments were made with knowledge of previous assessments.

Normal main-sequence relationships between maximum eyelid velocity and displacement for downward and upward movement were made by recording five consecutive spontaneous blinks from 30 volunteers.

RESULTS

Maximum Eyelid Displacement and Lagophthalmus

Voluntary Eye Closure. As described in the Methods section, eyelid displacement was measured using the line-scan imaging system. Unfortunately, this system was not complete when the first three patients began treatment, and we were unable to make pretreatment displacement assessments in

these patients. However, lagophthalmus measurements were made with a ruler in all 10 patients. Eyelid displacement measurements made with the line-scan imaging system before and after treatment in the remaining seven patients are listed in Table 1. These measurements show that treatment of paretic eyelids using this regimen of electrical stimulation therapy was successful in increasing the range of voluntary movement. Increases in eyelid displacement ranged from 1.4 mm to 4.1 mm with a mean value of 2.5 mm. Time to peak closure ranged from 0.4 to 1.8 seconds, with a mean of 0.9 seconds.

A one-sided paired *t*-test was performed on the eyelid displacement data derived from the line-scan system for each of the seven patients. Four of seven patients showed a significant improvement in the range of movement ($P < 0.005$). Two cases were significant at the 2% level. The observed differences in the seven patients overall were significant at the 5% level. Patient 7, who had undergone tarsorrhaphy, showed no difference before and after treatment. Extending the treatment period beyond 3 months provided no further benefit to any of the patients.

Lagophthalmus. Lagophthalmus measurements were performed with a ruler in all 10 patients over the treatment period and are listed in Table 2. Eight subjects showed a reduction in lagophthalmus (mean, 2.9 mm; range, 0–6 mm) including two subjects who improved enough to show no lagophthalmus. A one-sided *t*-test on the measurements in all 10 patients showed that the observed differences were significant at the 5% level. Patients 7 and 9, who showed no reduction in lagophthalmus after treatment, had undergone tarsorrhaphy.

Spontaneous Eyelid Movement. The maximum recorded spontaneous eyelid displacements before and after treatment in the seven patients for whom the line-scan system was available are shown in Table 3. Measurements in four subjects showed a small increase in displacement (<0.8 mm), whereas those in the other three showed a marginal decrease (<0.5 mm). A one-sided *t*-test on the differences showed that the observed changes were not significant ($P = 0.155$).

Dynamic Eyelid Measurements

Normal Data. The curve for upper eyelid displacement versus time obtained from the line-scan imaging system for a normal spontaneous blink is shown in Figure 2A. The corresponding relationship between velocity and time is shown in Figure 2B. The displacement curve shows rapid closure of the upper lid followed by a longer upward phase in which peak velocities are lower.

TABLE 2. Lagophthalmos Measured Initially and after 3 Months' Treatment

Patient	Age (y)	Palsy Duration (mo)	Grade*	Pretreatment Lagophthalmos (mm)	Posttreatment Lagophthalmos (mm)	Difference (mm)
1	74	30	III	4	2	2
2	74	24	IV	6	2	4
3	30	12	III	11	5	6
4	67	26	III	4	2.5	1.5
5†	73	14	IV	6	0	6
6	63	20	V	5	2.5	2.5
7‡	48	144	IV	3	3	0
8§	36	13	III	2	0	2
9‡	76	33	VI	7	7	0
10	33	48	V	7	2	5

* According to House and Brackmann.¹⁷

† Patient with Ramsay Hunt Syndrome.

‡ Patient with tarsorrhaphy.

§ Patient with paralysis secondary to a traffic accident.

The main-sequence data for downward and upward movements derived from five spontaneous blinks in each of 30 normal volunteers are shown in Figures 3 and 4, respectively. The maximum displacement measurements were banded into 0.5-mm intervals, and the mean displacement and velocity were calculated for each interval. The error bars represent the SD from the mean velocity for each interval. Data from at least five blinks were used for each interval.

In both cases, the data are variable, as indicated by the error bars. This observation is consistent with previous studies. Also, for both movements, higher values of eyelid velocity were associated with larger displacement amplitudes, as reported elsewhere.¹⁹ Linear regression analysis of the peak eyelid velocity (Y) and maximum eyelid displacement (X) data produced the relationships

$$Y = 21.4X + 31.7 \quad \text{for downward lid movement, and}$$

$$Y = 10.8X + 13.7 \quad \text{for upward lid movement.}$$

The corresponding regression equations published by Evinger in 1991¹⁹ are

$$Y = 29.2X - 35.9 \quad \text{for downward lid movement, and}$$

$$Y = 13.5X - 5.87 \quad \text{for upward lid movement.}$$

Although these regression equations are different, values of velocity obtained are in reasonable agreement over the range of blink amplitudes commonly observed (<12 mm).

Previous studies have shown that in normal subjects the dynamics of voluntary and spontaneous blinks are similar, although voluntary blinks are in general associated with larger lid movements.^{19,20} We assessed the dynamic performance of treated paretic eyelids by comparing the main-sequence relationship of voluntary and spontaneous movements to the normal data described above.

Main-Sequence Relationship in Patients with Palsy.

A typical upper eyelid displacement response for voluntary closure in a patient with palsy after treatment is shown in Figure 5A. The downward movement is much slower than in a normal blink sequence, the time to peak closure being approximately two to three times longer than that observed during normal blinks. The response also shows a plateau separating the closing and opening phases. This indicates that the subject is attempting to hold the eye closed for longer than during a spontaneous blink. The lid velocity (shown in Fig. 5B) in the opening phase is similar to that in the closing phase.

The largest spontaneous response obtained in the same

TABLE 3. Upper Eyelid Displacement Initially and after 3 months' Treatment during Spontaneous Blinking

Patient	Age (y)	Palsy Duration (mo)	Grade*	Pretreatment Displacement (mm)	Posttreatment Displacement (mm)	Difference (mm)	P †
1	74	30	III	1.5	1.9	0.4	0.44
2	74	24	IV	1.8	2.5	0.7	0.15
3	30	12	III	1.7	1.5	-0.2	0.35
4	67	26	III	2.4	2.2	-0.2	0.21
5	73	14	IV	1.5	2.6	1.1	0.31
6	63	20	V	1.5	1.7	0.2	0.26
7	48	144	IV	2.2	1.8	-0.4	0.41

* According to House and Brackmann.¹⁷

† By t -test paired for means.

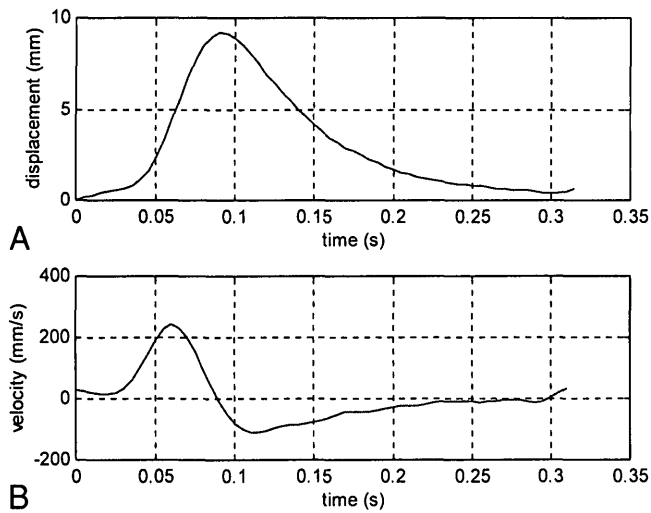


FIGURE 2. (A) Upper eyelid displacement (in millimeters) versus time (in seconds) in a normal volunteer during a spontaneous blink measured with data acquired from the line-scan system. (B) Velocity (in millimeters per second) versus time (in seconds) derived from the data in (A).

subject is shown in Figure 6A. Although the movement was similar in form to that illustrated in Figure 2A for a normal blink, the maximum displacement was small. The variation in velocity with time is shown in Figure 6B. Peak velocities were lower than those achieved during equivalent amplitude blinks in normal volunteers. In this patient, good recovery was made for voluntary closure, but spontaneous blink movement did not improve.

The main-sequence relationship for voluntary and spontaneous downward movements of parietic eyelids during treat-

ment is compared with our normal data in Figure 3. Parietic eyelid velocities for a given amplitude of movement are significantly lower than in normal subjects, and the populations of data points for the two groups are well separated. The regression equation for downward parietic eyelid movements was

$$Y = 5.7X + 6.2$$

In Figure 4, the main-sequence relationship for upward phase velocities in spontaneous and voluntary parietic eyelid movements are compared with our normal data. The regression equation for upward movements in parietic eyelids was evaluated as

$$Y = 4.4X + 31.7$$

Comparison of main-sequence data during upward movement in normal and parietic lids shows that the data points from the two populations were not well separated. These data indicate that upward eyelid movements in parietic lids are similar to those of normal subjects.

Comparison of the regression equations derived from downward and upward movement in normal subjects indicates that over the range of movement observed in this study, the upward eyelid movements of the patients with palsy were quicker than the corresponding downward movements.

Effect of Functional Electrical Stimulation. Application of this therapeutic regimen in normal volunteers causes the eyelid to twitch at a rate of 10 Hz. Strong contraction of the orbicularis oculi (OOc) and full eyelid closure can be obtained in normal subjects by increasing the stimulation frequency to approximately 40 Hz. Neither of these regimens produced detectable contraction of the OOc in the patients with palsy during or after the treatment period.

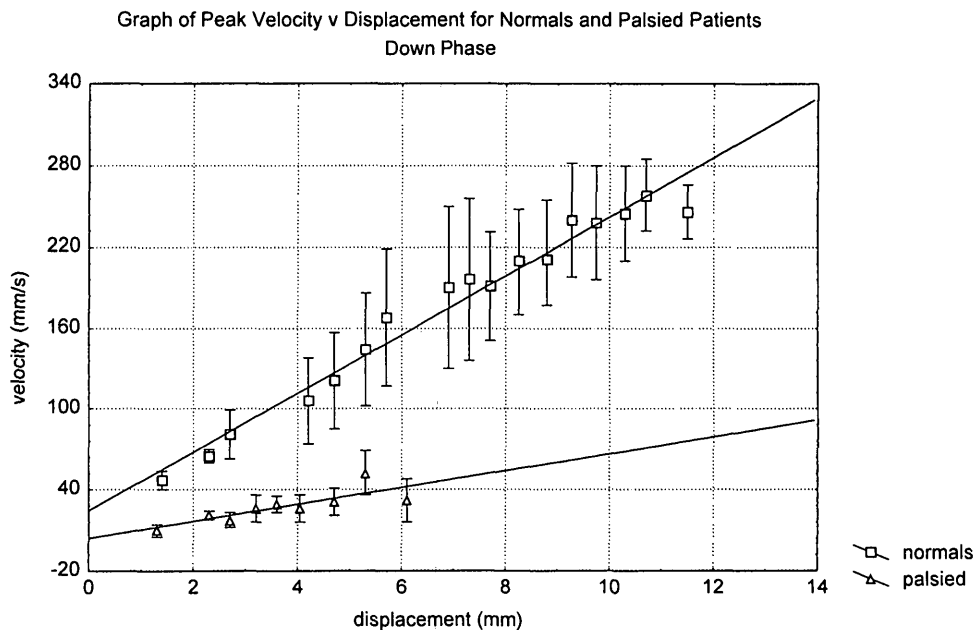


FIGURE 3. Velocity (in millimeters per second) versus displacement (in millimeters), showing main-sequence data of normal subjects and patients with palsy during the downward phase of spontaneous and voluntary blinks. Each point represents an average of at least five blinks. Error bar, the SD from the average for each point.

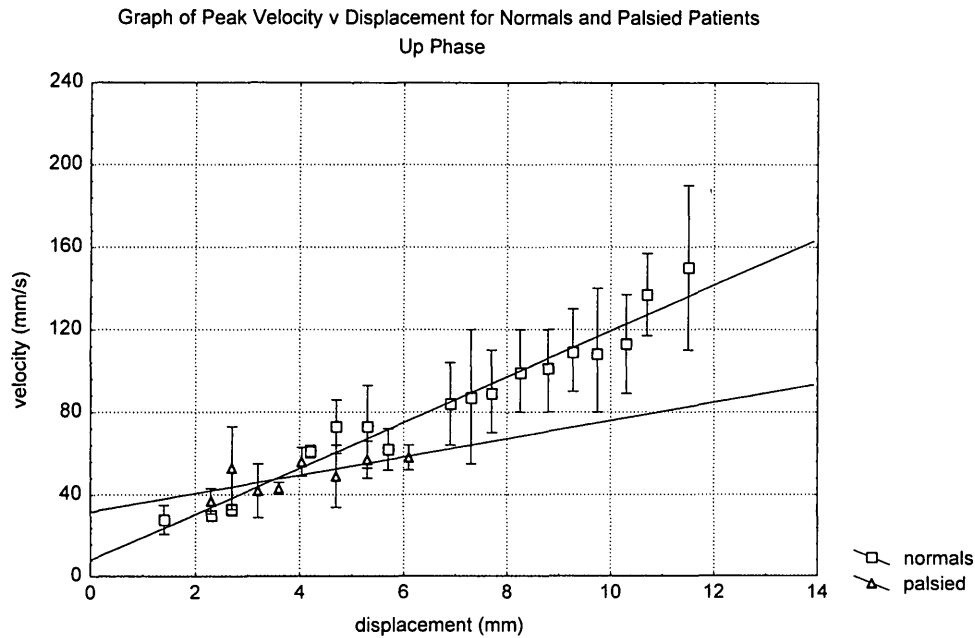


FIGURE 4. Velocity (in millimeters per second) versus displacement (in millimeters), showing main-sequence data of normal subjects and patients with palsy during the upward phase of spontaneous and voluntary blinks. Each point represents an average of at least five blinks. Error bar, the SD from the average for each point.

Further Observations

Four patients reported using less ointment than before treatment. During stimulation, one patient reported occasional slightly increased watering of the eye, which was not caused by pain or discomfort and returned to normal after each stimulation session.

Qualitative analysis of facial movements such as smiling, eyebrow movement, and puffing of the cheeks showed no change during the treatment period, which suggests that the gains observed in eyelid movement were attributable to the stimulation regimen.

DISCUSSION

The upper eyelid position is controlled by the levator palpebrae superioris (LPS), which keeps the upper lid open and is innervated by the third cranial nerve.^{19,22} Its action is augmented by the Müllers muscle and the frontalis muscle, which are innervated by the sympathetic nervous system and the seventh cranial nerve, respectively.^{19,22} The OOC is responsible for eyelid closure and is innervated by the seventh cranial nerve.^{19,22} The palpebral portion is associated with spontane-

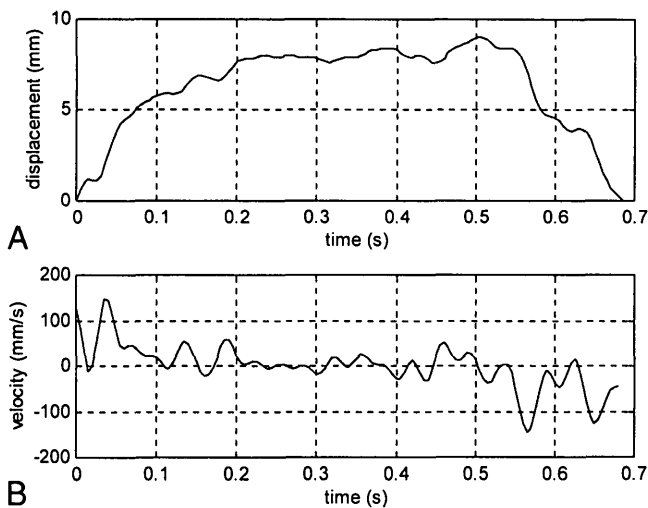


FIGURE 5. (A) Upper eyelid displacement (in millimeters) versus time (in seconds) in a patient with palsy during voluntary closure, measured with data acquired from the line-scan system. (B) Velocity (in millimeters per second) versus time (in seconds) derived from the data in (A).

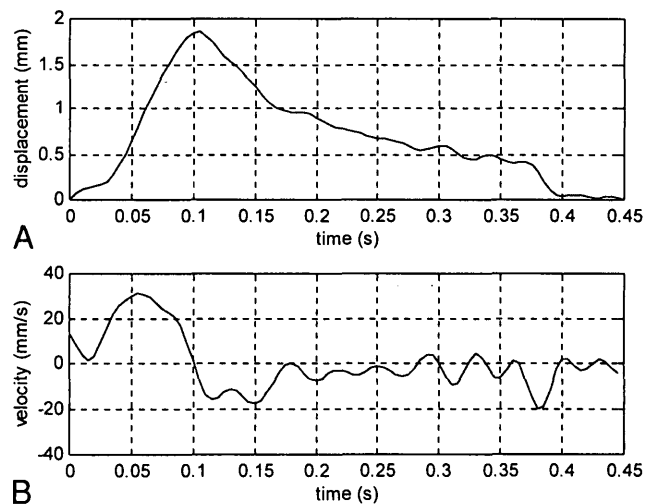


FIGURE 6. (A) Upper eyelid displacement (in millimeters) versus time (in seconds) in a patient with palsy during a spontaneous blink, measured with data acquired from the line-scan system. (B) Velocity (in millimeters per second) versus time (in seconds) derived from the data in (A).

ous eyelid movement, and the orbital portion is used during movements such as tightening the eyelids for protection. Electromyographic studies have shown that eyelid position is maintained by tonic activity within the LPS.¹⁹ Downward motion of the eyelid during a spontaneous blink is caused by cessation of LPS activity before contraction of the OOC.¹⁹ The eyelid is then raised by cessation of OOC activity and the onset of LPS activity.¹⁹ For voluntary eye closure, OOC activity precedes LPS inhibition.²³

Downward voluntary movement of paretic eyelids could be expected to provide complete closure, because there is no LPS activity holding the eyelid open. However, chronic severe seventh nerve paralysis results in limited voluntary and spontaneous eyelid movement.²⁴ This has been attributed to stiffening and contracture of the LPS arising from the absence of tonic OOC activity.²⁴ Changes to OOC fibers may also contribute to a reduction in elasticity of the eyelid structure.²⁴

The improvements seen in voluntary closure in this study caused by electrical stimulation may be explained by restoration of the OOC muscles' ability to contract or by a reduction in the stiffness of the eyelid structure that opposes eyelid movement. Recovery of function after reinnervation of the OOC muscle in this group of patients is unlikely. This is consistent with our observation that displacements and velocities in spontaneous blinks were not improved, and there was no observable direct response to electrical stimulation. During voluntary closure, the LPS is switched off and the eyelid allowed to relax downward. This movement is likely to be assisted by residual motor forces present in the palpebral and orbital regions of the OOC. The movement is slow, and maximum closure is achieved in a approximately 0.5 seconds. During spontaneous blinks, residual motor forces present only in the palpebral part of the OOC are thought to be involved, and the duration of reciprocal LPS and OOC activity is from 50 msec to 100 msec. Therefore, spontaneous blink amplitudes are relatively small in paretic eyelids. It is most probable that improvements seen in voluntary closure after treatment with electrical stimulation were caused by reductions in stiffness of the LPS and OOC muscles that allowed the eyelid to respond more freely to residual motor and elastic forces present during downward eyelid movement.

This explanation may seem inconsistent with the observation that denervated lid saccades, which depend on elasticity within the eyelid and a reduction in LPS activity shows only a small reduction in displacement and peak velocity in the presence of seventh nerve palsy.²⁴ However, the difference in the range of the two types of movement can be attributed to mechanical linkage between the superior rectus and the LPS.²⁰ Although this link assists downward saccadic movement as the superior rectus relaxes, it may hinder voluntary eye closure, because the latter is accompanied by upward rotation of the eyeball (Bell's phenomenon) and contraction of the superior rectus.

The slowness of voluntary eyelid closure after treatment coupled with the absence of improvement in spontaneous blink response means that a blink reflex suitable for maintaining physical protection to the cornea will not be recovered by this stimulation regimen, despite the observed improvements in voluntary eyelid closure, implying that current clinical practice for protecting the cornea should still be followed.

The absence of response to treatment in the two subjects who had undergone tarsorrhaphy may be attributable to the

effect of surgical intervention on the mechanics of eyelid movement. Such surgery may cause increased stiffness and a reduction in elasticity of the eyelid closure mechanism.

CONCLUSION

We have established that in patients with chronic facial palsy, daily treatment with transcutaneous electrical nerve stimulators for 3 months can significantly improve the range of voluntary eyelid movements. The increase in movement does not seem to be attributable to improvements in OOC muscle function directly but may be associated with a reduction in stiffness of the eyelid apparatus. Spontaneous blinks were not improved.

The stimulation protocols used here did not give detectable contractions in the OOC. Therefore, these protocols could not be used to restore eyelid function by stimulation therapy followed by implantation of stimulating electrodes. However, other benefits were achieved apart from the improvement in voluntary closure. These included a reduction in the amount of artificial tear preparations used in four patients and subjective improvement in facial tone reported by all patients.

Further study is currently under way to assess the effects of alternative stimulation protocols using purpose-built stimulators.

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ANNOUNCEMENTS

Johns Hopkins Center for Alternatives to Animal Testing 2000-2001 Grants Program Call for Proposals

The Center for Alternatives to Animal Testing (CAAT) is soliciting projects focused on investigating the fundamental knowledge needed to develop alternative methods to the use of whole animals for safety/hazard evaluation, risk assessment and efficacy. We encourage the investigation of in vitro approaches to evaluate cellular and target organ toxicity. AT THE PRESENT TIME, CAAT DOES NOT FUND PROJECTS RELATING TO CARCINOGENICITY OR MUTAGENICITY.

For further information and to apply, submit the preproposal on-line through our web site <http://altweb.jhsph.edu> by March 15, 1999. No other materials are required for this stage of the application process. Applications appropriate to the goals of CAAT will be invited to submit a complete grant application package. All responses will be forwarded by e-mail or U.S. mail. No telephone responses will be given.

Request for Proposals

The Glaucoma Research Foundation is requesting proposals to investigate the normal and glaucomatous functions of the anterior segment of the eye and conduct genetic analyses of sibpair and isolated populations as recommended by experts at our Genetics and Molecular Biology of Glaucoma Meeting.

Grants range from \$15,000 to \$50,000 per year and are awarded for one- or two-year projects.

Proposals must be submitted by May 1, 1999. Please contact Mel Garrett at mgarrett@glaucoma.org or (415) 986-3162 to obtain an application and a list of the specific meeting recommendations.