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Efficacy of gold weight implants in facial nerve palsy: quantitative alterations in blinking

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Abstract

Deficient eyelid closure is a major visual threat to patients with unresolved facial nerve palsy. Gold weight implants assisted eyelid closure in patients with paresis of the orbicularis oculi, ameliorating patient complaints of dry eye, excessive tearing, and corneal epithelial breakdown. We used dynamic measures to assess the efficacy of upper eyelid gold weight implantation surgery for facial nerve palsy. The search coil technique was used to record spontaneous blinks bilaterally in six patients, before and after unilateral gold weight implantations into the upper eyelid in severe facial nerve palsy. In uncomplicated facial nerve palsy, the amplitude of blink down-phases for the paretic eyelid was $28.6 \pm 5.7\%$ of the amplitude of the contralateral, normal eyelid. Following corrective surgery, closure of the paretic eyelid improved to $42.6 \pm 7.5\%$ ($P < 0.05$). There was not a commensurate increase in the peak velocity of blink down-phases, suggesting that gold weight effects are mediated by a passive improvement in blink dynamics.

Keywords: Bell's palsy; Facial nerve; Eyelid; Orbicularis oculi; Blink

1. Introduction

Facial nerve palsy may be traumatic, viral, iatrogenic or idiopathic in origin, and exhibits no identifiable age, gender or laterality preference [1,2]. While functional nerve palsy may be short-lived in idiopathic cases, the prognosis for recovery of muscles of facial expression is poor in traumatic or iatrogenic cases. There are potentially devastating ophthalmologic consequences for the patient, with exposure keritits and corneal ulceration, leading to decreased visual acuity, being the greatest concern [3,4].

Incomplete eyelid closure in facial nerve palsy patients may be addressed by either internal compensatory responses, generated by eyelid movement control systems, or external therapeutic interventions. For example, Bell's palsy patients increase the aggregate discharge of orbicularis oculi motoneurons bilaterally in an attempt to improve eyelid closure in monocular palsy [5,6]. The duration of the pulse of orbicularis oculi activity also can be adaptively lengthened to achieve a greater degree of closure in the paretic eyelid. These internal attempts to cope with facial nerve palsy are viewed as an adaptive response to ensure that the movement that actually occurs matches that intended by the motor control system [7,8]. Such adaptive responses have been described for several motor systems and may exert a positive influence upon functional status or, by contrast, may put the patient at risk for disease processes [9–11]. For example, we have suggested that the adaptive response to facial nerve palsy may be permissive for a subsequent episode of blepharospasm, a disorder termed Bell's palsy-induced blepharospasm [10,11].

A variety of external therapeutic measures have been explored in order to alleviate the ophthalmic consequences of facial nerve palsy [3,12,13]. The attachment of external weights to the upper eyelid, as a temporary measure to aid eyelid closure, has received support as a treatment in cases where some degree of recovery is anticipated [12,13]. When the paresis of eyelid closure

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Table 1
Patient clinical evaluations

Patient #	Etiology	Implant size (g)	Keratopathy pre-operative score[a]	Visual acuity pre-operative[b]	Keratopathy post-operative score[a]	Visual acuity post-operative[b]
1	Traumatic	1.2	1+	20/200	0	20/30
2	Traumatic	1.2	1+	20/30	0	20/100
3	Bell's	1.0	3+	20/50	0	20/60
4	Traumatic	0.8	1+	20/300	0	20/30
5	Bell's	1.6	Trace	20/60	0	20/20
6	Traumatic	1.8	3+	20/40	0	20/25

Etiology of facial nerve palsy is noted as traumatic, when clearly indicated by history, or Bell's, when idiopathic.
[a] Arbitrary numerical scoring of degree of keratopathy; higher values denote more severe symptoms.
[b] Visual acuity values are for paretic eye, by Snellen test.

appears to be long-term or irrecoverable, the preferred treatment of lagophthalmos is the surgical implantation of a gold weight in the upper eyelid.

The efficacy of gold weight implants for facial nerve palsy has largely been defined by qualitative criteria. An evaluation of the efficacy of this procedure in facioscapulohumeral muscular dystrophy-associated lagophthalmos [14] concluded that "a quantitative evaluation of blink kinematics is important to assess improvement and for follow-up evaluation of recovery of blink function." In this study, we used recorded instantaneous eyelid position in order to evaluate movement dynamics in facial nerve palsy patients selected for lid loading surgery. While these data show that blink down-phase amplitude was only modestly improved and down-phase peak velocity was virtually unchanged by gold weight implants, the improvement in eyelid closure yielded sufficient corneal coverage for an excellent clinical result.

2. Methods

Eyelid movements were recorded and analyzed in six facial nerve palsy patients (three male and three female; 23–62 years old) selected for gold weight implantation at the University of Kentucky Ophthalmology Clinic. Patients were symptomatic for severe, unilateral facial paralysis with deficient eyelid closure of greater than 4 months duration (two patients were idiopathic Bell's, four patients were traumatic facial nerve palsy cases). All patients were given thorough ophthalmologic examinations and informed written consent was obtained prior to study.

Patients were evaluated for the degree of exposure keratopathy, before and after surgery, using routine fluorescent staining techniques. Keratopathy was graded on a scale of 1–4, on the basis of examiner experience. Patient eyelid movements were recorded prior to surgery, and after adequate post-operative recovery, using the electromagnetic search coil technique, as described in our previous studies [6,10,11,15–17]. This technique relies upon measurement of the signal induced in a wire coil attached to the eyelid as a result of movement within a magnetic field. Briefly, subjects were seated in a chair centered in a 6-foot Robinson coil system (CNC Engineering) used to generate a low-level magnetic field. The patient's head was fixed in position and fine Teflon-coated copper wires coils (160 mg, 30 turns, 6 mm in outer diameter) were taped to the center of each upper eyelid, immediately adjacent to the eyelashes. Coils did not interfere with lid movements. Recordings of spontaneous blinks were obtained over a 10–15 min period and analog signals were digitized at 500 Hz and stored for off-line analysis. A minimum of 20 blinks per patient were analyzed from both pre-operative and post-operative recording sessions. Eyelid movement records were evaluated for both down- and up-phase amplitude, duration and peak velocity. At the end of each recording session, the coils were removed, the patient's spontaneous blink pattern was videotaped for 5 min, and resting palpebral fissure width was measured while fixating a target at eye level.

Patients meeting the selection criteria underwent an outpatient surgical procedure (performed by RSB or DEC), in which gold weights (Meddev) were implanted. On a follow-up visit at least 8 days after surgery, eyelid movements were studied with the search coil system.

3. Results

The criteria for clinical success in treatment of lagophthalmos were: (a) reduction or elimination of exposure keratopathy and (b) no significant post-operative complications. In clinical examinations of patients prior to surgery, modest to severe exposure keratopathy was present (Table 1). Two patients (# 2 and 4) presented with diminished corneal sensation. At a 2-week post-operative examination, slit lamp examination of fluorescein staining of corneal epithelium showed that

keratopathy was eliminated in all patients. In addition, all patient wounds healed adequately, with no signs of inflammation, and none of the implants were rejected or extruded. Corneal pathology also has the deleterious consequence of deterioration of vision. The outcome of Snellen visual acuity examinations was variable, with improvement seen in four of the six patients (Table 1). In patient #2, a gold weight-induced ptosis partially obscured vision and the weight subsequently had to be removed.

The facial nerve palsy patients in our study exhibited improvement in eyelid closure following implant of gold weights of 0.8–1.8 g. While prior studies of the efficacy of gold weight implantation provided only subjective evaluations, the search coil technique used here gives a precise assessment of the impact that the gold weight has on eyelid closure. Fig. 1A, B, C illustrates single blink lid position waveforms recorded from a patient (Subject #1) with right-sided VII nerve palsy due to trauma, before (Fig. 1A, B) and after surgery (Fig. 1C). In each trace, the down-phase (downward deflection of the position trace) and up-phase (upward deflection of the trace) for a representative spontaneous blink are portrayed for each eyelid. In each panel, the top trace illustrates movements of the right (paretic) eyelid and the bottom trace shows movements of the left (control) eyelid. Fig. 1A illustrates a representative pre-operative blink waveform. In this example, the amplitude of the paretic eyelid down-phase was approximately 30% of that of the normal eyelid, making adequate lid closure impossible. Also in Fig. 1A, we note that downward saccadic eyelid movements (asterisks) are preserved for the paretic eyelid in the absence of adequate blink function. Fig. 1B illustrates a second pattern of blinking commonly observed in patients with significant eyelid paresis. Note that the blink waveform includes an abnormally long down-phase and extended pause at the point of maximum eyelid downward excursion. We have previously interpreted this pattern in Bell's palsy patients as resulting from a prolonged, binocular pulse of innervation to the orbicularis oculi [6]. This is considered to be an adaptive means of obtaining the maximum possible closure of the paretic eyelid. Fig. 1C illustrates a representative blink trace after gold weight implantation. Paretic eyelid amplitude now was approximately 50% of control, representing an increase of 1.7 times from pre-operative measures. This level of recovery of function was not adequate to eliminate internal attempts to cope with eyelid paresis, as evidenced by the occurrence of the prolonged closures in Fig. 1B at the same frequency before and after the implantation surgery. The patient did experience symptomatic relief.

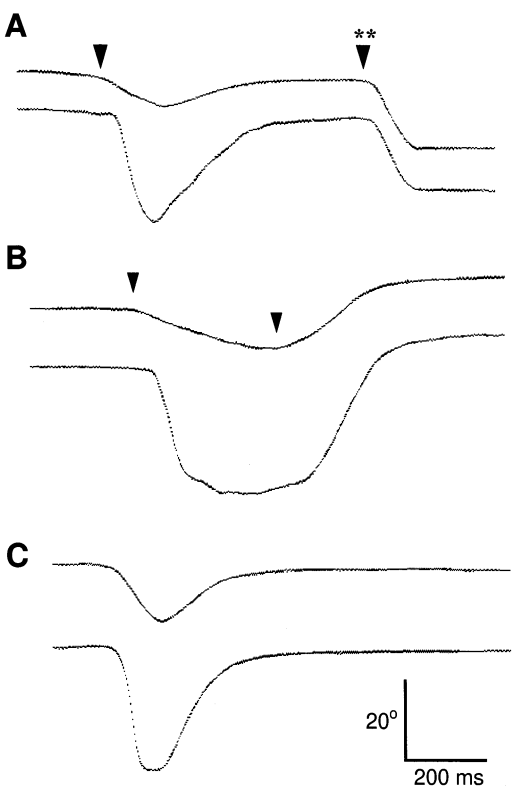

Fig. 1. Blink waveforms (subject #1) with right-sided VII nerve palsy. Pre-operative blinks displaying: (A) monocular weakness of blink (arrowhead), but normal down lid saccades (arrowhead with asterisks), and (B) binocular prolongation of the pulse of innervation to assist closure (arrowheads denote start and end of prolonged down-phase). After gold weight implantation (C), paretic lid function is improved, but still significantly less that control.

Table 2
Changes in eyelid kinematics from gold weight implant

Patient #	Pre-operative control (%)		Post-operative control (%)	
	Amplitude	Velocity	Amplitude	Velocity
1	23.3 ± 1.8	20.1 ± 1.1	38.5 ± 1.4*	32.7 ± 1.8*
2	45.9 ± 3.0	73.0 ± 4.3	72.1 ± 4.0*	67.0 ± 2.2
3	46.1 ± 5.8	64.3 ± 8.0	47.5 ± 6.0	56.0 ± 5.0
4	23.0 ± 5.4	38.1 ± 4.9	27.7 ± 0.7	22.0 ± 0.9*
5	20.5 ± 1.1	19.0 ± 0.6	50.0 ± 1.4*	34.1 ± 1.0*
6	13.0 ± 5.2	25.8 ± 2.5	20.0 ± 1.1	32.2 ± 2.6

Data expressed as % of contralateral, control eyelid; mean ± S.E.M.
* Denotes different from preoperative value at $P<0.01$.

All six patients exhibited improved eyelid closure when the percent of contralateral eyelid amplitude was used as a criterion (Table 2), although improvement did not always reach significance. In Table 2, the results of gold weight implantation are displayed as a percent of control amplitude. Improvement in the amplitude of eyelid closure reached significance in four of six facial nerve palsy patients. Patients who did not show significant recovery had received the smallest weights. The degree of improved eyelid closure that was obtained did

not appear to be associated with either the initial level of paresis or the etiology of facial nerve palsy. Mean pre-operative paretic eyelid amplitude, expressed as a percent of the control eyelid, was $28.6 \pm 5.7\%$. Patients improved to $42.6 \pm 7.5\%$ of control after surgery (significant at $P < 0.05$). By contrast, the passive manipulation of implanting a weight into paretic eyelids did not alter down-phase peak velocity (mean pre-operative value was $40.1 \pm 9.5\%$ of control and post-operative was $40.7 \pm 7.0\%$ of control; n.s. at $P > 0.9$; Table 2). In addition, neither blink rate nor palpebral fissure width exhibited a consistent pattern of change subsequent to gold weight implantation.

4. Discussion

We have provided documentation of the efficacy of a procedure long used to provide surgical relief of corneal dryness and ulceration accompanying persistent facial nerve palsy. While gold weight implantation is associated with an improved clinical outcome, and qualitatively improves eyelid closure, the actual degree to which eyelid function is restored is more limited than had been expected. These data suggest that effective resolution of the ocular signs that accompany orbicularis oculi paresis may be achieved by a procedure that only passively and incompletely improves eyelid dynamics.

Prior studies have demonstrated that the surgical attachment of a gold weight to the tarsus is an effective means of managing the corneal consequences of facial nerve palsy [14,18]. In normal blinks, the orbicularis oculi and levator palpebrae superioris act in a push-pull fashion [19]. The orbicularis is normally silent while tonic activity of the levator palpebrae maintains the eyelid in an open position. Blinks are triggered by the phasic activation of the orbicularis oculi coupled with the phasic inhibition of the levator palpebrae, thereby generating an eyelid closure. However, passive elements also contribute to the downward movement of the eyelid. The at-rest position of the upper eyelid is down, as determined by the anatomic organization of the levator aponeurosis, the superior transverse ligament, and the palpebral ligaments [19]. In addition to assisting in the down-phase of blinks, these passive elements are principally responsible for the downward movements of the eyelid with saccadic eye movements, lid saccades, which are generated by relaxation of the levator palpebrae without the participation of the orbicularis oculi. Facial nerve palsy patients then present with insufficient eyelid closure in blinking, but downward eyelid saccade function is preserved, as it is in experimental orbicularis oculi paralysis [20]. Gold weight is effective in ameliorating the ocular signs of facial nerve palsy because it provides an additional increment in downward passive force to close the eyelid when the levator palpebrae relaxes.

There are several potential mechanisms for evaluating closure of the eyelid, including alterations in blink dynamics, volitional sustained eyelid closure, and completeness of closure during blink. Generally, prevention of exposure keratopathy and/or relief of corneal dryness, rather than any measure of the degree of eyelid closure, has been the standard for evaluation of efficacy. In some studies, the improvement in eyelid closure appears to be assumed and success rate is based upon non-extrusion of the weight. By these measures, success rate is judged to be between 75–90% [3,4,13]. De Min et al. [4] have used a static measure of residual corneal exposure (palpebral fissure width upon sustained eyelid closure), finding that surgery significantly reduced corneal exposure.

In contrast to these prior studies, we have used an active measure of eyelid closure in order to assess the degree of improvement offered by gold weight implantation. While providing adequate eyelid closure by clinical criteria, this procedure rarely restored paretic eyelid function to $> 50\%$ when the down-phase amplitude of the contralateral, normal eyelid is used as a control. These results provide an interesting paradox since gold weight implantation provides symptomatic relief with only marginal improvement in the amplitude of closure during blinking. The efficacy of this procedure was not associated with the pre-existing level of paresis; patients with severe or moderate paresis were equally likely to achieve the same level of recovery. Importantly, the increase in amplitude of closure of the paretic eyelid was not accompanied by an appropriate increase in down-phase peak velocity. The relationship between down-phase amplitude and peak velocity, or main sequence, is linear [15,19,21–23] and the degree of improvement in amplitude would be associated with an appreciable increase in peak velocity if there was a substantive increase in active force of eyelid closure.

These results imply that there are two components to eyelid closure that must be considered in health and disease. First, the eyelid may close in a prolonged, 'static' manner, resulting from sustained levator palpebrae superioris relaxation, sustained recruitment of the orbicularis oculi (including palpebral and orbital portions), or both, acting in concert with the passive forces that close the eyelid. Prior reports and present data suggest that this component of eyelid movement is aided by gold weight implantation. Second, the eyelid closes in a rapid, 'dynamic' fashion, briefly covering the cornea in blinks. The mean down-phase amplitude generated by this active component also is augmented by the gold weight, and this likely aids corneal hydration, but there is not a commensurate increase in blink velocity. Our data suggest that corneal coverage is the key therapeutic issue. Normal amplitude/velocity rela-

tionships of blinks do not have to be restored in order to achieve an effective result. This evaluation of eyelid movements in facial nerve palsy leads us to the conclusion that the eyelid closure assistance that is provided by gold weight therapy is passive, and more limited than would have been expected by clinical observations alone.

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