Eyelid Movements in Facial Paralysis

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- We studied the eyelid movements of six patients with unilateral, isolated, facial paralysis using the magnetic search coil. The most significant abnormality consisted of a reduction in the magnitude of the orbicularis oculi contraction with slowing of the peak velocity of the closing phase of the blink. The closing phase blink velocity, which normally increases linearly as a function of amplitude (main sequence), in our patients displayed a relationship characterized by a slow saturating power function that fell off the main sequence. The contralateral normal lid in some cases can show adaptive signs of hyperactivity during the closing phase of the blink. Lid saccades showed a small but consistent decrease in amplitude and velocity compared with the contralateral unaffected eyelid. Unlike the closing phase of the blink, peak velocities of lid saccades stayed on the main sequence. In this study, we discuss how the eyelid executes downward lid saccades based purely on a passive mechanism.


Movement of the upper eyelid is produced by the activity of two reciprocally acting muscles: the levator palpebrae superioris (LPS) and the orbicularis oculi (OOc). The closing phase of the blink is the result of a phasic contraction of the OOc associated with a momentary pause in the tonic activity of the LPS; the opening phase begins with cessation of OOc activity and resumption of LPS tone. In contrast to other body movements that are usually controlled by the actions of agonist-antagonist pairs, the eyelid movements that accompany changes in vertical eye position are unique, in that they are governed by the activity of the LPS alone. Electromyographic (EMG) studies have shown that the tonic activity of the LPS increases in upward gaze and decreases in downward gaze.

The eyelid movements that accompany vertical eye saccades have trajectories and peak velocities very similar to eye saccades themselves, prompting Becker and Fuchs' to coin the term "lid saccades." There are no consistent differences between the trajectories of upward and downward saccadic movements of the eyelid,13,14 and yet the mechanism producing each is quite different. Where the trajectory of the upward lid saccade can be explained by a burst-tonic pattern of activity in the LPS, the explanation for the trajectory of the downward lid saccade has been problematic because there is no associated EMG activity in the OOc.15

A variety of downward forces opposing the LPS have been proposed to explain the downward lid movement. They include (1) gravitational forces; (2) frictional dragging of the lid by the globe; (3) undetected phasic bursts of OOc activity; and (4) passive elastic forces.

Gravitational forces on the eyelid would appear to have little effect because eyelid movements are said to remain unchanged when standing upside down.22 Likewise, mechanical coupling between the globe and lid on downward gaze probably also plays a small role, if any, because tapping the lid away from the globe has no effect on saccadic eye velocity23 and changing the viscosity of the ocular surface has minimal effects on downward lid movements.2 To account for the peak velocities of the downward lid saccades, it has been suggested that a phasic contraction of the OOc must exist, despite the fact that EMG studies of the OOc during vertical lid saccades have failed to document any signs of activity.13,14,15 While Evinger et al14 and others15 have observed small, inconsistent bursts of EMG activity in the OOc during both upward and downward lid saccades, such bursts are only seen with large-amplitude saccades, develop only after the lid has started its descent, and cease before completion of the lid saccade.

The force that probably plays the most important role is generated by the passive downward elastic properties of the eyelid. Kennard and Smyth26 have shown that the force required to elevate the eyelid from downward gaze increases exponentially as a function of upward displacement, that is, lid stiffness increases as the lids are elevated. These logarithmic properties suggest a group of parallel elastic elements that are successively called into play.27

To better define the role of the OOc in vertical eyelid movements, we studied a group of patients with unilateral facial paralysis.

SUBJECTS AND METHODS

Subjects

Six patients (four women, two men) between ages 28 and 47 years, with unilateral, isolated, facial neuropathies were studied. The causative factors included Bell's palsy (patients 1, 2, 5, and 6), Lyme disease (patient 3), and diabetes (patient 4). All patients underwent complete ophthalmic and neurologic examinations. All patients had grade IV (patients 1, 2, and 4 through 6) to grade V (patient 3) dysfunction based
on the House and Brackmier grading system, demonstrating an incomplete closure of the blink and forced lid closure. Patients were fully informed about the experimental procedures, and provided written consent in accordance with State University of New York–Stony Brook policy for testing human subjects.

**Measurement of Lid Position and Velocity and EMG Activity**

The methods used in this study have been described in more detail elsewhere. Briefly, a magnetic search coil (DC-300 Hz, CNC Engineering, Seattle, Wash) was used to record eyelid movements. A nonstick coated with Teflon wire (model 9710, AM Systems, Everett, Wash) was taped to the center of the lower margin of the upper eyelid. As the eyelid rotated over the ocular surface, the eyelid coil produced a voltage that was proportional to eyelid position. The system was capable of detecting lid rotations as small as 0.25°, equivalent to a lid movement of 0.05 mm. To correlate the magnetic search coils output with lid position, the subject fixated on different points along the vertical meridian, while the investigator measured the angle of lid rotation with a protractor.

Two miniature silver electrodes (<2 mm in diameter) taped to the lateral and medial portions of the upper lid near the lower margin monitored OoE muscle activity (300 Hz to 5 KHz; amplifier model 1706, AM Systems). A third gold-cup electrode (9 mm in diameter; Grass Instrument Co, Quincy, Mass) taped to the forehead served as a ground. To evoke reflex blinks, two additional skin electrodes (Grass) were taped over the supraorbital branch of the trigeminal nerve. One electrode was attached to the brow over the nerve as it exited the skull, and the second was placed 1 cm above the first. Prior to electrode application, the skin under the electrodes was cleaned with alcohol and all electrodes were coated with conductive paste (EC 2 Electrode Cream, Grass).

**Procedure**

In all conditions, the subjects faced a screen 116 cm from the eyelids with their heads maintained in a constant position parallel to the screen. Directly in front of the subject, a laser-generated red light at eye level was projected onto the screen and served as a fixation target. The following types of lid movements were recorded: voluntary, reflex blinks, and those accompanying vertical saccadic eye movements. Voluntary blinks occurred, and were recorded, when the subjects heard a computer-generated tone (20 trials) at interstimulus intervals ranging between 1 and 7 seconds. To obtain reflex blinks, the supraorbital nerve received 100-microsecond electrical stimulations (model A310 and A360R, WPI, New Haven, Conn). The exact parameters were established for each subject, and adjusted so as to evoke blinks in the normal eye with a variety of amplitudes. The computer triggered 15 reflex blinks, with an interstimulus interval ranging pseudorandomly from 12 to 18 seconds.

To produce saccadic lid movements, the subjects visually followed computer-controlled movements of the internal and external vertical meridian. A mirror galvanometer system (model CX6325, General Scanning, Watertown, Mass) moved the laser spot in steps of 4.5° to 27° of visual angle from initial positions in the range of 13.5° above to 13.5° below the horizontal meridian every 2 to 4 seconds. The sequence of 100 target positions were balanced, so that equal numbers of target steps occurred below and above the straight-ahead position. The entire procedure took less than 1 hour.

**Data Collection and Analysis**

Data were collected and stored on a personal computer for off-line analysis. Lid position and EMG activity in the OoE were sampled at 5000 Hz with 12-bit precision. For analysis, the computer displayed lid position, lid velocity (digitally calculated from the lid position record), and the rectified EMG activity in the OoE for each trial. For blinks and saccadic lid movements, the investigator marked the beginning and end of EMG activity in the OoE. For all lid movements, the investigator identified the maximum down-phase (closing phase) velocity, the maximum up-phase (opening phase) velocity, the beginning of the blink or lid saccade, the maximum downward lid excursion, and the end of the blink or lid saccade. From these points, the computer calculated and stored down- and up-phase blink amplitude or saccade amplitude, correlative saccade amplitude, duration, maximum velocity, latency from EMG activity in the OoE to blink onset, and the integrated EMG activity in the OoE. Velocity-amplitude figures depicting data obtained from blink experiments pooled the data from both voluntary and reflex blinks.

**Controls**

Eyelid movements of patients with facial paresis were compared with a group of normal subjects who were concurrently studied under identical test conditions. The results from these normal subjects have been reported elsewhere. Figures depicting the results from our patients will, where appropriate, also show results obtained from our normative database. The contralateral unaffected eyelid was also used as an additional control in three of the six patients studied (patients 3, 5, and 6).

**Videotape Analysis**

In an attempt to correlate the quantitative data with clinical observations, three of the six patients were videotaped at close range, using a standard commercially available video camera. Videotapes were then used to compare the movements of the normal and the affected eyelid.

**RESULTS**

**Blinks**

The trajectories of the normal and paretic eyelid are illustrated in Fig 1. Visual inspection of individual records shows that the EMG activity in the OoE was greatly diminished in magnitude, reducing the peak velocity of the closing phase. The effect varied from one patient to another. For example, small-amplitude blinks still occurred in three patients, despite the complete absence of any detectable EMG activity in the OoE. The EMG activity in the OoE normally precedes downward lid motion by 10 to 15 milliseconds (Fig 1, left); however, in the three remaining patients in whom EMG activity in the OoE was detected, its onset, relative to the start of downward lid motion, was delayed. For example, in one patient the onset of EMG activity in the OoE was detected simultaneously with the onset of eyelid motion; in two other patients, EMG activity in the OoE developed 5 to 25 milliseconds after the start of downward eyelid motion (Fig 1, right).

The peak velocity of both the closing and opening phase of the blink normally increases as a linear function of amplitude. The slope of the closing phase is nearly twice that of the opening phase (Table 1). The normal velocity-amplitude relationship, referred to as the “main sequence”, is shown in the velocity-amplitude plots as a pair of fine dotted lines defined to include 95% of the recorded peak velocities at any given amplitude. The normative data, based on a study of nine subjects, has been published elsewhere. Peak velocities that consistently fell outside this range were considered abnormal.

Figure 2, left, summarizes the peak velocities and amplitudes of the closing phase in the abnormal eyelid of all six patients in this study. That our patients were incapable of completely closing the affected eyelid is shown by the profound reduction in the range of blink amplitudes. Whereas normal subjects displayed amplitudes that range from 2° to 60°, our patients’ closing phase rarely exceeded 15° (Fig 2, left). This decrease in the size of the blink was accompanied by an equally significant reduction in peak velocities. For example, the normal peak velocity for the closing phase is about 400° per second for a 15° blink. Our patients’ closing phase velocity was only 100° per second for an equal amplitude blink (Fig 2, left).

Although the maximum velocities of the closing phase in the paretic eyelid increased as a function of blink size, they did so at a slower rate of increase that clearly fell off the main sequence. The nature of the velocity-amplitude relationship was transformed. Instead of the linear function seen in normal subjects, the velocity-amplitude rela-
tionship in our patients was best characterized by a slow saturating power function: \( y = 40.39x^{0.57} \) (Fig 2, left, and Table 1). Analysis of covariance showed that there was no statistical difference between each of the six patients with facial paralysis, there was a significant difference between the paretic eyelid of the patient group and the normal controls \( (P<.001) \).

The unaffected eyelid in two of our patients with facial paralysis was also abnormal. Figure 2, right, summarizes data obtained from both eyelids recorded simultaneously in patients 3, 5, and 6. The data from the paretic lid are displayed as open symbols (confined to the lower left corner of the plot) and those from the unaffected lid are represented by closed symbols. Patients 3 and 5 had peak velocities from the unaffected side that were consistently above and outside of the normal range, suggesting that the OoC may be hyperactive during the closing phase of the blink. Patient 6, however, had peak velocities consistently within the normal range.

There was an equally significant decrease in the amplitudes of the opening phase. The peak velocities of the opening phase were all on the main sequence, and were linearly related to amplitude \( (y = 9.74x \pm 34) \) but not statistically different from the normal subjects (Fig 3 and Table 1).

**Table 1.**—Peak Velocity-Amplitude Regression Equations

<table>
<thead>
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<th>Direction</th>
<th>Equation</th>
<th>Coefficients</th>
<th>Slope</th>
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<tbody>
<tr>
<td>Down</td>
<td>Normal</td>
<td>( y = 29.2x - 35.9 )</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Paretic</td>
<td>( y = 40.4x^{0.77} )</td>
<td>( -9.74x + 34 )</td>
</tr>
<tr>
<td>Up</td>
<td>Normal</td>
<td>( y = 29.2x - 35.9 )</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Paretic</td>
<td>( y = 61.7x^{0.53} )</td>
<td>( -10.3x + 55.1 )</td>
</tr>
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**Lid Saccades**

The maximum velocities for both upward and downward lid saccades
increased as a function of amplitude (Figs 4 and 5). Compared with the normative database values, lid saccades appeared to be normal. Peak velocities for upward lid saccades were linearly related to amplitude \( (y = 16.8x + 55) \) well within the normal range (Fig 4, Table 1). Similarly, the velocity-amplitude relationship for downward lid saccades, characterized by a power function \( (y = 61.17x^{0.66}) \), also with few exceptions were within the range of normal subjects (Fig 5 and Table 1). The difference between our patients and the normal subjects was not statistically significant.

Clinical observations failed to disclose any apparent disturbances in the eyelid movements that accompanied vertical eye saccades. However, on viewing sequential single frames from the videotapes, the paretic lid lagged behind the normal lid. The final position of the upper lid on completing the saccade remained slightly higher than the normal lid even after completion of the postsaccadic adjustments. It appeared that both amplitude and velocity were slightly smaller and slower on the paretic side.

Using the contralateral unaffected eyelid as a control, comparison of individual recordings confirmed that there was an asymmetry between eyelids. For example, Fig 6 shows simultaneously recorded downward lid saccade from both eyelids in response to a target step of 17.6°. While there was no difference in latency, the paretic eyelid was substantially smaller in amplitude and peak velocity than the normal eyelid. The final position of the affected eyelid failed to descend to the same level as the normal eyelid.

Figure 7 summarizes the peak velocities and amplitudes for downward lid saccades from both eyelids of patients 3, 5, and 6; the open symbols represent the unaffected eyelid and the closed symbols represent the paretic eyelid. Despite the fact that both eyelids, for the most part, are within the normal range, there is a tendency for the unaffected lid to exhibit velocities
along the upper limits of normal and the paretic lid along the middle to lower portion of the normal range.

A comparison of the gain in amplitude (ratio between eyelid and target amplitudes) shows that the affected eyelid is consistently smaller. The unaffected eyelid had a gain that varied between 0.91 and 1.05 in the downward direction and between 0.88 and 0.92 in the upward direction (Table 2). In contrast, the affected lid displayed downward saccadic gains of 0.65 to 0.68 and upward gains of 0.63 to 0.74.

The difference between the paretic and the unaffected amplitudes was highly significant in each of the three patients \( (P < .001; \ t\ test\ for\ paired\ data)\). With one exception, the same level of significance \( (P < .001; \ t\ test\ for\ paired\ data)\) was found between the paired peak velocities. The one exception (patient 5) failed to exhibit a statistically significant difference between eyelids for upward saccadic velocities, although the difference between eyelids in the downward direction was significant. This difference between the saccadic velocities of the paretic and the unaffected eyelid is summarized in Fig 8. The oblique line defines the points at which peak velocities from both eyelids would be equivalent. Peak velocities of the affected lid were diminished for nearly all but the slowest of saccades. The one exception can be seen as the open squares (patient 5) that were above the oblique line in the upper right quadrant.

The possibility that the paretic closing phase of the blink and the downward lid saccade were kinematically similar (since both are executed without significant EMG activity in the OOCs) led to a comparison of peak velocities for each of these movements. It was found that the peak velocities for downward lid saccades among all patients were consistently greater than the peak velocities of the closing phase. Figure 9 shows a patient displaying downward peak velocities for both blinks and lid saccades. The difference between the peak velocities of the closing phase of the blink and downward lid saccades was statistically significant \( (P < .001\ by\ analysis\ of\ covariance)\).

**COMMENT**

It is generally believed that the upper eyelid moves normally on downward gaze in patients with facial paralysis. The present study shows that our patients had peak velocities for downward lid saccades that fell within the range observed in normal subjects. However, paired comparisons between the affected and unaffected eyelid shows that the paretic eyelid movement has a consistently smaller amplitude, resulting in a downward saccadic lid lag. We observed a similar phenomenon in patients receiving botulinum to the OOC for hemifacial spasm, and others have described lid lag in patients with facial nerve palsies due to Guillain-Barré syndrome.

Indeed, this decrease in amplitude might support the contention that the OOC actively participates in the execution of downward lid saccades. However, we believe that the passive mechanism proposed by Kenward and Smyth is sufficient to account for the ballistic trajectory of the downward lid saccade and the findings in this study are consistent with such a mechanism.

A brief review of the pertinent part of the anatomy shows that movement of the upper eyelid is influenced by several interconnecting structures that oppose the upward force of the LPS (Fig 10). Whitnall's ligament is a transversely oriented fibrous sling in the anterosuperior orbit. It functions as a suspensory ligament and fulcrum for the LPS and the levator aponeurosis. When the eyelids are closed, Whitnall's ligament forms a straight line between its medial and lateral horns; when the eyelids are elevated, tension is increased as the ligament is bowed posteriorly.

The levator aponeurosis, a tendinous expansion of the LPS, extends from the anterior face of the tarsus to Whitnall's ligament. The medial horn atta-
Fig 9.—Comparison of the peak velocities and amplitudes for downward saccades and closing phase of the blink obtained from patient 3. Regression lines and equations are based on these data alone. Open circles represent blinks; closed circles, saccades.

Fig 10.—Schematic depiction of the elastic structures thought to be responsible for the downward movement of the eyelid in the absence of orbicularis activity. When the lid is elevated in the open position or in extreme upward gaze, tensions along the orbicularis oculi (not shown), Whitnall’s ligament, the canthal-tarsal complex, and Lockwood’s ligament are increased. Sudden inhibition of the levator palpebrae releases this stored energy and the lid drops (see text).

qesses loosely to the posterior portion of the medial canthal tendon. To the extent that the LPS, acting through the aponeurosis, pulls on the upper arms of the canthal tendons and the tarsal plate, tension along the entire tarsal-canthal complex presumably should also increase.

Lockwood’s ligament, a transverse fibrous sling extending from the inferomedial to the inferolateral walls of the anterior orbit, functions as a suspensory structure and a check ligament for the inferior rectus muscle. Fibrous extensions of Lockwood’s ligament insert onto the inferior margin of the tarsus, the marginal portion of the OOC, the overlying skin, and the inferior orbital septum.2,3,5,6 Upward eye movements are accompanied by slight lateral and upward movements of the lower lid.7 The upward movement of the lower lid is presumably driven by the effect of the LPS on the canthal tendons. Likewise, contraction of the inferior rectus in downward gaze pulls down on the canthal tendons and tarsus through its connections with the retractors. Thus, as suggested by Renard and Smyth,21 “multiple parallel elastic elements” would seem to accurately describe the functional anatomy of the eyelid.

The reduction in downward saccadic amplitude of the affected eyelid reported in the present study indicates that an otherwise normally functioning OOC might also play a role in the execution of a downward lid saccade. However, because the EMG activity in the OOC is quiescent during the normal and the paretic downward lid saccade, we propose that the OOC functions not as an agonist, but rather, as a passive participant. There are several ways in which paresis of the OOC may slow the downward lid saccade. For example, the resting tone of the OOC in normal subjects may passively oppose the LPS or may contribute to the frictional drag of the eyelid against the eyeball. In either case, laxity of the OOC might reduce the downward tension on the upper eyelid. It is also possible that the failure to blink effectively in patients with facial paresis may lead to contracture and thickening of the LPS, which would resist the passive downward elastic forces on the eyelid.

Consider then how these structures, acting in concert, generate a downward lid saccade (Fig 10). When the lid is elevated in the open position or in upward gaze, the tension along Whitnall’s ligament, the tarsal-canthal complex, Lockwood’s ligament, and the OOC increases. Sudden inhibition of the LPS releases this stored energy and, like the bowstring on a crossbow, the lid is pulled down.

If the movement of the upper eyelid during a downward lid saccade is, in fact, completely passive, then there should be differences between the kinematics of a downward and upward lid saccade. Prior studies have failed to show a consistent kinematic distinction21,22 between the two; however, Evinger et al21 showed that the velocity-amplitude relationships for upward lid saccades displayed a linear regression whereas downward lid saccades were best described by a slow saturating power function (Table 1).

In this regard, it should be noted that despite the profound degree of OOC weakness among our patients, we still observed some movement of the eyelid during a blink; in some cases, eyelid motion occurred in the absence of any apparent EMG activity in the OOC and in others, eyelid motion preceded the onset of the EMG in the OOC. Analysis of the velocity-amplitude relationship for the closing phase of the blink (characterized by a linear regression in normals) fell off the main sequence and displayed a saturation effect akin to the downward lid saccade (Fig 2, left, and Table 1). This suggests that the eyelid movements we observed in our patients during the closing phase of the blink were, to a variable extent, the result of passive forces on the eyelid. Thus, the closing phase of the paretic blink takes on some of the biomechanical features of the downward lid saccade.

If the closing phase of the blink and the downward lid saccade in patients with facial paresis are biomechanically analogous, then the kinematics of both should be similar. However, we consistently observed slower peak velocities for the closing peak of the blink than for the downward lid saccade (Fig 9). This apparent inconsistency can be explained by considering the associated
actions of the extraocular muscles during blinks and saccades. Prior studies have shown that the extraocular muscles (including the superior rectus) will cocontract during a blink, whereas contraction of the inferior rectus with reciprocal inhibition of both the LPS and the superior rectus occurs with downward lid saccades. Anatomically, the superior rectus and LPS are interconnected by a common sheath and intermuscular fibrous tissue. Thus, in the closing phase of the paretic blink, inhibition of the LPS with a contracting superior rectus, moving in opposite directions, should reduce the peak velocities of the closing phase to a greater extent than the downward lid saccade (where both the superior rectus and LPS are reciprocally inhibited).

We also observed that the peak velocities of the unaffected eyelid movement were increased above the normal main sequence among two of three subjects who underwent bilateral recordings (Fig 2, right). The increase presumably reflects enhanced EMG activity in the OoC on the unaffected side. While the numbers of patients were too small to make any definitive conclusions, this finding suggests that there may be an increased drive on both OoC muscles to compensate for the weakening of the affected eyelid. Normal subjects show similar adaptive modifications of the blink reflex. These findings may represent an expression of Hering's law as it pertains to the OoC.

An analysis of the eyelid movements in normal subjects and in patients with facial paresis shows that the ballistic features of the downward lid saccade can be explained on the basis of passive forces without invoking a burst tonic mechanism. The eyelid, however, appears to represent a unique system. Quantitative analysis of the eyelid movements in a variety of pathological states might, in the future, have clinical applications.

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