

Restoration of Blink Symmetry Following Unilateral Facial Paralysis

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Abstract

Electrical stimulation of the orbicularis oculi muscle has been demonstrated to cause eyelid closure in an animal model of surgically induced facial nerve paralysis. This study investigated the use of electromyography (EMG) recorded from a healthy orbicularis oculi muscle as a signal for triggering delivery of electrical stimulation to the contralateral eyelid in a unilateral model of facial nerve paralysis. The goal was to evaluate whether such triggering can restore blink symmetry. The timing and magnitude of closure achieved by a reflex blink on the normal side and the corresponding electrically stimulated blink on the paralyzed side were compared and found to be quite similar. This represents the first quantitative analysis of triggered electrical stimulation for symmetric blink restoration.

1 Introduction

Dysfunction of the seventh cranial (facial) nerve leads to the inability to close the eye during both reflex and voluntary blinking [1]. Electrical stimulation of the orbicularis oculi (OO) has been shown to generate eyelid closure in an animal model of surgically induced facial nerve paralysis and represents a potential treatment for this condition [2].

The vast majority of facial palsy cases are unilateral, paralyzing one side of the face while leaving the other side functional [1], [3]. The resulting lack of symmetry is a major cause of psychological distress [1], [4]–[6]. Of normal human facial movements, blinking function in particular exhibits marked bilateral symmetry [7]. The onset of eyelid movement in patients recovering from facial palsy retains this conjugacy [8]. Electrical stimulation of the paralyzed OO muscle can potentially restore near-normal blink dynamics [9]. Therefore, timing the onset of stimulation to the action of the healthy contralateral eyelid may have the

potential to restore this blink conjugacy and a semblance of lost symmetry resulting in cosmetic, psychological, and even functional benefit.

The use of contralateral control signals for the electrical stimulation of unilaterally paralyzed axial muscles was originally proposed by Zelear and Dedo [10]. Since then, other researchers have investigated the use of normal OO muscle contraction to trigger stimulated contraction of the contralateral pair [11], [12]. They have reported the ability to restore bilateral symmetry, but have provided no quantitative evidence. EMG activity has been investigated as a control signal for many neural prosthetic applications [13]. This study utilized EMG activity recorded from the healthy orbicularis oculi muscle as a trigger signal for the delivery of electrical stimulation to the contralateral pair in a unilateral rabbit model of facial paralysis, and quantified the resulting bilateral blink dynamics to assess the ability to restore blink symmetry.

2 Methods

A single rabbit underwent surgery to induce unilateral facial paralysis by sectioning the seventh cranial nerve and removing a 7 mm portion of the zygomatic branch, which innervates the OO muscle. Concurrently, a chronic electrode was implanted with bilateral leads that were routed across the eyelids placing contacts near the medial and lateral canthi.

After four weeks of paralysis, the lead on the healthy side was used to record the OO EMG response during reflex eyelid closure induced by a puff of air directed at the cornea. The OO EMG was amplified by a gain of 10,000 over the bandwidth 30-3000 Hz and the envelope was extracted by full-wave rectifying the signal and then lowpass filtering it with a cutoff frequency of 14.5 Hz. The OO EMG envelope was compared to a threshold value and a trigger

signal was generated when the threshold was crossed (see Figure 1). In response to the trigger signal, the lead on the paralyzed side was used to deliver a train of stimulation pulses to the contralateral paralyzed OO. This stimulus train consisted of a 50 Hz square wave with duration of 100 ms. The stimulus amplitude was tuned such that the magnitude of observed closure approximated that of the reflex closure on the healthy side. The movement of both eyelids was recorded simultaneously using a high-speed digital video camera with an integrated mirror setup and image processing software was used to quantify eyelid closure.

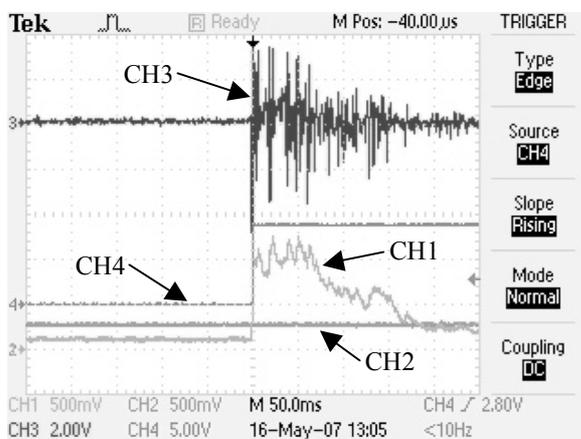


Figure 1 - EMG threshold signal generation. (CH3) EMG of a reflex blink bandpass filtered between 30-3000Hz and amplified by 10,000. (CH1) Envelope of EMG signal from CH3 produced by full wave rectification and lowpass filtering at 14.5 Hz. (CH2) Threshold level for trigger signal. (CH4) Trigger signal.

3 Results

EMG signals were robust with high signal to noise ratio and the delay between the onset of EMG activity and the activation of the trigger signal was minimal (< 5 ms). Stimulation of the paralyzed OO in response to EMG activity recorded from the healthy contralateral OO resulted in a symmetric looking blink. Sample frames from a recording of triggered eyelid stimulation are shown in Figure 2. From high speed video there is evidence of slight lag in the closing of the paralyzed eyelid, as well as slight mismatch in the residual opening of the palpebral fissure at peak closure, however these differences were not noticeable in real time. Plots of the kinematics for both eyelids during reflex blinking with no triggered stimulation, during untriggered electrical stimulation, and

during triggered stimulation based on a reflex blink are shown in Figure 3. There are slight differences in the overall amplitude of closure as well as the closing velocity between the two sides during triggered stimulation, with the electrically stimulated side demonstrating less overall closure and slower closing velocity than the normal side. The onset of eyelid movement resulting from electrical stimulation, however, was well matched with the onset of the reflex movement. Despite the differences in blink kinematics, the blink profiles were quite similar and in real time movement appeared conjugate.

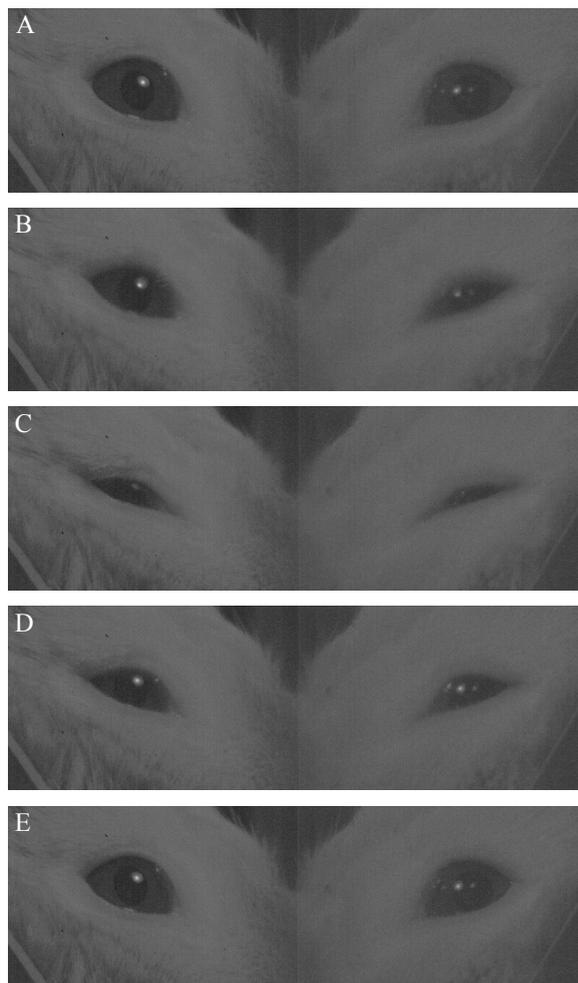


Figure 2 – Contralateral EMG triggered blink response. Images of eyelids recorded (A) prior to air puff and EMG triggered stimulation, (B) during eyelid closing, (C) at the peak of closure, (D) during eyelid opening, and (E) immediately after opening. The eye on the right side of the images (rabbit's left eye) was healthy and stimulated with an air puff to create a reflex blink, while the eye on the left side of the images (rabbit's right eye) was electrically stimulated based on a trigger produced by contralateral EMG.

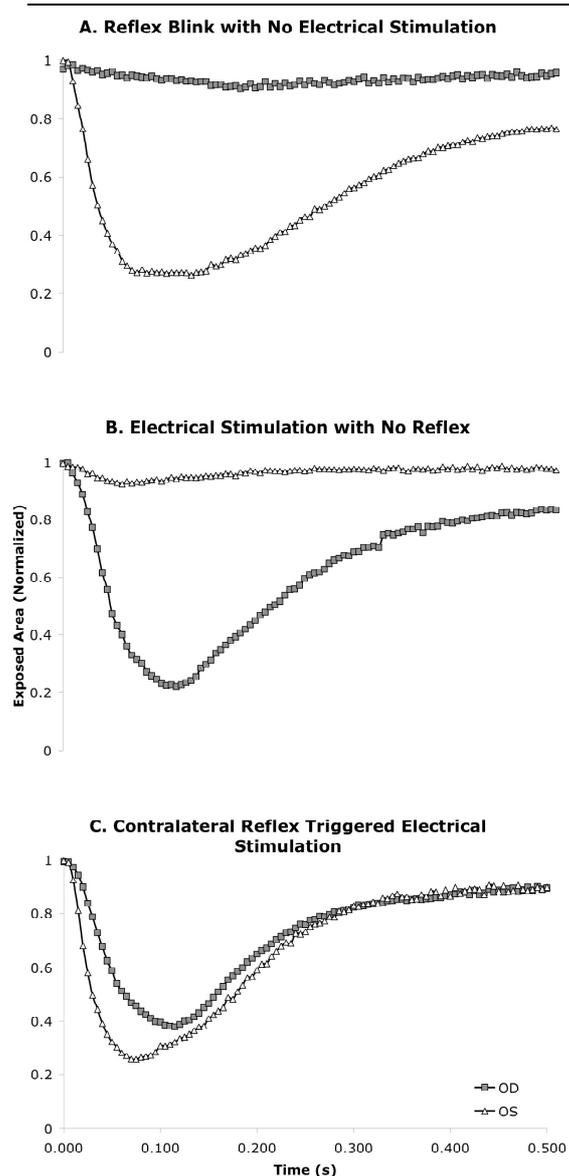


Figure 3 – Bilateral kinematics for EMG triggered stimulation. (A) Reflex blink initiated by an air puff in healthy eyelid (OS) with no electrical stimulation of paralyzed eyelid (OD). (B) Electrical stimulation of OD with no reflex initiation in OS. (C) Electrical stimulation of OD triggered by EMG recorded from OS during an air puff initiated reflex blink. Both eyes were recorded synchronously and movement was plotted as normalized exposed area, such that 1 corresponds to the eyelid being completely open and 0 corresponds to being completely closed. Initiation of movement was plotted at Time = 0.0s. Each plot is based on the average of five trials.

4 Discussion and Conclusions

Electrical stimulation of the denervated orbicularis oculi muscle triggered by EMG activity recorded in its healthy contralateral counterpart successfully restored symmetric blink function in a unilateral rabbit model of facial nerve paralysis.

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