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Blink reflex in patients with postparalytic facial syndrome and blepharospasm: Trigeminal and auditory stimulation

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HIGHLIGHTS

- We designed a prospective study of trigeminal and auditory blink reflex responses in postparalytic facial syndrome and blepharospasm.
- We observed enchancements in blink reflex circuits in both, probably via peripheral pathways in postparalytic facial syndrome, but via central mechanisms in blepharospasm.
- We also observed that enhanced excitability of blink reflex circuit is evoked by auditory stimulation in addition to trigeminal stimulation.

ABSTRACT

Objective: The enhancement of blink reflex (BR) excitability was shown in patients with postparalytic facial syndrome (PFS) and essential blepharospasm (EB). We prospectively investigated patients with PFS and EB whether BR alterations demonstrated by trigeminal stimulation will similarly be observed upon auditory stimulation.
Methods: Fifteen patients with PFS, 15 patients with EB, and 30 healthy volunteers were involved. Electrically stimulated trigeminal BR and auditory BR were studied bilaterally.
Results: The mean R2 amplitude and duration values were highest in EB patients, being significantly higher than PFS patients ($p < 0.05$) and control group ($p < 0.01$). The mean R2 duration in PFS patients were also significantly longer in compared to control group ($p = 0.025$). EB patients showed a higher mean *R* (auditory) amplitude and duration than PFS patients ($p < 0.05$) and controls ($p < 0.04$). The mean *R* (auditory) duration was longer on symptomatic side of PFS patients in compared to controls ($p = 0.05$).
Conclusions: We observed that there is an enhanced excitability of BR circuit in postparalytic facial syndrome and essential blepharospasm, which could be evoked by auditory stimulation in addition to trigeminal stimulation.
Significance: The enhanced excitability in patients with EB and PFS probably originates from the final common pathway of BR circuit, namely facial motor or premotor neurons.
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1. Introduction

The neuronal network underlying blink reflex is rather complex given the diversity of multimodal sensory inputs, and yet possesses many unresolved questions despite many comprehensive researches (Courville, 1966; Takeuchi et al., 1979; Travers and Norgren, 1983; Takada et al., 1984; Fort et al., 1989). Neurophysiological studies possess an important role to understand better the complex neuronal network of blink reflex and multimodal sensory projections. Disorders causing excitability changes in blink reflex circuits and responses are therefore being investigated to better understand this network, and in turn, the delineation of pathways involved in blink reflex responses will help understanding the underlying pathology of such disorders. Postparalytic facial syndrome (PFS) and idiopathic-essential blepharospasm (EB) are two different clinical entities causing changes in blink reflex circuits. PFS is a complication following peripheral facial paralysis, and characterised by muscle synkinesis, myokymia, and involuntary mass contractions of muscles on the affected side of the face (Valls-Sole, 2007). Benign essential blepharospasm is a focal dystonia characterized by sustained, involuntary blinking and closure of the eyelids typically caused by spasms of the orbicularis oculi muscles (Fahn, 1988). Postparalytic facial syndrome was shown to be resulted from motoneuronal excitability in facial nucleus secondary to maladaptive mechanisms due to enhancement of the blink

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reflex gain (Berardelli et al., 1985; Montserrat and Benito, 1988; Tolosa et al., 1988; Valls-Sole et al., 1991, 1992; Syed et al., 1999). This enhanced gain of blink reflex, caused by the incomplete eye closure, was explained by the sensitization of bilateral blink reflex polysynaptic pathways to inputs from the trigeminal nerve on the symptomatic side, and considered as one of the maladaptive mechanisms leading to involuntary muscles contractions, PFS, or even blepharospasm (Berardelli et al., 1985; Chuke et al., 1996; Huffman et al., 1996; Baker et al., 1997; Pastor et al., 1998; Miwa et al., 2002). It has been reported that in some cases, the ophthalmic diseases, especially corneal irritation could also trigger the development of blepharospasm (Grandas et al., 1988). In EB, on the other hand, interneuronal excitability in brainstem circuits was suggested to reflect alterations in descending projections to inhibitory neurons, which are indirectly modulated by the pallido-thalamo-cortical motor circuit (Vitek, 2002).

Actually, previous studies conducted by the paired pulse paradigm have shown that the enhancement of blink reflex excitability existed in various movement disorders, notably those involving the cranio-cervical area, hemifacial spasm, some cases of peripheral facial paralysis, and PFS (Berardelli et al., 1985; Tolosa et al., 1988; Valls-Sole and Tolosa, 1989; Eekhof et al., 1996; Syed et al., 1999; Oge et al., 2005). Loss of inhibition on brainstem reflex circuits was shown in EB patients by blink reflex studies (Tolosa et al., 1988; Valls-Sole et al., 1991). Similarly, enlargement of R2 responses were reported in patients with blepharospasm and on the symptomatic sides of patients with hemifacial spasm and PFS, which was atributed to the hyperexcitability of the facial nucleus (Berardelli et al., 1985; Tolosa et al., 1988; Valls-Sole and Tolosa, 1989; Valls-Sole et al., 1992). Moreover, it was rather recently highlighted that the reflex pathways in the generation and control of eyelid responses could also be evoked by different stimuli of trigeminal, auditory, vestibular or visual origins (Morcuende et al., 2002).

On this context, we designed a prospective study of blink reflex responses to investigate if blink reflex alterations demonstrated by trigeminal stimulation will similarly be observed upon auditory stimulation in patients with postparalytic facial syndrome, which is known to cause an increase in blink reflex circuits via peripheral pathways, and blepharospasm, which similarly shows an enhancement in blink reflex circuits, but via central mechanisms.

2. Methods

This study was carried out in our EMG department during the years 2006–2009, and consisted of 15 patients with postparalytic facial syndrome and synkinesia, 15 patients with essential blepharospasm, and 30 healthy volunteers served as control subjects. Patients were involved to the study consecutively. None of them had primary hemifacial spasm, history of any other movement disorders or extrapyramidal diseases, and neurological examinations were otherwise normal. None of the patients had botilinum toxin treatment before. The grade of facial paralysis was lower than three according to House-Brackmann grading (House and Brackmann, 1985). All participants signed a consent form and our study was approved by the Ethical Committee of our Institution.

An eight-channel electromyography (EMG) apparatus was used (Nihon – Kohden), and all instruments were calibrated before data collection. The electrophysiological measurements were assessed with the patients relaxed in sitting position in a quiet semidarkened room at ambient temperature. The principal investigator (M.E.K.) explained all procedures to the subjects before testing. Before applying electrodes, the skin was cleaned by wiping with 95% ethanol to aid electrode adhesion and increase conductivity. The electrodes used were pre-gelled Ag/AgCl surface recording electrodes. Care was taken to avoid potential visual or auditory prepulse stimuli (Valls-Sole et al., 1997).

Electrically stimulated trigeminal blink reflex (BR) and auditory blink reflexes (ABR) were studied bilaterally. A pair of surface recording electrodes was placed over the orbicularis oculi muscles on the lower eyelid in both sides, and a pair of stimulating electrodes was attached to the supraorbital nerve, with the cathode at the supraorbital notch, and the anode 3 cm away. The subjects were asked to keep their eyes slightly closed and fixed in neutral position. Each participant received the same amount and type of stimuli. Every stimulus was given three times consecutively, and the mean values for each variable were enrolled in the study. Auditory blink reflexes were elicited at the same experimental procedure by sudden onset acoustic stimuli at 105 DB tone delivered bilaterally through earphones. Consecutive stimuli were given at intervals of two to three minutes to avoid habituation. Filter setting was 3 kHz high cut, and 20 Hz low cut, with sensitivity of 0.2 mV and sweep speed of 20 ms/s. Then, the analysis of bilateral R1 and R2 latency (ms) elicited by electrical stimulation of the ipsilateral supraorbital nerve, and the R2c latency elicited by electrical stimulation of the contralateral supraorbital nerve, R2 amplitude (μV), R2 duration (ms), and the mean R2c/R2 ratio were made upon electrically evoked trigeminal blink reflex. The analysis of acoustically evoked blink reflex consisted of the latency, amplitude and the duration of the blink response.

The demographic data and electrophysiological measures obtained by electrical and auditory stimulation were compared using Pearson chi-square test, unpaired *t*-test and the Kruskal Wallis test. The correlation between demographical and electrophysiological data was explored by using Pearson and linear correlation analysis. The mean R2c/R2 ratio values for control subjects and patients were also compared using the unpaired *t* test. For statistical analysis, the mean values evoked from both right and left sides were used in control group and EB patients, while the responses obtained from both symptomatic and asymptomatic sides were used for statistical analysis in patients with PFS. We also performed ANOVA with post-hoc multiple analysis as Bonferroni correction test and Waller-Duncan analysis in order to avoid type I/type II error. The threshold level for statistical significance was established at $p \leqslant 0.05$. All values were reported as mean and 95% confidence intervals or percentiles.

3. Results

A total of 15 patients with postparalytic facial syndrome, 15 patients with essential blepharospasm, and 30 healthy volunteers as control subjects were enrolled in our study. The demographic features of PFS, EB patients and control group was given in Table 1. The gender distribution and the mean age were not statistically different among three groups. The electrophysiological measures were not correlated with gender or age, neither ($p > 0.05$ for all).

Both trigeminal and auditory blink reflex responses could be evoked in all patients and controls, as summarized in Table 2. Examples of original recordings of BR and ABR responses typical

Table 1
The demographic features of study population.

Groups	Gender		Age (yrs)	
	Males *n* (%)	Females *n* (%)	Mean ± s.d.	Min–max
PFS patiens	5 (33.3)	10 (66.7)	43.5 ± 8.9	30–59
EB patiens	8 (66.3)	7 (46.7)	49.6 ± 11.6	27–66
Control group	12 (40)	18 (60)	41.6 ± 10.8	25–65
p Values[a]	0.056		0.417	

[a] Pearson chi-square test.

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