Somatosensory-evoked blink reflex in peripheral facial palsy

Sarah S. El-Tawab, Emmanuel K.A. Saba

Department of Physical Medicine, Rheumatology and Rehabilitation, Faculty of Medicine, University of Alexandria, Alexandria, Egypt

Correspondence to Emmanuel K.A. Saba, MD, Department of Physical Medicine, Rheumatology and Rehabilitation, Faculty of Medicine, University of Alexandria, Ramel Station, Alexandria, Egypt
Tel: +20 354 45423;
e-mail: emadaziz55@yahoo.com

Received 19 November 2014
Accepted 29 March 2015

Egyptian Rheumatology & Rehabilitation
2015, 42:94–99

Introduction
Somatosensory-evoked blink reflex (SBR) is an eye blink response obtained from electrical stimulation of peripheral nerves or the skin area of the body away from the face.
Aim
The aim of this study was to investigate the presence of SBR in peripheral facial palsy (PFP) and its relation with clinical and electrophysiological changes occurring in PFP as compared with postfacial syndrome (PFS).
Setting and design
This was a single-center, public hospital-based electromyography laboratory study. It was designed as a cross-sectional examination of consecutive patients with PFP and PFS and apparently healthy volunteers.
Patients and methods
The study included 25 patients with PFP, 25 patients with PFS, and 31 healthy volunteers. Facial nerve motor conduction, trigeminal blink reflex, and SBR were studied.
Statistical analysis
Quantitative data were compared using the Mann–Whitney test and the Kruskal–Wallis test. Qualitative data were analyzed using Pearson's Chi-square test.
Results
SBR was elicited in 67.7% of controls, in 68% of PFS patients, and in 32% of PFP patients. In the PFP group, SBR was found on the nonparalytic side in 28% of patients with paralyzed side stimulation and in 24% of patients with healthy side stimulation. For the PFS group, SBR was found on the nonparalytic side in 48%. Bilateral SBR elicitability was higher than its unilateral elicitability.
Conclusion
SBR occurs in patients with PFP and PFS and in healthy individuals. It has no relation with the clinical and electrophysiological changes occurring in PFP and PFS. Increased brainstem interneurons excitability is not essential to generate SBR. The hypothetical sensory-motor gating mechanism could be responsible for SBR generation.

Keywords:
blink reflex, facial nerve, peripheral facial palsy, postfacial syndrome, somatosensory-evoked blink reflex

Egypt Rheumatol Rehabil 42:94–99

1110-161X

Introduction

Somatosensory-evoked blink reflex (SBR) is a blink reflex (BR) obtained through electrical stimulation of peripheral nerves or skin areas away from the face [1,2]. The presence of SBR was studied in various neurological diseases [3,4]. It has been reported that SBR is more frequent in postfacial syndrome (PFS) with synkinesis compared with other neurological diseases [5,6]. There are very few studies that have assessed SBR in peripheral facial palsy (PFP). The aim of the current work was to study the presence of SBR in PFP and its relation with the clinical and electrophysiological changes occurring in PFP as compared with PFS.

Patients and methods

This is a cross-sectional study comprising 25 patients with PFP, 25 patients with PFS, and 31 apparently healthy volunteers as a control group. The volunteers consisted of medical staff, their relatives, and patients' relatives. Clinical diagnosis of PFP was based on the presence of acute onset of weakness of the facial muscles due to unilateral dysfunction in the seventh cranial nerve [7]. Clinical diagnosis of PFS was based on the presence of movement triggered muscle spasms and activation of facial muscles together with voluntary or automatic activation. It occurs in most patients who have suffered from significant axonal degeneration after PFP [8]. Exclusion criteria included diabetes mellitus, endocrine disorders, metabolic disorders, rheumatological disorders, and neurological disorders including peripheral neuropathy. The study was explained to the participants and an informed consent was given by each. The study was approved by the Institutional Ethical Committee of the Faculty of Medicine, Alexandria University, Egypt.

All patients were subjected to the following:

(1) History taking, including demographic data and history of the present condition was performed.
(2) Clinical examination: the House–Brackmann (HB) grading scale was used to score the strength and function of facial muscles. It ranged from normal (grade I) to total paralysis (grade VI) on a six-grade scoring system [9].
(3) Electrophysiological studies were conducted on a Nihon Kohden Neuropack MEB-7102 mobile unit with a two-channel evoked potential/electromyography measuring system (Nihon Kohden Corp., Tokyo, Japan). The study included the following tests, which were performed for the patients and controls while they were seated comfortably in a quiet room at a temperature between 22 and 24°C.
    (a) Facial nerve electroneuronography (ENoG) was obtained by transcutaneous stimulation of the facial nerve behind the ear, below the mastoid process and behind the neck of the mandible. The compound muscle action potential (CMAP) was recorded from the nasalis muscle. The process was performed for the right and left facial nerves. Measurements, including latency (L), amplitude (peak to peak) of the facial nerve CMAP, and the percentage decrease in the CMAP amplitude on the symptomatic side compared with the contralateral asymptomatic (healthy) side, were ascertained to obtain the facial nerve ENoG percentage of degenerated fibers innervating the nasalis muscle [10].
    (b) BR was obtained by transcutaneous stimulation of the supraorbital nerve of one side and by recording the responses of both sides (ipsilateral R1 and R2 and contralateral R2). Two pairs of surface disc recording electrodes were placed on the right and left orbicularis oculi muscles. The shortest latency was taken into consideration [6].
    (c) SBR was obtained by applying a supramaximal transcutaneous electrical stimulus of 0.2 ms duration to the median nerve at the wrist, which evoked a brief twitch of the abductor pollicis brevis muscle. Stimulation of the median nerve was carried out on one side and by recording the responses of both sides (symptomatic and asymptomatic sides) with the same recording electrode montage of BR on orbicularis oculi muscles. Absent SBR was interpreted when there was no response detected with repeated stimulation of the median nerve, applied at least four times at intensities above three times the stimulus intensity adequate to produce twitches in the abductor pollicis brevis muscle, and when there was failure to elicit SBR. The shortest latency was taken into consideration. Responses evoked at or later than 75 ms following the stimulus were regarded as startle blink and were excluded [3].

Statistical analysis

Statistical analysis of data was performed using statistical package for the social sciences (SPSS, version 17; University of Cambridge computing service, London) software [11]. Descriptive measures (count, frequency, minimum, maximum, mean, and SD), as well as analytic measures (Mann–Whitney test, Kruskal–Wallis test, and Pearson's Chi-square test) were used. Statistical significance was assigned to any P value at 0.05 or lesser. The reference cutoff values of the electrophysiological studies were equal to mean + 2 SD for latency measurements.

Results

The present study included 25 patients with PFP [16 (64%) women and nine (36%) men]. Their mean age was 36.96 ± 10.65 years (range 25–59 years). Twenty-five patients with PFS were also included [13 (52%) women and 12 (48%) men]. Their mean age was 37 ± 16.19 years (range 18–63 years). There were 31 apparently healthy participants [20 (64.5%) women and 11 (35.4%) men] who served as the control group. Their mean age was 37.96 ± 11.63 years (range 21–65 years).

There were no statistically significant differences between patients and controls as regards sex ($\chi^2 = 1.093$, $P = 0.579$) or age ($K = 0.382$, $P = 0.826$).

In the PFP group, all patients had idiopathic PFP (Bell's palsy). The mean duration of illness was 3.40 ± 0.86 weeks (range 2–4 weeks), and their median HB scale was V (range III–VI). HB scale grade V was the most common grade present in 10 (40%) patients. The right side was affected in 16 (64%) patients.

As regards the PFS group, all patients were secondary to idiopathic PFP (Bell's palsy). The mean duration of their illness was 83.92 ± 169.69 weeks (range 6–700 weeks). Their median HB scale was III (range II–V). HB scale grade III was the most common grade present in 12 (48%) patients. The right side was affected in 15 (60%) patients. Synkinesis was present in 11 (44%) patients.

In PFP patients, SBR was elicited in six (24%) patients, in five (20%) patients by ipsilateral stimulation and in three (12%) patients by contralateral stimulation on recording the symptomatic side. SBR was elicited in

eight (32%) patients on recording the asymptomatic side, in six (24%) patients by ipsilateral stimulation and in seven (28%) patients by contralateral stimulation (Tables 1 and 2).

In PFS patients, SBR was elicited in 15 (60%) patients, in 12 (48%) patients by ipsilateral stimulation and in 10 (40%) patients by contralateral stimulation on recording the symptomatic side. SBR was also elicited in 16 (64%) patients on recording the asymptomatic side, in 12 (48%) patients by ipsilateral stimulation and in 12 (48%) patients by contralateral stimulation (Tables 1 and 2).

In the control group, SBR was elicited in 20 (64.5%) participants, in 17 (54.8%) participants by ipsilateral stimulation, and in 15 (48.4%) participants by contralateral stimulation on recording the right side. SBR was also elicited in 18 (58.1%) participants, in 16 (51.6%) participants by ipsilateral stimulation, and 14 (45.2%) participants by contralateral stimulation on recording the left side (Tables 1 and 2).

Table 1 Frequencies of somatosensory-evoked blink reflex elicitability in the patient and control groups on symptomatic side recording (right side for controls)

Study groups	*n* (%)	
	Ipsilateral stimulation	Contralateral stimulation
PFP (*n* = 25)	5 (20.0)	3 (12.0)
PFS (*n* = 25)	12 (48.0)	10 (40.0)
Control (*n* = 31)	17 (54.8)	15 (48.4)
χ^2	7.435	8.573
P	0.024*	0.014*

PFP, peripheral facial palsy; PFS, postfacial syndrome; *$P \leq 0.05$, significant.

Table 2 Frequencies of somatosensory-evoked blink reflex elicitability in the patient and control groups on asymptomatic side recording (left side for controls)

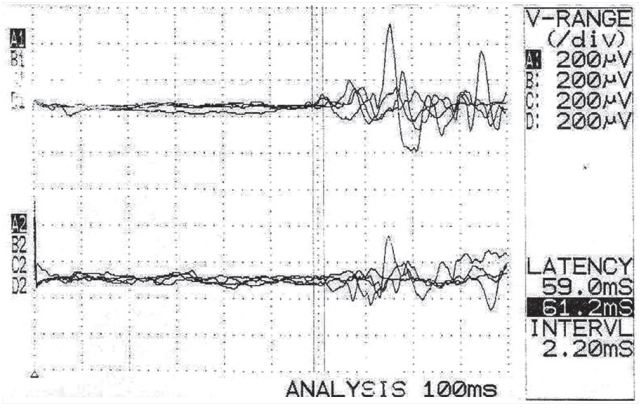
Study groups	*n* (%)	
	Ipsilateral stimulation	Contralateral stimulation
PFP (*n* = 25)	6 (24.0)	7 (28.0)
PFS (*n* = 25)	12 (48.0)	12 (48.0)
Control (*n* = 31)	16 (51.6)	14 (45.2)
χ^2	4.871	2.478
P	0.088	0.290

PFP, peripheral facial palsy; PFS, postfacial syndrome; *$P \leq 0.05$, significant.

SBR was elicited unilaterally and bilaterally (whatever the side of stimulation) among patients and control groups. However, the frequency of bilateral SBR elicitability among patients and controls was higher compared with the frequency of unilateral SBR elicitability (Table 3). When stimulation was performed from either side, the same SBR unilateral response or bilateral response was present in three (12%) patients in the PFP group and in five (20%) patients in the PFS group. In the control group, this occurred in 12 (38.7%) patients. There was no statistically significant difference between the three groups as regards the occurrence of the same SBR response unilaterally or bilaterally ($\chi^2 = 5.738$, $P = 0.057$). Illustrations of SBR obtained in a PFS patient is shown in Fig. 1.

The frequency of SBR elicitability in the PFP group was significantly lower compared with its frequency in the PFS and control groups. There were statistically significant differences between the three groups as regards the elicitability of SBR recorded on the symptomatic side of the face. The frequency of SBR elicitability recorded on the symptomatic side of the face in the PFP group (24%) was significantly lower compared with its frequency in the PFS (60%) and control (64.5%) groups ($\chi^2 = 10.362$, $P = 0.006$). In contrast, the frequency of SBR elicitability recorded on

Figure 1



Sample tracings showing somatosensory-evoked blink reflex recording of a postfacial syndrome patient from ipsilateral stimulation to the symptomatic side. The upper traces from symptomatic side recording, and the lower traces from asymptomatic side recording.

Table 3 Frequencies of somatosensory-evoked blink reflex elicitability among patients and controls (whatever the side of recording or stimulation)

State and side of SBR elicitability	*n* (%)			χ^2	*P*
	PFP (*n* = 25)	PFS (*n* = 25)	Control (*n* = 31)		
SBR elicitability (per participant)	8 (32.0)	17 (68.0)	21 (67.7)	9.056	0.011*
SBR side					
Unilateral SBR elicitability	2 (8.0)	3 (12.0)	4 (12.9)	9.193	0.056
Bilateral SBR elicitability	6 (24.0)	14 (56.0)	17 (54.8)		

PFP, peripheral facial palsy; PFS, postfacial syndrome; SBR, somatosensory-evoked blink reflex; *$P \leq 0.05$, significant.

the asymptomatic side of the face in the PFP group (32%) was not significantly lower compared with its frequency in the PFS (64%) and control (58.1%) groups ($\chi^2 = 5.903$, $P = 0.052$).

Electrophysiological parameters of facial nerve ENoG, BR, and SBR in the PFP, PFS, and control groups are tabulated (Table 4). There were no statistically significant differences in SBR latency between symptomatic and asymptomatic side recording with ipsilateral or contralateral stimulation. There were statistically significant differences between the PFP and PFS groups as regards facial nerve ENoG parameters and percentage of degeneration (Table 4).

There were no statistically significant differences between PFP patients with prolonged R1 and those with absent R1 as regards the elicitability of SBR ipsilaterally and contralaterally (Table 5). This was also applied for the status of symptomatic side R2 ipsilateral among PFP patients. This was also applied for PFS (Table 6).

There were no statistically significant differences in facial nerve CMAP latency, amplitude, and ENoG percentage of degeneration between PFP patients with elicited SBR (on either side) and those with unelicitable SBR ($P > 0.05$). There were no statistically significant differences in facial nerve CMAP latency, amplitude, and ENoG percentage of degeneration between PFS patients with elicited SBR (on either side) and those with unelicitable SBR ($P > 0.05$). There were no statistically significant differences between HB grades and the elicitability of SBR (on either side) among PFP ($Z = -0.495$, $P = 0.621$) and PFS patients ($Z = -0.312$, $P = 0.755$).

Discussion

SBR was reported for the first time in patients with Miller Fisher syndrome and later in various neurological diseases with a known increased excitability of the blinking pathway. This included hemifacial spasm, Parkinson's disease, dystonia, stroke, and also in some forms of peripheral neuropathy [3,4,12]. The nature of SBR has been a point of concern for the researchers: is it a variant of the startle BR or one of the release phenomena of the basically programmed BR? [1,3]. Most of the researchers agreed that SBR is not a startle BR based on the latency and duration of SBR, which were shorter; in addition, the startle BR usually occurs together with generalized startle jerks of the body and limbs [3].

In the current work, SBR elicitability was most frequently observed in patients with PFS (68%), whereas SBR was elicited only in 32% of patients in

Table 4 Comparison of peripheral facial palsy, postfacial syndrome, and the control group as regards facial nerve electroneuronography, blink reflex, and somatosensory-evoked blink reflex parameters

Electrophysiological parameters of facial nerve	Mean ± SD			Test of significance	*P*
	PFP	PFS	Control		
CMAP L (ms)	3.95 ± 1.08	4.60 ± 1.60‡	3.45 ± 0.64	*K* = 13.307	0.001*
CMAP amp (mV)	0.52 ± 0.27†	1.05 ± 1.19‡	4.26 ± 1.51	*K* = 52.642	<0.0001*
Facial ENoG percentage of degeneration (%)	61.21 ± 24.15	71.02 ± 25.82	NA	*Z* = −2.019	0.043*
Blink reflex					
Symptomatic side stimulation					
R1 L (ms)	12.80 ± 0.81†	11.21 ± 1.77	10.28 ± 0.88	*K* = 7.499	0.024*
R2 ipsi L (ms)	36.20 ± 1.00†	37.32 ± 5.02‡	30.36 ± 2.87	*K* = 21.207	<0.0001*
R2 cont L (ms)	31.31 ± 5.30	33.64 ± 4.43	32.95 ± 2.95	*K* = 5.438	0.066
Asymptomatic side stimulation					
R1 L (ms)	9.99 ± 0.81	9.89 ± 0.75	10.09 ± 0.53	*K* = 2.740	0.254
R2 ipsi L (ms)	31.39 ± 1.45	32.15 ± 3.89	30.33 ± 2.11	*K* = 4.845	0.089
R2 cont L (ms)	33.56 ± 3.74	38.80 ± 6.67‡	31.54 ± 2.40	*K* = 12.112	0.002*
SBR					
Symptomatic side recording					
SBR ipsi stim (ms)	64.08 ± 2.60	62.49 ± 4.96	58.03 ± 8.84	*K* = 2.198	0.333
SBR cont stim (ms)	64.66 ± 4.16	62.35 ± 5.28	59.34 ± 7.09	*K* = 1.891	0.389
Asymptomatic side recording					
SBR ipsi stim (ms)	59.55 ± 5.74	54.25 ± 7.04	57.73 ± 8.69	*K* = 2.775	0.250
SBR cont stim (ms)	62.25 ± 5.10	61.48 ± 6.87	59.52 ± 9.53	*K* = 0.018	0.991

amp, amplitude; CMAP, compound muscle action potential; cont, contralateral; ENoG, electroneuronography; ipsi, ipsilateral; *K*, Kruskal–Wallis test; L, latency; PFP, peripheral facial palsy; PFS, postfacial syndrome; SBR, somatosensory-evoked blink reflex; stim, stimulation; *Z*, Mann–Whitney test; †Statistical significance between the PFP group and the control group ($P < 0.0001$); ‡Statistical significance between the PFS group and the control group ($P < 0.0001$); *$P \leq 0.05$, significant.

Table 5 Association between somatosensory-evoked blink reflex and blink reflex in symptomatic and asymptomatic sides of patients with peripheral facial palsy

SBR elicitability	*n* (%)			χ^2	*P*
	R1 normal[a] (*n* = 0)	R1 prolonged[a] (*n* = 3)	R1 absent[a] (*n* = 22)		
SBR on symptomatic side	0	1 (33.3)	5 (22.7)	0.163	0.687
SBR on asymptomatic side	0	1 (33.3)	7 (31.8)	0.003	0.958
SBR elicitability (on any side)	0	1 (33.3)	7 (31.8)	0.003	0.958
	R2 normal[a] (*n* = 1)	R2 prolonged[a] (*n* = 2)	R2 absent[a] (*n* = 22)		
SBR on symptomatic side	0	1 (50.0)	5 (22.7)	1.077	0.584
SBR on asymptomatic side	0	1 (50.0)	7 (31.8)	0.769	0.681
SBR elicitability (on any side)	0	1 (50.0)	7 (31.8)	0.769	0.681

R1, blink reflex R1 response; R2, blink reflex R2 ipsilateral response; SBR, somatosensory-evoked blink reflex; [a]Results for symptomatic side; *$P \leq 0.05$, significant.

Table 6 Association between somatosensory-evoked blink reflex and blink reflex in symptomatic and asymptomatic sides of patients with postfacial syndrome

SBR elicitability	*n* (%)			χ^2	*P*
	R1 normal[a] (*n* = 7)	R1 prolonged[a] (*n* = 4)	R1 absent[a] (*n* = 14)		
SBR on symptomatic side	5 (71.4)	2 (50)	8 (57.1)	0.595	0.743
SBR on asymptomatic side	5 (71.4)	2 (50)	9 (64.3)	0.508	0.776
SBR elicitability (on any side)	5 (71.4)	3 (75)	9 (64.3)	0.217	0.897
	R2 normal[a] (*n* = 7)	R2 prolonged[a] (*n* = 4)	R2 absent[a] (*n* = 14)		
SBR on symptomatic side	5 (71.4)	2 (50)	8 (57.1)	0.595	0.743
SBR on asymptomatic side	5 (71.4)	2 (50)	9 (64.3)	0.508	0.776
SBR elicitability (on any side)	6 (85.7)	2 (50)	9 (64.3)	1.694	0.429

R1, blink reflex R1 response; R2, blink reflex R2 ipsilateral response; SBR, somatosensory-evoked blink reflex; [a]Results for symptomatic side; $P \leq 0.05$, significant.

the PFP group. This finding is in accordance with that in previous published studies [5,6].

A finding in concordance with lesion severity was found in the PFS group, in which SBR elicitability was much lower in patients whose R1 and R2 responses in trigeminal BR were absent, compared with those with normal or prolonged R1 and R2 responses. These findings could be possibly explained by the existence of a common premotor mechanism of SBR and R2 response [1]. Two points are against this speculation. First, the current study could not find this in the PFP group, which is attributed to the fact that the majority of these patients had absent R1 and R2 responses (more severe facial palsy). Second, the lack of a statistically significant difference between facial palsy severity, measured clinically with the HB scale and electrophysiologically by calculating the percentage of degenerated facial nerve axons, and the elicitability of SBR.

An expected finding in the current study was the presence of SBR elicitability on the asymptomatic (healthy) side of the face. SBR was present in 28% of patients on symptomatic side stimulation and in 24% of patients on asymptomatic (healthy) side stimulation in the PFP group. In contrast, for the PFS group, SBR was found in 48% of patients on both symptomatic and asymptomatic sides' stimulation. This expectation came from previous clinical and electrophysiological observations [6,8,13].

Clinically, the increased rate of blinking, eyelid narrowing, and blepharospasm on the healthy side of the face were previously documented. Electrophysiological published data including trigeminal BR abnormalities on the healthy side were also observed in the form of enhancement of BR excitability [5,14–16]. The physiological background of the motor hyperexcitability has been described as enhanced BR gain as a bilateral compensatory mechanism of unilateral facial weakness [17–19]. One mechanism had been discussed for these results. The lack of appropriate wetting of the cornea could generate an increased number of noxious sensory input causing repeated activation and sensitization of the reflex [18]. It is worth mentioning that, in this work, bilateral SBR elicitability was higher than unilateral SBR elicitability among all the studied groups (i.e. PFP, PFS, and controls).

In this study, SBR was elicited in 67.7% of normal controls. This is higher and not in agreement with that reported in previous published studies [1,6]. This could be due to the difference in the age (mean and range) of the controls between those included in these studies and those of the current study [1,6].

It has been previously suggested that increased excitability of the interneurons of the brainstem reticular formation generates SBR [4,8,15,20]. In 1998, Miwa *et al*. [1] studied BR recovery and compared it between SBR-positive and SBR-negative participants. They found that there was no significant difference as regards BR recovery between the R1 and R2 components in SBR-positive and SBR-negative participants, suggesting that increased brainstem interneurons excitability is not essential to generate SBR [1]. Although in the current work the BR recovery in SBR elicitability and SBR-negative participants was not studied, the high frequency of SBR positivity in healthy controls supported the fact that increased excitability is not crucial to generate SBR. Hence, the findings of current study are in agreement with the suggestions of Miwa *et al*. [1] on the hypothetical sensory-motor gating mechanism, which exists before somatosensory signals enter the common blink interneuronal networks, modulating the functionally organized linkage between a sensory input and the corresponding motor output [1]. The suggested possibility is that the appearance of SBR depends on the level of activity in such a gating mechanism, which exerts inhibition on the inflow of the somatic input. In other words, participants with less habituation of the sensory-motor gating mechanism either normally or diseased would be SBR positive [1]. All of the above could explain the lack of significant difference between the healthy side in the PFP and PFS groups and the control group as regards SBR elicitability.

The current study had three limitations. First, the lack of long follow-up, and so the current study could not predict whether or not SBR has a prognostic importance in PFP. Second, all included PFP patients were idiopathic PFP (Bell's palsy) and all patients with PFS developed it following idiopathic PFP (Bell's palsy). The lack of variability in etiologies could alter the obtained results. Third, the small number of patients included in the current study. Further studies with larger sample size are recommended.

In conclusion, SBR occurs in PFP, PFS, and healthy participants. It has no relation to clinical and electrophysiological changes occurring in PFP and PFS. Increased brainstem interneurons excitability is not essential to generate SBR. The hypothetical sensory-motor gating mechanism could be responsible for SBR generation.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

References

1 Miwa H, Nohara C, Hotta M, Shimo Y, Amemiya K. Somatosensory-evoked blink response: investigation of the physiological mechanisms. Brain 1998; **121**:281–291.

2 Esteban A. A neurophysiological approach to brainstem reflexes. Blink reflex. Neurophysiol Clin 1999; **29**:7–38.

3 Miwa H, Imamura N, Kogahara K, Ohori T, Mizuno Y. Somatosensory evoked blink response: findings in patients with Miller Fisher syndrome and in normal subjects. J Neurol Neurosurg Psychiatry 1995; **58**:95–99.

4 Cha JK, Kim SH, Kim JW. Somatosensory Evoked Blink Reflex in neurologic diseases. J Korean Neurol Assoc 1998; **16**:660–665.

5 Erdemir-Kiziltan M, Uzun N, Kiziltan G, Karaali-Savrun F. Somatosensory evoked blink response in hemifacial spasm and peripheral facial palsy. Electromyogr Clin Neurophysiol 2005; **45**:203–207.

6 Erkol G, Kiziltan ME, Uluduz D, Uzun N. Somatosensory eye blink reflex in peripheral facial palsy. Neurosci Lett 2009; **460**:201–204.

7 Ozgur A, Semai B, Hidir UU, Mehmet Fatih O, Tayfun K, Zeki O. Which electrophysiological measure is appropriate in predicting prognosis of facial paralysis? Clin Neurol Neurosurg 2010; **112**:844–848.

8 Valls-Solé J, Valldeoriola F, Tolosa E, Marti MJ. Distinctive abnormalities of facial reflexes in patients with progressive supranuclear palsy. Brain 1997; **120**:1877–1883.

9 Syed NA, Delgado A, Sandbrink F, Schulman AE, Hallett M, Floeter MK. Blink reflex recovery in facial weakness: an electrophysiologic study of adaptive changes. Neurology 1999; **52**:834–838.

10 Lew HL, Tsai S. Pictorial guide to nerve conduction techniques. In: Pease WS, Lew HL, Johnson EW, editors. *Practical electromyography*. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2007;213–255.

11 SPSS. *Statistical package of social science, version 17*. London: University of Cambridge; 2007.

12 Miwa H, Yamaji Y, Abe H, Mizuno Y. Evaluation of the somatosensory evoked blink response in patients with neurological disorders. J Neurol Neurosurg Psychiatry 1996; **60**:539–543.

13 Cattaneo L, Chierici E, Pavesi G. Bell's palsy-induced blepharospasm relieved by passive eyelid closure and responsive to apomorphine. Clin Neurophysiol 2005; **116**:2348–2353.

14 Chuke JC, Baker RS, Porter JD. Bell's palsy-associated blepharospasm relieved by aiding eyelid closure. Ann Neurol 1996; **39**:263–268.

15 Valls-Solé J, Cammarota A, Alvarez R, Hallett M. Orbicularis oculi responses to stimulation of nerve afferents from upper and lower limbs in normal humans. Brain Res 1994; **650**:313–316.

16 Oge AE, Yayla V, Demir GA, Eraksoy M. Excitability of facial nucleus and related brain-stem reflexes in hemifacial spasm, post-facial palsy synkinesis and facial myokymia. Clin Neurophysiol 2005; **116**:1542–1554.

17 Evinger C, Bao JB, Powers AS, Kassem IS, Schicatano EJ, Henriquez VM, Peshori KR. Dry eye, blinking, and blepharospasm. Mov Disord 2002; **17**(Suppl 2):75–78.

18 Manca D, Muñoz E, Pastor P, Valldeoriola F, Valls-Solé J. Enhanced gain of blink reflex responses to ipsilateral supraorbital nerve afferent inputs in patients with facial nerve palsy. Clin Neurophysiol 2001; **112**:153–156.

19 Schicatano EJ, Mantzouranis J, Peshori KR, Partin J, Evinger C. Lid restraint evokes two types of motor adaptation. J Neurosci 2002; **22**:569–576.

20 Kimura J. Disorder of interneurons in Parkinsonism. The orbicularis oculi reflex to paired stimuli. Brain 1973; **96**:87–96.