ORIGINAL ARTICLE
Korean J Audiol 2011;15:129-132 pISSN 2092-9862 / eISSN 2093-3797

Evaluation of Trigeminal Nerve Involvement Using Blink Reflex Test in Bell's Palsy

Ki Bong Lee[1], Ji Hoon Kim[1], Yoon Ah Park[1], Yoon Gil Park[2], Won-Sang Lee[1] and Eun Jin Son[1]

[1]Departments of Otorhinolaryngology, [2]Rehabilitational Medicine, Yonsei University College of Medicine, Seoul, Korea

Received November 7, 2011
Revised November 28, 2011
Accepted December 1, 2011

Address for correspondence
Eun Jin Son, MD, PhD
Department of Otorhinolaryngology,
Yonsei University
College of Medicine,
211 Eonju-ro, Gangnam-gu,
Seoul 135-720, Korea
Tel +82-2-2019-3460
Fax +82-2-3463-4750
E-mail ejson@yuhs.ac

Background and Objectives: Facial nerve dysfunction in Bell's palsy is evaluated using various parameters including physical examination and electrophysiological studies. Associated symptoms of facial numbness or paresthesia are reported by some patients with Bell's palsy. The aim of this study was to investigate trigeminal nerve involvement in Bell's palsy using blink reflex test. **Subjects and Methods**: Facial nerve and trigeminal nerve functions were assessed using House-Brackmann (HB) grading system, electroneuronography (ENoG) and blink reflex tests in 28 patients diagnosed as Bell's palsy. **Results**: HB grades correlated with degeneration ratio from ENoG (p=0.002, chi-square test). The ipsilateral R1 reponse of the blink reflex was absent or abnormal in 27/28 patients (96.4%), and ipsilateral R2 response was absent or abnormal in 26/28 patients (92.8%). Contralateral R2 was abnormal in 5/28 patients (17.8%), suggesting involvement of trigeminal nerve in a portion of patients. The results of blink reflex test showed no significant correlation to the outcome of facial nerve function (HB grade) in Bell's palsy patients. **Conclusions**: Blink reflex test provides information about trigeminal and facial nerve functions in addition to ENoG results. Our study suggests that subclinical involvement of trigeminal nerve may accompany facial nerve dysfunction in Bell's palsy.

Korean J Audiol 2011;15:129-132

KEY WORDS: Bell's palsy · Blink reflex · Trigeminal nerve.

Introduction

Bell's palsy is the most common cause etiology of acute facial nerve palsy occurring in about 10−30 per 100,000 of the general population.[1)] Since the facial nerve is primarily a motor nerve, dysfunction of the seventh cranial nerve is usually evident by limitation of voluntary movements by facial expression muscles. Taste disturbance from the small sensory component through the chorda tympani may be present. Some patients also complain of facial numbness or paresthesia, not confined to the postauricular area related to the auricular sensory branch, but on the lateral side of the face. Such symptoms may be related to trigeminal nerve involvement.

Electrophysiological studies are widely utilized in facial palsy patients in addition to physical examination of voluntary movements. Nerve excitability test (NET) and electroneuronography (ENoG) are widely used to assess the functional status and to predict the outcome.[2-4)] The blink reflex (BR) is a polysynaptic reflex responseof the orbicularis oculi muscle elicited by electricalstimulation of the supraorbital nerve and is mediated by the afferent trigeminal nerve, the brainstem and the efferent facial nerve. The BR test can be used to evaluate the function of the involved nerves.[5,6)] The BR responses consist of two different temporaryresponses: early ipsilateral R1 and late bilateral R2. The R1 is generated through a reflex arc in the ipsilateral part of the brainstem with a direct connection between trigeminal pathway and the facial motor nucleus, and the R2 is presumed to be mediated through polysynaptic interneurons in the lateral reticular formation in the brainstem.[7)]

The aim of this study was to investigate trigeminal nerve involvement in Bell's palsy patients using blink reflex test.

Subjects and Methods

The clinical data of patients presenting with acute onset unilateral facial nerve palsy to the Yonsei University College of

Medicine Gangnam Severance Hospital from between October 2010 to June 2011 were reviewed. Twenty-eight patients who were diagnosed as Bell's palsy and followed up for more than 1 month were included in the study. Exclusion criteria were previous history of facial palsy, Ramsay-Hunt syndrome, traumatic facial palsy, diabetes mellitus, and facial palsy due to central or peripheral nervous system disorders. Bell's palsy was diagnosed when other recognizable causes were ruled out by careful history review, neurological examinations, serological and radiological studies. The facial nerve function was assessed by House-Brackmann (HB) facial nerve grading system at initial presentation and at each follow up visit. Complete recovery to HB grades I or II were considered satisfactory compared to incomplete recovery to HB grades III to IV. Electrophysiological tests were performed using performed using a Viking IV electromyographysystem (Nicolet, Madison, WI, USA) between 3 to 14 days since the onset of facial palsy. On ENoG, supramaximal stimulation of 0.2-ms duration at a rate of 1 Hz was provided through bipolar surface electrodes. Electrodes were placed with the anode just outside the stylomastoid foramen and the cathode in front of the ear lobe and manipulated to obtain the maximal compound action potential amplitude on the display. For recording, the surface disc electrodes were placed in the nasolabial fold. The ground electrode was placed on the lower jaw. Peak-to-peak amplitudes of ENoG response on the affected side were compared with those on the unaffected side. Percentage of response amplitude on the affected side compared with that on the unaffected side was designated the degeneration ratio (DR) value for the patient. For evaluation of trigeminal nerve function, BR tests were performed. In BR testing, 18-mA stimulation of 0.2-ms duration was applied via the supraorbital margin, and responses of the orbicularis oculi muscles were recorded through surface disc electrodes. The responses of both sides were recorded (ipsilateral R1 and R2 and contralateral R2). In normal cases with intact trigeminal and facial nerve function, the elicited blink reflex consists of an early ipsilateral, R1, with a latency about 10 ms (range 8−13 ms). The late polysynaptic response, R2, has a latency of 30−45 ms. Latencies of the ipsilateral R1 and R2 and contralateral R2 were measured. Delayed contralateral R2 response was considered indicative of impaired trigeminal nerve function. The correlation between HB grade of last visit and the results of EP studies (ENoG and BR) was assessed to evaluate the value of the EP tests as prognostic factor in Bell's palsy. The statistical analysis was performed with chi-square test using SPSS statistical software (SPSS, Chicago, IL, USA) and p value of < 0.05 was considered significant.

Results

Patient characteristics

Among the 28 patients with Bell's palsy, the male to female ratio was 13 : 15 and the facial palsy developed in the right side in 12 patients. The average age was 44.71 ± 18.3 years. The mean interval from the onset of facial palsy to the initial visit was 2.3 ± 2.4 days, and the electrophysiological tests were performed after 8.3 ± 3.5 days since onset.

Facial nerve outcome

Facial nerve function was evaluated clinically using House-Brackmann grading system at the initial visit, at nadir during progress, and at last follow up (mean 58.1 days, range 30−270 days)(Fig. 1). Most of the patients presented with HB grade III or worse (25/28 patients, 89.3%). ENoG was performed in all patients and degeneration ratio on ENoG was calculated as 53.8 ± 23.5%. Facial nerve function finally recovered to HB grades I or II in most patients (19/28 patients, 67.9%).

BR responses

The BR test results are presented in Table 1. As expected, ipsilateral R1 response was normal in only one patient, and ab-

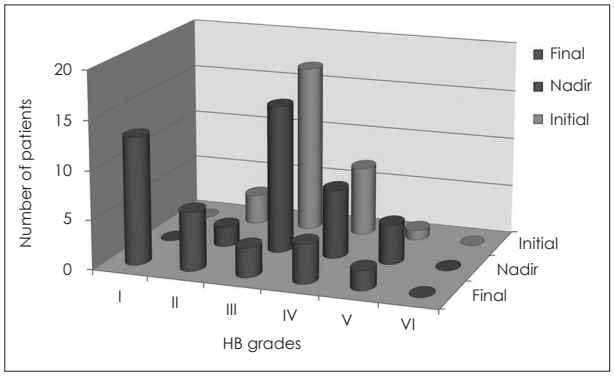

Fig. 1. Facial nerve function evaluated by House-Brackmann grades.

Table 1. Results of BR test

		Number of patients	%	Latency (ms) Mean ± SD
R1	Normal	1	3.6	9.1
	Absent	21	75.0	–
	Prolonged	6	21.4	13.15 ± 4.6
R2	Normal	2	7.1	32.55 ± 1.2
	Absent	18	64.3	–
	Prolonged	8	28.6	36.93 ± 1.7
Contralat R2	Normal	23	82.1	29.98 ± 1.9
	Absent	0	0	–
	Prolonged	5	17.9	36.96 ± 3.9

BR: blink reflex

Table 2. Correlation between electrophysiological studies and prognosis of facial palsy

Variables		Number of patients with favorable outcome (HB grade I or II)(n=19)	Number of patients with poor outcome (HB grade III or worse) (n=9)	*p* value
Initial DR on EnoG (mean±SD)		45.62±23.3	71.02±14.9	0.002
BR: ipsilateral R1	Normal	1	0	0.491
	Absent or prolonged	18	9	
BR: ipsilateral R2	Normal	2	0	0.312
	Absent or prolonged	17	9	
BR: contralateral R2	Normal	14	9	0.090
	Absent or prolonged	5	0	

BR: blink reflex, DR: degeneration ratio, ENoG: electroneuronography

sent (21/28, 75.0%) or prolonged (6/28, 21.4%) in most patients. Ipsilateral R2 was normal in only 2 patients and absent (18/28, 64.3%) or prolonged in (8/28, 28.6%) in others. While abnormalities in the ipsilateral R1 or R2 responses can be readily explained by facial nerve dysfunction, it can be expected that contralateral R2 will be normal in case of normal trigeminal nerve function in facial palsy patients. However, contralateral R2 response was prolonged in 5 of 28 patients (17.9%), suggesting abnormality in the input through the ipsilateral trigeminal nerve. Interestingly, contralateral R2 was not absent in any of the cases, suggesting that it was unlikely that a brainstem lesion was responsible for absence of ipsilateral R1 or R2 in our facial palsy patients.

Prognosis of facial palsy

Patients who recovered to HB grade I or II were considered as favorable outcome and those with facial palsy of HB grade III or worse at last visit as poor outcome. The electrophysiological test results were reviewed for their value in predicting facial nerve outcome (Table 2). ENoG is widely used to evaluate the various degrees of axonal degeneration to predict the outcome of facial nerve function, and a statistically significant correlation was observed between degeneration ratio (DR) and facial nerve outcome (p=0.002). However, no significant correlation was observed between abnormalities in ipsilateral R1 or R2 response of BR tests and facial nerve outcome. Likewise, no predictive value was shown for abnormal R2 response on the contralateral side.

Discussion

The BR responses consist of two different temporary responses: early ipsilateral R1 and late bilateral R2. The R2 response is thought to be consisted of one or two interneurons, located near the main sensory nucleus of the trigeminal nerve. The input for R2 responses is conducted through the descending spinal tract of the trigeminal nerve in the pons and medulla oblongata to the caudal spinal trigeminal nucleus. The impulses are then conveyed to the facial nuclei in the pons, passing through the lateral tegmental field, medial to the spinal trigeminal nucleus. The contralateral R2 response is generated by an ascending trigeminofacial connection that crosses the midline at the level of the lower third of the medulla oblongata.[8] Thus, abnormalities in the responses of ipsilateral R1 and R2 are thought to arise from injury to the afferent pathway in facial paralysis. In a patient with facial nerve palsy and normal trigeminal function, R1 and R2 responses are missing on the ipsilateral side due to the facial involvement, while the R2 on the contralateral side is normal, indicating an adequate afferent, trigeminal function. When stimulating the contralateral side normal R1 and R2 are present, but the R2 is missing on the facial palsy side. Interestingly, in 6 of our patients, BR tests were consistent with simultaneous trigeminal dysfunction on facial palsy side. The involvement of the afferent, trigeminal, part of the reflex arc is suggested by prolonged response of the contralateral R2. This pattern is consistent with affection of the peripheral part of the trigeminal nerve on the facial palsy side. The possibility of trigeminal dysfunction due to a brainstem lesion in patients with apparently isolated facial palsy was explored using BR responses.[7] However, we did not find any case with BR results indicating the facial nerve dysfunction and simultaneous trigeminal impairment at the brainstem level, which would show as the absence of the ipsilateral R2 when the facial palsy side is stimulated and also the loss of contralateral R2 indicating brainstem dysfunction. Further studies are needed to evaluate the prevalence of multiple cranial neuropathy or brainstem lesion in acute facial palsy patients.

Although the overall facial nerve outcome of Bell's palsy is acceptable and reported as about 70% of patients recovering to HB grade I, the patients experience significant distress on social activities. Several factors have been reported to be prognostic for facial palsy such as age of the patient and the initial severity of facial nerve dysfunction. The results of ENoG, that is the degeneration ratio (%) greater than 90%, has been re-

ported to predict poor outcome of the facial palsy, and proposed as an index for surgical intervention. Some reports also evaluate the predictive value of BR tests on the prognosis of facial nerve. Incomplete resolution was predicted if BR responses were not obtained in the first week.[5] Another study predicted poor prognosis when ipsilateral R1 and R2 responses could not be obtained in the first 3 weeks, poor prognoses were predicted.[3] Other reports also evaluated the BR responses in the first 1 to 5 weeks.[7,9,10] In our study, we could not find correlation the BR responses and the facial nerve outcome. It may be attributed to the difference that the tests were performed on the same day as the ENoG tests, which covered a longer range from 4−20 days (mean 8.3 days) after the onset of facial palsy. Also, it still poses a challenge to evaluate the prognostic values of multiple clinical features in facial palsy and the clinical implication of the BR test results should be considered as another compounding factor.[4,11]

The facial nerve function shows gradual recovery in patients with Bell's palsy, and one of the limitations of this study is that longer follow up is warranted to accurately assess the final outcome. It is possible that the correlation between the results of BR tests and the facial nerve function may have been obscured due to insufficient follow up duration in cases with slow recovery. Although shorter follow up periods may be enough to assess the final outcome in patients with complete recovery to HG grades I or II within given period of time, future studies should schedule at least 6 months of follow up in all cases for accurate assessment of facial nerve recovery.

Since the BR is influenced not only by trigeminal and facial nerve, but by many factors including including the motor cortex, the postcentral area of the cortex, and the basal ganglia, different tests cans be utilized to diagnose trigeminal nerve dysfunction. The masseter inhibitory reflex (MIR), also called the cutaneous or exteroceptive suppression reflex, can be elicited by delivering electrical stimuli to the mentalis or infraorbital nerves during voluntary clenching of teeth. The silent periods (SP1 and SP2) are observed during MIR recording of muscle activity, and their changes reflect trigeminal dysfunction.[3,8] Also the excitability of a reflect circuit can be measured using dual stimuli at various intervals to obtain recovery curves for the BR or MIR. In order to evaluate the possibility of trigeminal nerve involvement in Bell's palsy patients, BR responses were examined in our study. In order to describe the trigeminal involvement more comprehensively and possibly to localize the site of involvement in trigeminal nerve dysfunction, trigeminal reflexes should be evaluated in all three divisions. Subclinical involvement of the trigeminal nerve has been demonstrated in patients with diabetic polyneuropathy, trigeminal neuralgia, postherpetic neuralgia, vascular malformations, benign tumors of the cerebellopontine angle, and multiple sclerosis using neurophysiological tests.[8,12] Further evaluation of trigeminal dysfunction in facial palsy patients may offer useful information about development of sensory symptoms.

Conclusion

Blink reflex test provides information about trigeminal and facial nerve functions in addition to ENoG results. Our study suggests that subclinical involvement of trigeminal nerve may accompany facial nerve dysfunction in Bell's palsy.

Acknowledgment

This study was supported by a faculty research grant of Yonsei University College of Medicine (6-2008-0249).

REFERENCES

1) Katusic SK, Beard CM, Wiederholt WC, Bergstralh EJ, Kurland LT. Incidence, clinical features, and prognosis in Bell's palsy, Rochester, Minnesota, 1968-1982. Ann Neurol 1986;20:622-7.
2) Mantsopoulos K, Psillas G, Psychogios G, Brase C, Iro H, Constantinidis J. Predicting the long-term outcome after idiopathic facial nerve paralysis. Otol Neurotol 2011;32:848-51.
3) 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;112:844-8.
4) Ushio M, Kondo K, Takeuchi N, Tojima H, Yamaguchi T, Kaga K. Prediction of the prognosis of Bell's palsy using multivariate analyses. Otol Neurotol 2008;29:69-72.
5) Kimura J, Giron LT, Young SM. Electrophysiological study of Bell palsy: electrically elicited blink reflex in assessment of prognosis. Arch Otolaryngol 1976;102:140-3.
6) Kokubun N, Hirata K. Neurophysiological evaluation of trigeminal and facial nerves in patients with chronic inflammatory demyelinating polyneuropathy. Muscle Nerve 2007;35:203-7.
7) Hanner P, Badr G, Rosenhall U, Edström S. Trigeminal dysfunction in patients with Bell's palsy. Acta Otolaryngol 1986;101:224-30.
8) Aramideh M, Ongerboer de Visser BW. Brainstem reflexes: electrodiagnostic techniques, physiology, normative data, and clinical applications. Muscle Nerve 2002;26:14-30.
9) Heath JP, Cull RE, Smith IM, Murray JA. The neurophysiological investigation of Bell's palsy and the predictive value of the blink reflex. Clin Otolaryngol Allied Sci 1988;13:85-92.
10) Mikula I, Miskov S, Negovetić R, Demarin V. Blink reflex in the prediction of outcome of idiopathic peripheral partial facioparesis: follow-up study. Croat Med J 2002;43:319-23.
11) Ikeda M, Abiko Y, Kukimoto N, Omori H, Nakazato H, Ikeda K. Clinical factors that influence the prognosis of facial nerve paralysis and the magnitudes of influence. Laryngoscope 2005;115:855-60.
12) Cruccu G, Agostino R, Inghilleri M, Innocenti P, Romaniello A, Manfredi M. Mandibular nerve involvement in diabetic polyneuropathy and chronic inflammatory demyelinating polyneuropathy. Muscle Nerve 1998;21:1673-9.