Blink Reflex in the Prediction of Outcome of Idiopathic Peripheral Partial Facioparesis: Follow-up Study

Ivan Mikula, Snježana Miškov, Ružica Negovetić, Vida Demarin

Department for Neurology and Clinical Neurophysiology, Sisters of Mercy University Hospital, Zagreb, Croatia

Aim. To determine the value of the blink reflex as a predictor of outcome of idiopathic peripheral partial facial paresis.

Methods. The study included 30 patients suffering from acute idiopathic peripheral facioparesis and 30 age- and sex-matched healthy controls. Patients with symptomatic disease were excluded on the basis of neuroradiologic and laboratory findings. We stimulated the supraorbital foramen and recorded the evoked response from both orbicularis oculi muscles. We measured the ipsilateral early phasic component (R1) and bilateral late tonic component (ipsilateral R2 and contralateral R2') immediately, and a week and 6 months after the first test.

Results. In the acute phase of idiopathic peripheral partial facioparesis, the blink reflex showed slightly prolonged latencies and greatly reduced amplitudes of both R1 and R2 and the normal latencies and amplitudes of R2'. All subjects showed clinical symptoms and typical electromyographic (EMG) changes, whereas 24 had blink reflex abnormalities. One week after the onset, all patients were still symptomatic and showed EMG changes, but blink reflex abnormalities remained in only 11 patients. Six months after the onset, 21 patients became asymptomatic and showed no EMG changes, 7 had no clinical symptoms but showed chronic neurogenic EMG changes, whereas 2 showed both clinical symptoms and EMG changes. Blink reflex abnormalities were observed in 6 patients. The amplitudes of R1 immediately and one week after the onset were the best predictors of residual motor deficit.

Conclusions. Blink reflex is a useful tool for follow-up and recovery prognosis in patients with partial idiopathic facioparesis, especially in the early recovery phase.

Key words: blinking; electromyography; facial muscles; facial paralysis; nervous system physiology; reflex

Blink reflex can be elicited by either electrical or magnetic stimulation of the supraorbital nerve (1,2), and the responses of bilateral orbicularis oculi muscles can be recorded. The ipsilateral early blink component (R1) can be observed together with an ipsilateral early eye movement at a latency of 10-15 ms. Bilateral late components (R2, R2') can be observed at 40-70 ms (3). R3 component has also been described and considered nociceptive (4). Studies of the blink reflex provide useful information on neurophysiologic status of the trigeminal and facial nerves (5), the brainstem, and the cranial end of the cervical segment of the spinal cord (6), and show a high level of correlation with magnetic resonance imaging (MRI) findings (7).

The test is particularly useful in Bell’s palsy patients with hemifacial spasm and synkinesis (8,9). It can demonstrate hyperexcitability of central motor neurons and its return to normal level after recovery (10). It can also indicate the quality of lower brain functions, such as feeding (11,12). Blink reflex latencies are prolonged and amplitudes decreased in peripheral facial palsy, some polyneuropathies (e.g., diabetic and uremic), Guillain Barre’s syndrome (13,14), Behçet’s disease (15), Chiari II malformation (16), and Wallenberg’s syndrome (17). R2 time can be used to diagnose demyelinating diseases (18). Absence of the blink reflex has been demonstrated in Holmes-Adie syndrome (19). Dysfunction of small afferent fibers of the trigeminal nerve is frequent in patients with diabetic and other sensory polyneuropathies (20). Habituation of blink reflex and occurrence of the late R3 component are found in psychotic disorders and migraine (21,22). Blink reflex has also been used for follow-up after a cross facial nerve graft (23). Blink reflex can be inhibited by anti-migraine drugs, sumatriptan and zolmitriptan (24). Excess thyroid hormone inhibits the blink response (25), as well as clonidine (26) and tension headache (27). Its parameters can also be modified by a pre-pulse caused by somatosensory inputs (28,29). Teeth clenching facilitates blink reflexes (30). The facilitation of the blink reflex and the appearance of R3 are also seen in tetanus and stiff person syndrome (31). The same effect may be observed contralaterally to the affected side in patients with peripheral facial palsy when the affected side is stimulated (32).
Although the blink reflex has been accepted as a valuable diagnostic tool, there have been no studies on specific parameters of the blink reflex, describing which of the parameters are diagnostically useful and prognostically valuable, in which cases, and when.

In our clinical practice we have observed that peripheral facial paresis inhibits the blink reflex and that its recordable abnormalities precede other clinical and neurophysiologic changes. These abnormalities seem to correlate with the disease outcome, or more specifically, with the residual motor deficit.

In this study we assessed the value of the blink reflex as a predictor of the outcome of idiopathic peripheral partial facial paresis. We aimed to determine the applicability of several blink reflex parameters in the prediction of residual motor deficit, such as latencies and amplitudes of ipsilateral R1 and R2 components, and contralateral R2′ component.

Subjects and Methods

Subjects

The study comprised 30 patients (17 men and 13 women; mean age 37.7±7.2 years) diagnosed with idiopathic peripheral facial paresis according to the International Classification of the Diseases (ICD-X) criteria (an abrupt, isolated, unilateral, peripheral facial paralysis without detectable cause). The patients were included in the study within 3 days after the onset of the disease.

Patients suffering from tumors, diabetes, thyroid disease, and other neuropathies or taking medications that could affect the test results (clonidine, sumatriptan or zolmitriptan) were excluded from the study (7 subjects: 3 with diabetes, 1 taking sumatriptan, 1 with neurofibromatosis, 1 with neuroma of the statoacoustic nerve, and 1 with Melkersson-Rosenthal syndrome).

The control group comprised 30 age- and sex-matched healthy controls (17 men and 13 women, mean age 35.2±8.1 years).

The symptomatic disease was ruled out by means of thorough disease history taking and neurological examination, as well as by specific laboratory and radiological tests. Laboratory findings (sedimentation rate, complete blood count, and differential white blood cell count) showed no sign of an inflammatory disease, blood glucose was normal, as well as serum electrolytes, triiodothyronine (T3), thyroxin (T4), thyrotropin (TSH), serum gamma-glutamyl-transpeptidase (SGGT), serum glutamic-oxaloacetic-transaminase (SGOT), and serum glutamic-pyruvic-transaminase (SGPT). X-ray examination of the paranasal sinuses and pyramids gave normal findings. Computerized tomography (CT) of the brain, with special consideration to basal structures, showed no pathology.

Methods

Recordings were made with the Medelec “Sapphire” equipment (Old Woking, Surrey, UK) via cup electrodes located over both orbicularis oculi muscles (active electrode above the lateral and reference electrode above the lower part of the muscle) and one non-cephalic ground electrode located over the left wrist.

The supraorbital foramen was stimulated with a single stimulus of 15-20 mA (sweep 100 ms, division 10 ms, gain 5 mV). The evoked response was recorded from both orbicularis oculi muscles. The amplitudes and latencies of ipsilateral early phasic component (R1) and bilateral late tonic components (R2, R2′) were measured at the first negative peak.

The measurements were made in the acute phase of the illness and repeated 1 week and 6 months after the onset of symptoms. All patients began taking oral prednisone (80 mg/day) within 3 days after the onset of disease. The initial dose of prednisone was administered for a week and then tapered gradually over the following week. The results were compared with those of the age and sex-matched normal controls (volunteers among the hospital staff and students). Due to the generally unpleasant nature of electrical stimulation, only a single testing was performed on subjects from the control group. All the participants gave their informed consent.

Statistics

Median, minimum, and maximum values of amplitudes and latencies were calculated for the blink reflex components R1, R2, and R2′ in the two groups of subjects (patients being tested three times and the controls only once).

As the data were not normally distributed, non-parametric Wilcoxon Mann Whitney’s test on ranks was used to determine significant differences between the patient and control group (p<0.05). The C5.0 algorithm was used to determine the input variables on the basis of which the patients divided in two groups according to the residual motor deficit (recovered and non-recovered) could be discriminated.

Results

Median values of amplitudes (mV) of both ipsilateral components R1 and R2 were lower in patients suffering from idiopathic peripheral facial paresis, especially in the acute phase, than the median values of amplitudes of contralateral R2′ component, which were similar to those in the control group (Fig. 1).

Median values of latencies (ms) of both ipsilateral components R1 and R2 were higher in patients with idiopathic peripheral facial paresis, especially in the later phases, whereas the latencies of contralateral R2′ component showed values similar to those in the control group (Fig. 2).

Wilcoxon Mann Whitney’s test on ranks showed that the patients differed significantly from the control group, both in the amplitudes and latencies. Significant differences were found between the amplitudes of R1 components measured in patients immediately after the onset of disease and a week later, and those measured in healthy controls. Significant differences were also found between the amplitudes of R2 component measured in patients immediately after the onset of disease and a week later, and those measured in healthy controls. Six months after the onset, the differences ceased to be significant. R2′ amplitude values in patients never differed significantly from those measured in the control group (Fig. 1). The latencies of R1 and R2 components immediately after the onset, and especially after a week, differed significantly from those in the control group (Fig. 2).

Figure 1. Median and minimum and maximum values of amplitudes (mV) of blink reflex components R1, R2, and R2′ immediately after the onset of the disease (open circle) and a week (closed circle) and six months after (open rhomb) the onset, compared with amplitude values of the control group (closed rhomb). Asterisk indicates p<0.05 vs controls, Wilcoxon Mann Whitney’s test.
from those of normal controls. The latency values measured six months after the onset of the disease did not differ significantly from those of normal controls, and neither did R2' latencies (Fig. 2).

Clinical symptoms and neurogenic EMG changes were found in all patients in the acute phase of disease, whereas blink reflex abnormalities were observed in 24 of them. A week after the onset, all symptoms and EMG changes were still present in all patients, but the latency and amplitude abnormalities of R1 and R2 remained in only 11 out of 30 patients. Six months after the onset of symptoms, 21 patients became asymptomatic and their EMG showed no changes, 7 had no clinical symptoms, but the chronic neurogenic EMG changes were observed, whereas 2 showed the residual weakness of the facial muscles and the signs of denervation in EMG. Blink reflex abnormalities, especially the decrease of the R2 amplitude, were observed in 6 patients (Fig. 3).

Twenty-one patients showing neither the residual motor deficit nor chronic neurogenic EMG changes recovered from the disease, whereas the remaining 9 did not. The C5.0 algorithm was used to determine the input variables that differed between the recovered and non-recovered patients (Fig. 4). The decision tree calculation showed that the amplitudes of R1 immediately (accuracy 83.3%) and a week after the onset (accuracy 90.0%) were the best classifiers for predicting the residual motor deficit. Accuracy calculation remained stable after the cross-validation testing of classifiers. The decision tree calculation showed that the amplitudes of R1 immediately and a week after the onset were the best classifiers for predicting the residual motor deficit.

Discussion

In patients with idiopathic facial paresis in the acute phase, the blink reflex showed slightly prolonged latencies and severely reduced amplitudes of both R1 and R2 on the afflicted side and the normal latencies and amplitudes on the contralateral side, proving the dysfunction in the efferent part of the reflex cycle. All patients had clinical symptoms, and their EMG revealed acute neurogenic changes. Twenty-four patients showed blink reflex abnormalities. Both the axon damage and (less pronounced) conduction block were significant in this phase.

Figure 2. Median and minimum and maximum values of latencies (ms) of blink reflex components R1, R2, and R2' immediately after the onset of the disease (open circle) and a week (closed circle) and 6 months after (open rhomb) the onset, compared with latency values of control group (closed rhomb). Asterisk indicates *p<0.05 vs controls, Wilcoxon Mann Whitney’s test.

Figure 3. Presence of symptoms (%) on physical examination (open columns), electromyography findings (closed columns), and blink reflex abnormalities (gray columns) in patients with idiopathic peripheral facial paresis at different time points after the onset of disease.

Figure 4. The decision tree for the amplitudes of R1 immediately and a week after the onset of disease as classifiers for predicting the residual motor deficit. Sample divided in two groups according to the residual motor deficit (group a – recovered, group b – non-recovered), number and percentage of prediction errors, accuracy calculation and cross-validation testing of classifiers.

Discussion
These findings were concordant with those of Eekhof et al (8) and Calleja et al (13). A week after the onset of the disease, all patients were still symptomatic and there were changes in their EMG findings, but the latency and amplitude changes of R1 and R2 remained in only 11 patients. It should be emphasized that the conduction velocities – indicators of a conduction block – were significantly changed at the time, whereas the reduction of amplitudes – indicators of an axonal damage – was not so pronounced any longer. However, the amplitudes were still significantly lower than those measured six months after the onset or in the group of healthy controls. To our knowledge, this is the first report on the blink reflex after a week of follow-up. Celik et al (9) investigated the disease 3 weeks after the onset and had similar findings. In our study, 21 patients became asymptomatic 6 months after the onset of symptoms, and their EMG showed no changes; 7 had no clinical symptoms, but the chronic neurogenic EMG changes could have been observed; and 2 showed the residual weakness of the facial muscles and the signs of denervation in EMG. Blink reflex abnormalities, especially the decrease of the R2 amplitude, were observed in 6 patients, 4 of them in the group with EMG changes and no clinical symptoms and 2 of them in the group with both EMG changes and clinical symptoms. The patients showing residual clinical and EMG changes were those 11 who still had blink reflex abnormalities a week after the onset of symptoms. These findings are concordant with most clinical investigations dealing with the residual impairment after the chronic course of idiopathic peripheral facial paresis (14,17,18).

In conclusion, the blink reflex is a valuable tool for follow-up and recovery prognosis of the partial idiopathic facial paresis, especially in the early recovery phase and when combined with EMG and neurography of the facial nerve. Furthermore, amplitudes of ipsilateral R1 and R2 components could be used as the primary predictive factors in the earliest course of illness. R1, and particularly R2 latency, should assume that role in later phases of the disease, but not sooner than a week after the onset.

References


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Correspondence to:
Ivan Mikula
Department for Neurology
Sisters of Mercy University Hospital
Vinogradska cesta 29
10000 Zagreb, Croatia
ivan.mikula@zg.tel.hr