Original Article / Çalışma - Araştırma

Understanding the features of posterior auricular muscle response in a facial nerve disease prototype

Fasiyal sinir hastalığı prototipinde posteriyor auriküler kas yanıtının özelliklerinin anlaşılması

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Objectives: This study aims to compare and investigate the changes of posterior auricular muscle response (PAMR) following peripheral facial nerve palsy (PFP) with blink reflex (BR).

Patients and Methods: A total of 75 idiopathic PFP patients and age and sex-matched 38 healthy volunteers were included. Patients with PFP were classified into two groups including early (≤6 months) and late (>6 months) based on the duration of symptoms. Following clinical evaluation, PAMR and BR were recorded. A comparison was performed among the early PFP group (n=52), late PFP group (n=23) and healthy volunteers (n=38).

Results: We obtained PAMR in 78.9% (n=30) of healthy volunteers, in 60.9% (n=14) of late and in only 38.5% (n=20) of early PFP patients (p=0.001). No habituation was observed following repeated stimuli. The mean latencies were 9.1±1.6, 10.9±1.9 and 12.3±2.0 msec., respectively (p=0.000). R_1 and R_2 latencies were longer in the early PFP group, compared to other groups (p=0.000).

Conclusion: Posterior auricular muscle response exhibits changes in a manner similar to R_1 and R_2 of BR in early and late PFP. The occurrence rate of PAMR seems to be lowest or its latency is prolonged in the early PFP.

Key Words: Blink reflex; electromyography; peripheral facial paresis; posterior auricular muscle response; posterior auricular muscle.

Amaç: Bu çalışmada, periferik fasiyal sinir parezi (PFP) sonrası posteriyor auriküler kas yanıtı (PAMR) değişiklikleri, göz kırpma refleksi (GKR) ile karşılaştırılarak inceledi.

Hastalar ve Yöntemler: Çalışmaya toplam 75 idiyopatik PFP hastası ve yaş ve cinsiyet uyumlu 38 sağlıklı gönüllü dahil edildi. Periferik fasiyal sinir parezi hastaları, semptomların süresine göre erken (≤6 ay) ve geç (>6 ay) olmak üzere iki gruba ayrıldı. Klinik değerlendirme sonrası PAMR ve BR kaydedildi. Erken PFP grup (n=52), geç PFP grup (n=23) ve sağlıklı gönüllüler (n=38) arasında karşılaştırmalar yapıldı.

Bulgular: Sağlıklı gönüllülerin %78.9'unda (n=30), geç PFP hastalarının %60.9'unda (n=14) ve erken PFP hastalarının sadece %38.5'inde (n=20) PAMR elde edildi (p=0.001). Tekrarlayan uyarılar sonrası habitüasyon gözlenmedi. Ortalama latanslar sırasıyla 9.1±1.6, 10.9±1.9 ve 12.3±2.0 msn idi (p=0.000). R_1 ve R_2 latansları, erken PFP grubunda, diğer gruplara kıyasla, daha uzundu (p=0.000).

Sonuç: Posteriyor auriküler kas yanıtı, erken ve geç PFP'de GKR'nin R_1 ve R_2 bileşenlerine benzer değişiklikler göstermektedir. Erken PFP'de PAMR oranı en düşüktür ya da latansı uzundur.

Anahtar Sözcükler: Göz kırpma refleksi; elektromiyografi; periferik fasiyal parezi; posteriyor auriküler kas yanıtı; posteriyor auriküler kas.

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The posterior auricular muscle (PAM) is located behind the auricle. It contributes to the postauricular muscle complex which actively enhances hearing in lower species, but is a remnant in humans.[1] However voluntary activation and response recordings have been reported in humans.[2] The posterior auricular muscle complex is mostly active during yawning and smiling.[3]

Focal dystonia of the PAM was previously observed.[4] As it is innervated by the most proximal branch of the facial nerve, it may also be affected in some facial nerve disorders like hemifacial spasm (HFS).[5]

The post-auricular muscle response (PAMR) is obtained after auditory stimulus. It has a multisynaptic pathway which consists of the brainstem and cochlear nerve as the afferent loop, and facial nerve as the efferent loop. This is a variable response and not all people exhibit this response. Because there are techniques to obtain a stabilized PAMR[6] with a peak latency of 12-13 ms, it was previously suggested as a screening test in newborn deafness.[7]

Here, we aim to analyze changes of PAMR after peripheral facial palsy (PFP) and compare these changes with changes of blink reflex (BR) which is a more standardized response that is obtained consistently in all people and represents widely known changes in facial nerve disorders.

PATIENTS AND METHODS

This was a retrospective study involving all idiopathic PFP patients who were referred for routine electrophysiological examinations between May 2005 and May 2009. In this period, a total of 114 PFP patients were evaluated and all underwent detailed neurological examination. Additional neuroimaging studies were done when indicated by neurological examination. Peripheral facial palsy cases with underlying intracranial lesions (n=39) were excluded from the analysis.

Out of 114 patients, we included 75 (30 males, mean age 41.8±14.8) with different symptom durations. This group partly consisted of patients with post-paralytic facial syndrome (PFS) which resulted from facial nerve hyperexcitability after injury and functional reorganization of synapses who were included in a previous study.[8]

Thirty-eight gender and age-matched healthy volunteers (14 males) with a mean age of 41.7±11.6 (p=0.963) were constituted for comparisons. The presence rate of PAMR in the healthy volunteer group was ensured to be statistically similar to the asymptomatic sides of the PFP patients.

Clinical evaluation specific to facial nerve functions was performed in all patients using the House Brackmann scale (HBS) which was adapted in 1985 by the American Academy of Otolaryngology-Head and Neck Surgery[9] and comprises a six-point subjective grading scale.

All PFP patients were subdivided into an early and late period according to symptom duration. Early PFP (n=52) was defined as symptom duration shorter than six months whereas late PFP (n=23) as symptom duration longer than six months.

Following clinical evaluation, standardized electrophysiological investigations were performed in both groups. The auditory thresholds were determined prior to electrophysiological investigations and required to be below 25 dB in all patients and healthy volunteers. Recordings were performed using Ag-AgCl pair of cutaneous electrodes (Nihon Kohden Medical, Tokyo, Japan).

The recordings from PAM were made in accordance with previously published reports.[10] Briefly, the active electrode was placed on the surface of the skin directly overlying the PAM; the reference electrode was on the pinna, and the ground electrode on the sternum. The monophasic 100 μs click auditory stimulus produced by Neuropack Sigma MEB-9100 (Nihon Kohden Medical, Tokyo, Japan) was delivered bilaterally through earphones as five bursts, with an intensity of 105 dB and at random intervals of 2-5 minute while patients remained in sitting position with their eyes slightly closed. Stimulus rise time was 1 ms and stimulus consisted of two stimuli at an interval of 3 ms. The filter settings were 2 kHz high cut and 20 Hz low cut. We recorded the latencies of bilateral non-rectified five consecutive reflex responses after each stimulus and measured them. In the literature, generally the peak latency is used for PAMR and in normal individuals is 12-13 ms However, since we measured onset latencies (distal latencies of initial deflection) in other reflex studies and included normal individuals for comparisons in this study, we also used onset latency for PAMR.

For BR recordings, the active recording electrode was placed over the middle part of the inferior orbicularis oculi (OOc) while the reference electrode was located 2 cm lateral to the rima oculi. The ground electrode was placed over the forehead. The responses were recorded while patients were in sitting position with their eyes slightly closed. A single electrical stimulus of 0.2 ms duration with an intensity of three times of R_2 threshold (8-14 mA) was applied percutaneously to the supraorbital nerve at its exit from the supraorbital foramen. The stimulus was given randomly as five consecutive bursts with a minimum interval of 20 sec. Onset latency (ms) and presence rate of R_1, R_2 responses were measured and mean values of five responses for each parameter were calculated. The filter settings were 3 kHz high cut and 20 Hz low cut. Analysis time was adjusted as 10 ms/div and amplitude sensitivity was 200 μV.

Statistical analysis

To determine differences between early and late phases of PFP patients, we compared PAMR presence, latencies and amplitudes between the two patient groups and control group as well as between two sides within each group. When calculating mean PAMR latency and amplitudes in the control group, we excluded cases without any PAMR and among healthy participants with bilaterally evoked PAMR, we chose one side randomly because they were exactly the same or were quite close.

House Brackmann scale scores were also compared in all groups and latencies of PAMR were arranged and compared according to HBS scores.

The analyses were performed using the SPSS version 11.5 statistical software package (SPSS Inc., Chicago, Illinois, USA). Comparisons of categorical values like sex and HBS score as well as presence of PAMR were made by Fisher's exact test or chi-square test. The response latencies and amplitudes were compared between early PFP, late PFP and healthy volunteers by one way ANOVA when distributed normally and by Kruskal-Wallis test in the case of nonhomogenous distribution. A p-value of <0.05 was considered statistically significant.

RESULTS

We obtained stabilized PAMR which did not habituate by repeated responses in healthy volunteers. Post-auricular muscle response was observed in 30 (78.9%) healthy volunteers, 24 (80%) of which were bilateral. Mean latency in normal controls was 9.1±1.6 msec. Presence of PAMR on asymptomatic sides of early PFP and late PFP groups was quite close to that of healthy volunteers (74.3% and 77.8%, respectively). Presence of PAMR was prominently low on the symptomatic sides of patients (45.3%) compared to healthy volunteers and asymptomatic sides. Post-auricular muscle response was more commonly absent in the early PFP group. It was elicited in 60.9% (n=14) of late and in only 38.5% (n=20) of early PFP patients (p=0.001). Furthermore, PAMR latencies were more prolonged and amplitudes were lower in both patient groups and latency difference was statistically significant (plat=0.000 and pamp=0.101). Figure 1 shows examples of PAMR in one normal subject and one PFP patient. Post-auricular muscle response latencies and amplitudes are shown in Table 1.

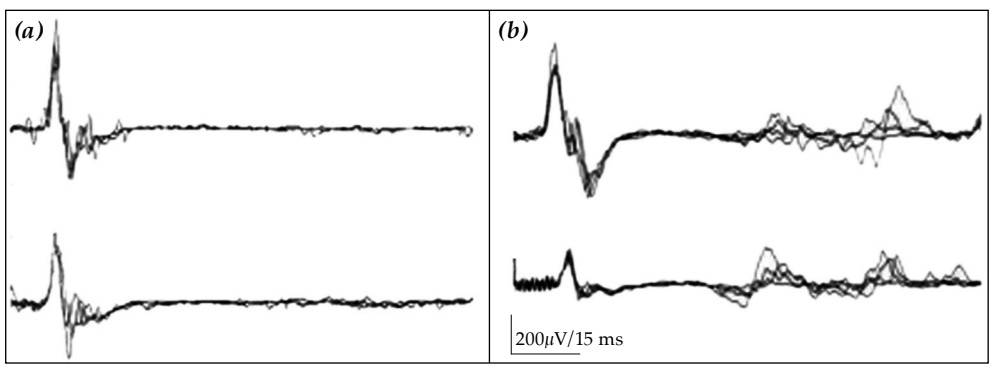

Figure 1. ***(a)*** *Normal bilateral posterior auricular muscle response showing some degree of amplitude asymmetry.* ***(b)*** *Decreased amplitude of right posterior auricular muscle response in a 17-years-old female with right sided peripheral facial nerve (symptom duration 12 days, House-Brackman score 1).*

Table 1. Comparison of post-auricular muscle response latency, amplitude and presence rate between early peripheral facial palsy (group 1), late peripheral facial palsy (group 2) and control (group 3) groups

	PAMR Latency ms	PAMR Amplitude ms	PAMR Presence rate	R1 latency	R2 latency
	Mean±SD	Mean±SD	%	Mean±SD	Mean±SD
Group 1	12.3±2.0	55.7±37.0	38.5	12.4±3.0	38.2±6.2
Group 2	10.9±1.9	74.7±63.9	60.9	11.2±1.9	32.7±5.8
Group 3	9.1±1.6	94.3±63.4	78.9	9.9±1.9	32.6±3.4
p	0.000	0.101	0.001	0.000	0.000

PAMR: Post-auricular muscle response; SD: Standard deviation.

Similarly, R_1 and R_2 latencies were also more prolonged among early PFP patients compared to other groups (pR_1=0.000 and pR_2=0.000, Table 1). Figure 2 shows similar findings regarding PAMR and BR in one PFP patient.

Comparisons within each patient group showed that PAMR latencies were longer and amplitudes were lower over symptomatic sides compared to asymptomatic sides in early PFP group (plat=0.000 and pamp=0.006) (Table 2). However, occurrence rates, latencies and amplitudes of PAMR were similar between symptomatic and asymptomatic sides of late PFP patients (pPR= 0.143, plat=0.314, pamp=0.115) (Table 2).

Among PFP patients in the early period, 48 (92.3%) had HBS scores of 3, or above whereas in the late period only one patient (4.3%) had an HBS score of 3 and the rest had mild clinical findings (p=0.005) and in the early PFP group as HBS score increased, presence rate of PAMR over the symptomatic side decreased (p=0.028).

DISCUSSION

The major findings of our study are that PAMR is mostly affected in the early period of PFP in parallel to BR and abnormal findings are directly related to worse clinical findings.

Traditionally, idiopathic PFP is believed to result from markedly demyelinating and sometimes axonal lesions of the nerve in the temporal bone before or after the geniculate ganglion. As PAMR is the most proximal branch after the lesion, the earliest changes probably develop involving the PAMR in the case of idiopathic PFP. In the acute phase the demyelinating lesions lead to weakness of ipsilateral mimic muscles which generally improve in time. Initially, clinical findings may be severe in almost half of patients. More than 40% of patients were reported to have HBS V-VI grades of paralysis in a study performed among patients who were treated conservatively in a tertiary center.[11] However, 85% of patients improved to some extent in six months time and severe sequelae are rare (15%) even in groups

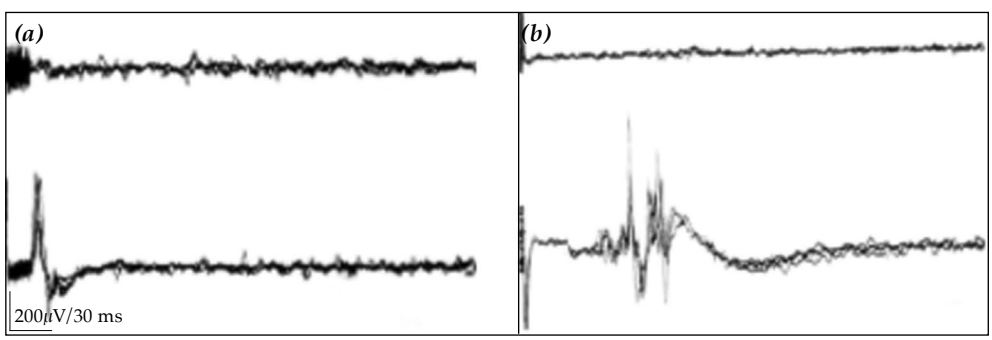

Figure 2. ***(a)*** *Loss of posterior auricular muscle response in a 51-years-old male with left sided peripheral facial nerve paresis (symptom duration 15 days, House-Brackman score 4).* ***(b)*** *Loss of both R_1 and R_2 after left supraorbital stimulation on symptomatic side in the same patient.*

Table 2. Comparison of post-auricular muscle response latency, amplitude and prensence rate between symptomatic and asymptomatic sides in early (group 1) and late (group 2) peripheral facial palsy groups

	Latency ms	Amplitude ms	Presence rate
	Mean±SD	Mean±SD	%
Group 1			
Symptomatic side	12.3±2.0	55.7±37.0	38.5
Asymptomatic side	9.7±2.1	111.7±82.2	74.3
p	0.000	0.006	0.000
Group 2			
Symptomatic side	10.3±1.8	75.0±66.4	60.9
Asymptomatic side	10.9±1.9	125.4±103.7	77.8
p	0.314	0.115	0.143

SD: Standard deviation.

without treatment.[12] Therefore, our patient group was distributed very similarly to the spectrum of the natural course of PFP and thus was likely to reflect the changes appropriately.

Clinical findings were more severe and R_1 and R_2 components of BR were much more prolonged in the early PFP group. Post-auricular muscle response measurements revealed results very similar to former findings. In the late phase, on the other hand, higher HBS scores were missing and this group reflects recovery in time for both clinical findings and electrophysiological measurements, R_1, R_2 and PAMR. Although this was not a follow-up study and we did not perform successive studies in one selected patient, we propose that PAMR is affected in the early phase and its recovery follows improvements of clinical findings and other electrophysiological parameters.

Post-auricular muscle response is a reflex with relatively shorter latency similar to R_1. Latencies of PAMR in our control group were shorter than those reported in the literature. However, this difference was secondary to our measurement technique because we measured onset latency instead of peak latency. As we obtained well-shaped responses, onset of waveforms and onset latencies were well-defined. Relatively shorter latency and its morphology which is monophasic similar to R_1 support that PAMR probably has a short circuit which may be oligosynaptic or monosynaptic. The BR is valuable in all phases of PFP.[13] it may show conduction block localizing the lesion in the acute phase, comparisons of amplitudes and latencies may bear importance for prognosis within days to months. The BR abnormalities like slightly prolonged latencies and reduced amplitudes of both R_1 and R_2 were previously shown in the acute phase of idiopathic PFP and amplitudes of R_1 were suggested as the best predictors of residual motor deficit.[14] Therefore, it may be considered as one of the gold standards and in our opinion, observation of similar results with PAMR reveals that it is also valuable. However, PAMR exhibits a major drawback, as it is not elicited in all humans.

As demyelinating lesions start to recover from the most proximal part, we expect to see recovery of PAMR before recovery of BR. Investigations regarding evolution of PAMR changes in a given patient may expose its prognostic significance. However, we did not analyze the order of recovery for BR and PAMR and thus this remains only a proposal.

It is known that the largest amplitude of PAMR is obtained directly over the pinna and its amplitude decreases getting through the pinna.[10] We also applied the same technique to obtain maximum amplitude. Although PAMR may also be related to the stimulus intensity, we think that 105 dB click stimulus is enough to elicit PAMR. Because PAMR was seen to be present at click stimulus levels just 10 dB above the auditory threshold. Click stimuli between 20-100 dB were generally used[7,10] and even monaural stimuli evoked bilateral PAMR.[15] The intensity of 105 dB may also elicit a BR (auditory BR, ABR) and a generalized startle reflex in some occasions. Although these late responses are beyond the scope of this article, PAMR may be suggested to originate from the anatomical proximity of

sternocleidomastoid muscle and spread of startle reflex over this muscle. However, there are several evidences demonstrating that PAMR is a distinct reflex. Firstly, auditory stimulus did not evoke R_1-like response over the OOc or any other muscles and the PAMR latency was shorter than those anticipated in startle reflex and ABR. Secondly, synkinetic spread of auditory startle reflex and ABR occurs in patients with PFS in contrast to PAMR.[8] Lastly, the startle response habituates by repeated stimuli whereas PAMR does not.

In the literature, there is another response which is defined over PAM after auditory stimuli: the posterior auricular reflex (PAR). We think that they probably represent the same response. Bochenek and Bochenek[16] studied PAR in the case of intracranial and extracranial facial nerve damage. Posterior auricular reflex could not be obtained in cases of intracranial lesions whereas it was obtained in cases of extracranial facial nerve damage. Posterior auricular reflex amplitude was correlated with the presence of motor paresis associated with intracranial lesions. Response did not disappear in patients with extracranial damage because the posterior auricular nerve which is the most proximal branch arises after the facial nerve gives off the chorda tympani nerve and emerges at the stylomastoid foramen just before the facial nerve enters the substance of the parotid gland and possibly before the extracranial lesion.

The major limitation of the study is the improper distribution of clinical findings between early and late PFP. However, this is a reasonable result correlating with the natural course of the disease. Designation as a follow-up study would overcome this challenge.

In conclusion, the post-auricular muscle response seems to be a relatively stabilized response and exhibits changes in a manner similar to R_1 and R_2 of BR in early and late PFP. The presence rate of PAMR is lower or its latency is prolonged in the early phase of PFP. Abnormalities of PAMR are directly related to clinical findings. Patients with severe clinical involvement had higher rates of PAMR loss which showed consistency of reflex changes with clinical findings.

Declaration of conflicting interests

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