A-WAVES IN PATIENTS WITH BELL’S PALSY

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Summary. The aim of this study is recording and investigation of multiple A-waves from frontalis and orbicularis oris muscles in patients with Bell’s palsy. Twenty-one males and 34 females with Bell’s palsy participated in electrophysiological studies. The control group consisted of 28 subjects (16 women and 12 men). A-waves were obtained during conventional F-wave investigation with 16 supramaximal stimuli with a frequency below 1Hz. Electrical stimulation with square wave pulses 0.2 ms in duration was applied via a bipolar surface electrode fixed over the nerve just below the ear and anterior to the mastoid process. The evoked compound muscle action potential (CMAPs) of frontalis and orbicularis oris muscles was recorded with concentric needle electrode (diameter of 0.45 mm) at an amplification of 200 μV/div. On the basis of the electromyographic manifestations (parameters of recorded CMAP of m. orbicularis oris and m. frontalis and conduction time (CT) of n. facialis), the patients were separated in three groups – with mild, moderate and severe facial paresis. A-waves were observed between CMAP and F-wave. They were with lower amplitude, approximately constant shape and latency. In 2 patients, A-waves were not found in both investigated muscles. Single A–wave recorded from frontalis muscle were observed in 8 (14.5%), multiple A-waves – in 35 (63.6%), and in 12 (21.8%) of patients, A-waves were not found. Multiple and single A-waves were observed in orbicularis muscle in 33 (60%) and 8 (14.5%) of patients respectively and in 14 (25.5%) of them, A-waves were not recorded. The multiple A-waves are common signs in the early stage of Bell’s palsy in cases of mild and moderate degree of paresis.

Key words: A-waves, Bell’s palsy, EMG, m. frontalis, m. orbicularis oris, multiple A-waves
INTRODUCTION

Bell’s palsy is a common neurological disease causing a considerable loss of self-esteem among patients. The term Bell’s palsy is accepted to describe an acute, monosymptomatic, peripheral, unilateral paresis of the facial nerve with unknown etiology [11]. A number of diseases as Lyme disease, leukemia, sarcoidosis, infectious mononucleosis etc might be a cause of facial paralysis, but these pareses are never classified as Bell’s palsy. In all pareses mentioned above, there is a disturbance in conduction of excitation along the facial nerve and respective changes in parameters of evoked compound muscle action potential (CMAP), recorded from facials muscles, (reduction of amplitude, prolongation of CMAP latency, and changes of wave form). Concomitant sign at these CMAP changes is the presence of abnormal late responses, so called A-waves.

A-waves have been observed between CMAP and F-wave in routine F-wave study. A-waves are independent of stimulus intensity and have a lower amplitude, relatively constant shape and latency in comparison with F-waves. Their amplitude does not change with increasing stimulus intensity from threshold to supramaximal in contrast to H-reflexes [15]. A-waves have been shown to be present in different neuropathy, and they are a pathological sign of demyelinating process [1, 2, 3, 4, 5, 7, 8, 10, 14].

Multiple A-waves are defined as the presence of three or more A-waves in a single nerve [1, 4, 8] and they are a frequent and typical early finding in Guillain-Barre syndrome (GBS) soon after the onset of symptoms [1, 4].

In the present study, multiple A-waves, recorded from frontalis and orbicularis oris muscles in patients with Bell’s palsy are shown and their diagnostic value is estimated.

PATIENTS AND METHODS

Twenty-one males (48.8±19.5, ranged from 13 to 73 years) and 34 females (44.0±16.5, ranged from 10 to 77 years) with Bell’s palsy participated in electrophysiological studies. The control group consisted of 28 subjects (16 women (41.0±16.8, ranged from 11 to 69 years) and 12 men (46.1±14.5, ranged from 20 to 60 years).

A-waves were obtained during conventional F-wave investigation with 16 supramaximal stimuli with a frequency below 1Hz. Electrical stimulation with square wave pulses 0.2 ms in duration was applied via a bipolar surface electrode fixed over the nerve just below the ear and anterior to the mastoid process. The evoked CMAPs of frontalis and orbicularis oris muscles were recorded with concentric needle electrode (diameter of 0.45 mm) at an amplification of 200μV/div. The parameters (latency and amplitude - peak-to-peak) of evoked CMAP were measured. Conduction time (CT) of facial nerve was estimated by latency of evoked CMAP.

A-waves were identified according to the following characteristics: constant amplitude, stable wave form and variation of latency less than 1.5 ms.
All studies were performed using an Esaote Biomedica electromyograph linked to a personal computer for analysis and presentation of the data. Room temperature was between 21°C and 23°C. The investigation had the approval of the Ethics committee of the Medical University, Sofia.

In view of the small number of patients and the skewed distributions of the obtained values of CMAP parameters, the non parametric two-tailed Mann-Whitney U-test was used to determine the significance of differences among the patient groups and control group of healthy subjects. A value of $p < 0.05$ was considered as significant. All values of latency and amplitude were normalised as a ratio between individual values and mean value of healthy subject in order to evaluate the changes. The comparison was made by the Spearman rank correlation test. The results are presented as mean value ± standard deviation of the mean (SEM).

RESULTS

In 50% of patients, the disturbance of two facial nerve branches was different. This was the reason to separate the patients in two main groups (for upper and lower branch). All investigated patients by dependence on the stage of pareses determined according to electromyographic findings were classified in three groups (table 1).

Table 1. The parameters of evoked CMAPs of mm. frontalis and orbicularis oris in patients with Bell’s palsy

<table>
<thead>
<tr>
<th>Group</th>
<th>m.frontalis</th>
<th>m.orbicularis oris</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Latency [ms]</td>
<td>Amplitude [mV]</td>
</tr>
<tr>
<td>I (n=14)</td>
<td>3.57±0.28*</td>
<td>1.31±0.59*</td>
</tr>
<tr>
<td>II (n=26)</td>
<td>4.43±0.31*</td>
<td>0.87±0.56*</td>
</tr>
<tr>
<td>III (n=15)</td>
<td>6.67±1.86*</td>
<td>0.87±0.60*</td>
</tr>
<tr>
<td>Healthy (n=28)</td>
<td>3.89±0.53</td>
<td>2.16±0.58</td>
</tr>
</tbody>
</table>

The parameters were presented as mean value and ± SD of the mean.
The statistically significant differences are marked with asterisks.

The first group included patients with mild palsy of the upper or lower branches of facial nerve. The recorded motor unit action potentials (MUAPs) at voluntary muscle contraction and CT of facial nerve were unchanged. In the second group of patients, with moderate palsy, the recorded MUAPs were polyphasic with significant reduced amplitude, and fibrillation potentials were observed. CT of facial nerve was reduced also. In the patients (third group) with severe palsy, a chronic partial denervation with reinervation (reduced motor pattern with MUAPs of increased amplitude and duration) were observed. In the control group consisting of healthy subjects, the parameters of recorded MUAPs and electromyogram pattern were without changes.
The number of patients with identical damage of two nerve branches was different between the groups and increased with the level of injury – I group – 4 patients (7.3%); II group – 8 patients (14.5%); III group – 11 patients (20%).

Changes of CMAP latency and amplitude were observed in the last two groups of patients for both investigated (frontalis and orbicularis oris) muscles in comparison with those in control healthy subjects group (table 1).

Changes of skewness in CMAP latency and CT distribution in comparison with control were observed in all groups of patients for investigated muscles.

There was a significant difference (p<0.05) in the normalized CMAP parameters (latency and amplitude) for the frontalis and orbicularis oris muscles between investigated patients of all three and control groups (table 1). CT of facial nerve, estimated by latency of recorded CMAP for frontalis and orbicularis oris muscles was significantly slower (p < 0.05) in the second and the third group of patients in comparison with the control group. The changes of CMAP parameters for m. orbicularis oris were larger. The average CMAP latency was 1.14 and was 1.69 times prolonged for m.frontalis or 1.26 and 2.01 times for m.orbicularis oris respectively. Averaged CMAP amplitude of frontalis muscle for all groups was reduced approximately by 50% and 70% for orbicularis oris in comparison with the control group.

In routine F-wave studies, A-waves were not observed in the control group of healthy subjects. On the opposite, in 41 of patients, single and multiple A-waves were elicited (fig. 1A, B).
Fig. 1. Single and multiple A-waves recorded from the mm. frontalis (A) and orbicularis oris (B, C) of patients with Bell’s palsy. 16 consecutive supramaximal stimuli to the facial nerve. Dark arrows show the A-waves. Dashed arrow – suspected A-wave

In 12 (21.8%) of patients, A-waves were not found for m. frontalis and in 14 (25.5%) of patients for m. orbicularis oris. In 2 patients from the third group, A-waves were not found in both investigated muscles. Single A-waves recorded from frontalis muscle were observed in 8 (14.5%), multiple A-waves in 35 (63.6%) of patients. Multiple and single A-waves were elicited in orbicularis oris muscle in 33 (60%) and 8 (14.5%) of patients respectively. The percent of patients with registered multiple A-waves from m.frontalis is statistically significant (p<0.05) in all groups higher than those with recorded single A-wave. For m.orbicularis oris only in second and third groups, there is a statistical significance (p<0.05) (fig. 2A, B).
The symptoms of Bell’s palsy are not specific and it is difficult to determine the etiology by using a wide range of tests. Independently of the etiology, injuries of nerve fibres in patients with Bell’s palsy are existing. It is associated with a disturbance in conduction of excitation along the nerve fibres. The diagnostic value of A-waves can be evaluated by routine F-wave test in which they can be observed. It is the reason to evaluate the stage of paresis by electrodiagnostic findings described...
in part of results. The grading system of Pietersen [11] and that of House and Brackmann [6] are inconvenient for our patients. In the patients with total or complete paralysis, we were not able to register A-waves. Those subjects were removed from our population of patients. Moreover in the patients with normal function, classified as O or I group by Pietersen [11] and House and Brackmann [6], we found the changes of CMAP parameters. On the other hand, it was difficult to determine the individual onset of the disease for all patients. It is the reason to determine our patient groups using first electromyographic findings as a base. In the acute phase of the palsy, usually there are affected all branches [11] but it is possible to be disturbed one or two branches. In our patients, there was an interpersonal difference in the damages of both investigated facial nerve branches and we had determined the degree of the disturbances separately for two branches.

The definition of A-waves used by us is the same as that described by Bischoff et al. [3] and other authors [8, 9, 12, 14]. We have included all A-waves regardless of the generation mechanism to obtain an information on frequency used in routine F-wave tests.

We had not observed a positive correlation between prolonged motor latency and recorded A-waves described by Rowin and Meriggioli [14]. In our patients, CT of lower facial nerve branch was statistically significant longer (p<0.05) than of the upper, but the percent of patients with registered A-wave from m. orbicularis oris was smaller (74.5%) than from m. frontalis (78.2%). Moreover the percent of patients with recorded multiple A-waves from m. frontalis was lower (60%) in comparison with m. orbicularis oris (63.6%).

These differences may be explained partly with the small number of patients and the difficulties connected with differentiation of multiple A-waves from motor responses (fig 1A, B, noted with dashed arrows and C). Because of anatomic structure of m. orbicularis oris, the wave form of CMAP was poliphasic with dispersion. We did not make any attempt to systematically distinguish such data as shown in fig.1C and they were removed. That is the reason for decreasing of the patient’s number.

In spite of obstacles to determine the individual onset of the disease in all patients, this study can be accepted as an examination in the early stages of Bell’s palsy. The higher percent of patients with recorded multiple A-waves in the first and the second group for m. frontalis (fig. 2 A) is statistically significant (p<0.05) compared with the third group that suggests appearing of multiple A-waves in the light cases of palsy (first and second groups).

Registered A-waves in patients with Guillain-Barre syndrome (GBS) are the symptoms of inflammatory nerve lesions [8,13] and a relatively sensitive early indicator for GBS. The demyelination is the crucial underlying pathophysiologic correlate of the supramaximally elicited A-wave [14]. Current studies postulate that mul-

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