Single Fiber Electromyography (SFEMG) in Bell’s palsy: Prognostic Value

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ABSTRACT

Bell’s palsy is a disfiguring disorder that has a great impact on the patient then on the community. Approximately 50% of cases will have essentially complete recoveries in a short time; doubted prognosis awaits the other half. The aim of our study is to delineate the value of single fiber EMG in estimating the degree of degeneration of the facial nerve fibers and to monitor the course of regeneration. Patients & Methods: eighteen patients with Bell’s palsy were included in our study. Thorough clinical neurological examination with meticulous grading of facial nerve paralysis via “House-Brackmann Classification of facial function was done. EMG (conventional and single fiber), nerve excitability test (NET), maximal stimulation test (MST) and nerve conduction velocity (NCV) were done. First evaluation was accomplished within the first two weeks of insult and a second one took place one month after the onset of illness. Results: Mean jitter and percent blocking were found to be significantly increased within 15 days from the onset of paralysis and tended to return back to normal one month later. Amplitude of CMAP was significantly reduced at the onset of illness while percentage of generation was greatly increased, then they improved thereafter. Fiber density was found to be increased after the first two weeks. Conclusion: Single fiber EMG (SFEMG) is shown to contribute information concerning the motor unit that cannot be obtained with other techniques and the method has been applied in both research and routine neurophysiological investigations. SFEMG is complementary to, and does not replace, ordinary concentric needle electrode EMG. (Egypt J. Neurol. Psychiat. Neurosurg., 2005, 42(1): 83-96).

INTRODUCTION

Eye cannot close and constantly weeps, mouth dribbles, speech is interfered with and mastication are lost, Joy, happiness, sorrow, shock, surprise; all the emotions have for their common expression, the same blank stare; all of this refers to a situation called Bell’s palsy.¹

It can strike almost anyone at any age; however, it disproportionately attacks pregnant women and people who have diabetes, influenza, or a cold. M: F ratio is equal, but it is twice as common in females for the 10-19 year age group and 1.5 times more common in males for the >40 age group; left/right sides are equally involved being bilateral in less than 1%; recurrence rate is about 10% and can be ipsi-/bi-lateral; diabetics have 4.5 times more risk to develop Bell's palsy; family history of Bell's palsy is present in 10% of patients.²

Bell’s palsy induces a wide range of facial movement dysfunction from mild paresis to total paralysis. The pathophysiology associated with this injury is suspected to be due to edema within the facial nerve, with maximal injury in the labyrinthine segment.

A carefully planned electrodiagnostic study is paramount in the evaluation of nerve injuries. They are useful for establishing prognosis for return of facial nerve function and for determining endpoint of degeneration by serial testing.

Conventional EMG testing in the acute phase is primarily a complementary test as it is unable to
distinguish a totally neurapraxic injury from a completely degenerated nerve or a regenerating nerve. Early in the disease, EMG will not demonstrate any voluntary motor units, and if voluntary active motor units are present, the nerve is probably intact with incomplete injury. After 10-14 days, fibrillation potentials become evident (degeneration). Then the axons will regenerate through the intact neural tubules allowing complete return of motor function to the muscle fiber innervated by that nerve fiber (reinnervation). Polyphasic potentials are seen during nerve regeneration and may be seen as early as 4-6 weeks after onset of paralysis. EMG may be complementary to NET; MST, and ENoG during the first 3-4 days after injury.  

NET, MST and ENoG are most useful in evaluating acute paralysis while the nerve is in the degenerative phase, however, they may show normal results for the first 3-4 days after nerve injury. This is due to the stimulating and recording electrodes being both distal to the lesion. It takes about 3-4 days for the nerve degeneration to reach the site of stimulation. Also NET, MST, ENoG will only work if the patient has unilateral involvement since the tests are of value in the presence of a "normal" contralateral side for comparison.

Nerve excitability test (NET) and maximal stimulation test (MST) are quantitative measures of the electrical excitability of the peripheral nerves. These tests are often used to assess the prognosis of Bell’s palsy. If they are performed within a few days after the onset of Bell’s palsy, they will give valuable information regarding the eventual prognosis of the condition.

Undoubtedly, there are some types of injuries that may necessitate the use of unconventional studies to adequately assess the degree of axon loss in each individual nerve branch or fascicle.

Single fiber EMG (SFEMG) is a technique to study the microphysiology of an individual motor unit. It can be employed to study the process of denervation and reinnervation. The selectivity of the technique results from the small recording surface (25 um in diameter), which is exposed at a port on the side of the electrode, which is 3 mm from the tip.

Electrophysiologically, the process of reinnervation can be monitored by SFEMG. Fiber density will increase as early as three weeks. The fiber density will continue to increase and will reach a plateau when reinnervation is completed. Early in reinnervation, with early contact of the new axon sprout with the previously denervated muscle fibers, jitter is greatly increased and if transmission fails at this site, concomitant blocking, or neurogenic blocking, can occur. The previously denervated muscle fiber has atrophied and with reinnervation it now conducts impulses at slower speeds. As it recovers, if its muscle fiber propagation velocity becomes variable, this can add to the increased jitter recorded early in reinnervation. With time and maturation of the new axon sprout and new endplate structures, the blocking stops and the jitter reverts towards normal. In some cases it may take 1 to 1 1/2 years for the jitter to revert back to normal, while in some other cases there are individual motor units which never regain normal transmission.

The use of SFEMG provides satisfactory information on the quality and quantity of axonal regeneration following an injury. Misdirect regeneration and developing synkinesia is a problem that can be prevented with early diagnosis and intense rehabilitative course.

The aim of our study is to demarcate the extracranial etiology of facial nerve paralysis, to delineate the value of serial single fiber electromyography in estimating degree of degeneration of fibers in Bell’s palsy and to monitor the course of regeneration and also give hints about clinical and technical diagnostic and prognostic scenarios that outline decision-making and patient treatment protocols in such a disease.

**PATIENTS AND METHODS**

Eighteen patients afflicted by acute isolated cases of Bell’s palsy participated in this research. Onset of illness with Bell’s palsy never exceeded their first two weeks. There were 12 males
(66.6%) and 6 females (33.3%) with a mean age of 33.11±17.39 years.

**Methods:**
All patients had the following assessments and tests:
1- Careful history taking and meticulous neurological examination with thorough cranial nerve evaluation. This narrows the scope of the differential diagnosis.
2- Further clinical grading of the degree of Bell’s palsy was achieved via the “House-Brackmann Classification of Facial Function”: Such grading was accomplished in two sessions; one with the first experience with the patient, and the second session one month from the onset of illness. Table (1) views the classification used in the present research.
3- Electrophysiological study:
   **Equipment:** A reporter ESAOTE BIOMEDICA instrument was used.
   The study was performed in two sessions within the 1st 2 weeks and one month from the onset of illness. In each setting the following tests were done:
   a- *Conventional EMG* was carried using concentric needle electrodes:
      The frontalis and orbicularis oris muscles were tested at rest, during mild & maximum contraction.
   b- *NET:* was performed only on the first visit.
   **Methods:**
   Both facial nerves were stimulated and when minimal twitching of the distal facial muscles as the orbicularis oculi or orbicularis oris occurs, the intensity (strength) of the stimulus should be recorded in milliamperes.
   I. A difference of 4 mA or more in the nerve excitability test between the symptomatic and asymptomatic sides, indicates partial nerve degeneration.
II. Absence of any response beyond 20 mA indicates complete nerve degeneration.
   c- *NCV:* was performed on the affected and unaffected side.
   The facial nerve was stimulated in front of the ear tragus with bipolar electrodes. Responses to maximal electrical stimulation were recorded by concentric needle electrodes in the frontalis and orbicularis oris muscles. The peak-to-peak amplitude of the evoked compound action potential was compared to the amplitude of the normal side. A reduction of the amplitude of the CMAP to 5% or less of normal (95% reduction) is indicative of poor prognosis in Bell’s palsy.

**Data analysis:**
The evoked responses were analyzed for:
I- The amplitude of the compound muscle action potential.  
   Mean amplitude ± SD
II- The latency of the compound muscle action potential: 
   Mean latency ± SD
III- The percentage of degeneration
IV- The duration of the compound muscle action potential.

**d- SFEMG**
SFEMG studies were performed in the frontalis muscle. The single fiber needle was inserted into the weakened slightly contracting muscle with the subject comfortably seated. A clear, high-pitched sound of a single-fiber discharge indicates a suitable site for further study. Careful rotation, advancement or retraction of the needle maximizes the potential on the oscilloscope. The needle is then further advanced until another single fiber action potential is recorded; at least 20 peak pairs were recorded.

**Recommended criteria:**
   a- Peak to peak amplitude > 200 uV.
b- Rise time from positive to negative peak <300 us.

A constant waveform of the successive tracings confirms a suitable single fiber discharge for the following automatic analysis:
1- FD (fiber density): the number of simultaneously firing single muscle fibers is counted for at least 5ms interval after the triggering spike.
2- MISI (mean inter spike interval): was calculated by dividing the total duration by the number of intervals (number of spikes minus 1).
3- MCD (mean consecutive difference or jitter): it is expressed as the mean value of consecutive differences; it represents the degree of variability in the interpotential interval (IPI).
4- MSD (mean sorted interval difference): it is a parameter similar to the MCD where the differences are calculated on potentials of non consecutive traces.
N.B.: we use MSD if the MCD/MSD exceeds 1.25.
5- Range: it represents the range of variability of the ISI.
6- Blocking: it represents the percent value of blocks.
7- R-RV: it represents the percent value of the R-R variability.
8- Firing rate: is the firing rate of a single fiber discharge.
9- MIDI (mean interdischarge interval): It represents the mean value of differences in conduction time from the common branching point to each fiber within the same motor unit.

Statistical analysis:
Data were included in a database and analyzed by means of statistical software package namely SPSS Windows V.8. All variables were expressed as means and standard deviations (SD).

Two-Independent-Samples test was performed to compare the diabetic and non-diabetic groups on each variable and Wilcoxon Rank W test to compare the distributions of two variables for a single group. Pearson’s correlation was also performed to measure how each pair of variables are related; correlation coefficients range in value from -1 (negative correlation) and +1 (positive correlation).

RESULTS

This study was carried out in 18 patients with acute Bell’s palsy (12 males and 6 females); their ages ranged from 9 to 65 years with a mean age of 33.11±17.39; 5 (27.8%) of cases were diabetic. We classified our sample based on the presence of diabetes into group I and group II (Table 8).

I- Clinical data (Tables 1, 2):
According to House Brackmann grading, 7 patients (38.9%) had grade III, 8 (44.4%) had grade IV, and 3 (16.7%) patients had grade V. 11 (61.1%) of cases had left facial palsy, 5 (27.8%) have right paresis and only 2 cases (11.1%) had bilateral affection.

II- Electrophysiological data:
Table (3) is a summary table describing the sensitivity of each test in detecting signs of denervation and early signs of reinnervation within the first two weeks.
a- On EMG testing:
* Signs of denervation were present in only 1 case (5.5%).
* Broad polyphasic MUPs were detected in 4 cases (22.2%).
b- On single fiber EMG:
* FD was normal (<2) in 13 cases (72.2%).
* Mean jitter was abnormal (>45 us) in 13 cases (72.2%).
* Mean sorted interval difference was abnormal (>45 us) in 13 cases (72.2%).
* Percentage of blocking was abnormal (>40%) in 16 cases (88.8%).

**c**- On nerve excitability test (NET):
* A side-to-side difference > 4 mA was found in 7 cases (38.8%).

**d**- On NCV, percentage of degeneration >90% was demonstrated in 17 cases (94.4%).

Table (4) correlates the age, duration of illness and degree of paralysis with the electrophysiological parameters:
* A highly statistically significant positive correlation was found between the degree of clinical paralysis and reduction in the interference pattern (P<0.002).
* A statistically significant positive correlation was also found between the degree of clinical paralysis and the percentage of degeneration (P<0.01), the jitter (MCD) (P<0.05), MSD (P<0.04), FD (P<0.04) and the percentage of blocking (P<0.03).

Table 5 (I & II) compares the electrophysiological parameters (Conventional EMG and NCV on the two sessions: Group A (represents the first visit) and group B (represents the second one):
* The percentage of polyphasic MUPs was significantly increased (P<0.05), while the reduction in the interference pattern was significantly decreased (P<0.03).
* The latency of the compound motor action potentials (CMAP) was unaffected while its amplitude and duration were significantly increased (P<0.02 & 0.04). The percentage of degeneration also significantly improved (P<0.03).

Table 6 compares the parameters of single fiber EMG studies on the two sessions: Group A (represents the first visit) and group B (represents the second one):
* The percentage of blocking highly significantly decreased (P<0.003), while the FD was extremely significantly increased (P<0.001).
* The R-R variability was significantly reduced (P<0.02).

Table (7) correlates the electrophysiological parameters of regeneration in conventional EMG and conduction studies (% of polyphasic MUPs, % of degeneration, NET and SFEMG parameters (FD, mean jitter and percent blocking):
* A highly statistically significant positive correlation was found between the percentage of polyphasic MUPs and the fiber density (P<0.004).
* A statistically significant positive correlation was found between the percentage of polyphasic MUPs and the mean jitter (P<0.03).

Table (8) compares the clinical data in Group I (diabetic) and group II (non-diabetic).

Table 9 (I & II) compares the electrophysiological parameters (Conventional EMG and NCV in Group I (diabetic) and group II (non-diabetic):
* There were no statistically significant difference between Group I & II.

Table (10) compares the parameters of single fiber EMG studies in Group I and group II:
* There were also no statistically significant difference between Group I & II.
Table 1. House-Brackmann Classification of facial function.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Normal</td>
<td>Normal facial function in all areas.</td>
</tr>
</tbody>
</table>
| II. Mild dysfunction | Gross  
| | o Slight weakness noticeable on close inspection  
| | o May have slight synkinesis  
| | o At rest, normal symmetry and tone  
| | **Motion**  
| | o Forehead-moderate-to-good function  
| | o Eye-complete closure with minimal effort  
| | o Mouth-slight asymmetry  |
| III. Moderate dysfunction | Gross  
| | o Obvious but not disfiguring difference between the two sides  
| | o Noticeable but not severe synkinesis, contracture, or hemifacial spasm.  
| | o At rest, normal symmetry and tone.  
| | **Motion**  
| | o Forehead-slight-to-moderate function  
| | o Eye-complete closure with effort  
| | o Mouth-slightly weak with maximal effort  |
| IV. Moderately severe dysfunction | Gross  
| | o Obvious weakness and/or disfiguring asymmetry.  
| | o At rest, normal symmetry and tone.  
| | **Motion**  
| | o Forehead-none  
| | o Eye-incomplete closure.  
| | o Mouth-asymmetry with maximal effort  |
| V. severe dysfunction | Gross  
| | o Only barely perceptible motion  
| | o At rest, asymmetry  
| | **Motion**  
| | o Forehead-none  
| | o Eye-incomplete closure.  
| | o Mouth-slight movement  |
| VI. Total paralysis | No movement |

Table 2. Grading of our patients according to House-Brackmann classification:

<table>
<thead>
<tr>
<th>House Brackmann classification</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Grade II</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Grade III</td>
<td>7</td>
<td>38.9</td>
</tr>
<tr>
<td>Grade IV</td>
<td>8</td>
<td>44.4</td>
</tr>
<tr>
<td>Grade V</td>
<td>3</td>
<td>16.7</td>
</tr>
<tr>
<td>Grade VI</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 3. Electrophysiological tests carried for our patients.

<table>
<thead>
<tr>
<th></th>
<th>No. of patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EMG:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At rest</td>
<td>1</td>
<td>5.5%</td>
</tr>
<tr>
<td>- Mild contraction (presence of polyphasics)</td>
<td>4</td>
<td>22.2%</td>
</tr>
<tr>
<td><strong>NET</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(percentage of degeneration &gt;= 90%)</td>
<td>7</td>
<td>38.8%</td>
</tr>
<tr>
<td><strong>NCV:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>94.4%</td>
</tr>
<tr>
<td><strong>Single fiber EMG:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- FD (&lt;2)</td>
<td>13</td>
<td>72.2%</td>
</tr>
<tr>
<td>- Mean jitter (&gt;45us)</td>
<td>13</td>
<td>72.2%</td>
</tr>
<tr>
<td>- MSD (&gt;45us)</td>
<td>16</td>
<td>88.8%</td>
</tr>
<tr>
<td>- Blocking (&gt;40%)</td>
<td>16</td>
<td>88.8%</td>
</tr>
</tbody>
</table>

Table 4. Correlates the age, duration of illness and degree of paralysis with the electrophysiological parameters.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Age</th>
<th>Duration of illness</th>
<th>Degree of paralysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>P</td>
<td>r</td>
</tr>
<tr>
<td><strong>A. EMG</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. At rest</td>
<td>.17</td>
<td>.4</td>
<td>.48</td>
</tr>
<tr>
<td>2. Mild contraction (Polyphasics %)</td>
<td>-.48</td>
<td>.04*</td>
<td>.24</td>
</tr>
<tr>
<td>3. Max. contraction (IPs)</td>
<td>.18</td>
<td>.46</td>
<td>-.03</td>
</tr>
<tr>
<td><strong>B. NCV studies:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Latency (ms)</td>
<td>.04</td>
<td>.85</td>
<td>.33</td>
</tr>
<tr>
<td>- Amplitude (uV)</td>
<td>-.05</td>
<td>.81</td>
<td>-.28</td>
</tr>
<tr>
<td>- % of degeneration</td>
<td>-.16</td>
<td>.53</td>
<td>.46</td>
</tr>
<tr>
<td>- duration of CMAP (ms)</td>
<td>.31</td>
<td>.19</td>
<td>-.00</td>
</tr>
<tr>
<td><strong>C. Single fiber EMG</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Jitter (MCD)</td>
<td>-.01</td>
<td>.95</td>
<td>.03</td>
</tr>
<tr>
<td>- MISI</td>
<td>.19</td>
<td>.43</td>
<td>.30</td>
</tr>
<tr>
<td>- MSD</td>
<td>-.40</td>
<td>.1</td>
<td>.31</td>
</tr>
<tr>
<td>- Range</td>
<td>.29</td>
<td>.23</td>
<td>.40</td>
</tr>
<tr>
<td>- Blocking</td>
<td>.02</td>
<td>.93</td>
<td>-.04</td>
</tr>
<tr>
<td>- R-RV</td>
<td>.11</td>
<td>.65</td>
<td>-.37</td>
</tr>
<tr>
<td>- FD</td>
<td>-.05</td>
<td>.83</td>
<td>.41</td>
</tr>
<tr>
<td>- Firing rate</td>
<td>-.16</td>
<td>.51</td>
<td>.03</td>
</tr>
<tr>
<td>- MIDI</td>
<td>.03</td>
<td>.89</td>
<td>.02</td>
</tr>
</tbody>
</table>

nP<0.05 (significant), **P<0.01 (highly significant), r-value: Pearson correlation coefficient.
Table 5 I and II. Compares the electrophysiological parameters (Conventional EMG and NCV on the two sessions: Group A (represents the first visit) and group B (represents the second one).

Table 5 I:

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A</th>
<th>Group B</th>
<th>Z</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1- at rest(signs of denervation)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>1</td>
<td>5.6%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Absent</td>
<td>17</td>
<td>94.4%</td>
<td>18</td>
<td>100%</td>
</tr>
<tr>
<td>2-mild contraction (Polyphasics %)</td>
<td>4</td>
<td>27.2%</td>
<td>11</td>
<td>61.1%</td>
</tr>
<tr>
<td>3- Max.contraction (IPs)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mildly reduced</td>
<td>8</td>
<td>44.4%</td>
<td>12</td>
<td>66.7%</td>
</tr>
<tr>
<td>Moderately reduced</td>
<td>5</td>
<td>27.8%</td>
<td>5</td>
<td>27.8%</td>
</tr>
<tr>
<td>Grossly reduced</td>
<td>5</td>
<td>27.8%</td>
<td>1</td>
<td>5.6%</td>
</tr>
</tbody>
</table>

* P< 0.05 (significant)

Table 5 II:

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A</th>
<th>Group B</th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. NCV studies:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Latency (ms)</td>
<td>4.2±1.6</td>
<td>2.9±0.9</td>
<td>-1.2</td>
<td>0.2</td>
</tr>
<tr>
<td>- Amplitude (uV)</td>
<td>0.6±0.6</td>
<td>1.1±0.9</td>
<td>-2.3</td>
<td>0.02*</td>
</tr>
<tr>
<td>- % of degeneration</td>
<td>60.4±28.4</td>
<td>46.9±26</td>
<td>-2.1</td>
<td>0.03*</td>
</tr>
<tr>
<td>- Duration(ms)</td>
<td>7.2±5.6</td>
<td>10.3±5.4</td>
<td>-2.0</td>
<td>0.04*</td>
</tr>
</tbody>
</table>

* P< 0.05 (significant)

Table 6. Compares the parameters of single fiber EMG studies on the two sessions: Group A (represents the first visit) and group B (represents the second one).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A</th>
<th>Group B</th>
<th>Z</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single fiber EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Jitter(MCD)(us)</td>
<td>175.0</td>
<td>174.02</td>
<td>77.0</td>
<td>126.73</td>
</tr>
<tr>
<td>- MISI (us)</td>
<td>1629.55</td>
<td>1922.66</td>
<td>878.2</td>
<td>895.16</td>
</tr>
<tr>
<td>- MSD (us)</td>
<td>349.55</td>
<td>386.74</td>
<td>159.6</td>
<td>246.13</td>
</tr>
<tr>
<td>- Range</td>
<td>519.74</td>
<td>697.96</td>
<td>186.47</td>
<td>369.4</td>
</tr>
<tr>
<td>- Blocking (%)</td>
<td>23.27</td>
<td>13.43</td>
<td>8.2</td>
<td>9.0</td>
</tr>
<tr>
<td>- R-RV (%)</td>
<td>49.77</td>
<td>41.4</td>
<td>20.8</td>
<td>39.29</td>
</tr>
<tr>
<td>- FD</td>
<td>1.88</td>
<td>3.67</td>
<td>3.2</td>
<td>9.4</td>
</tr>
<tr>
<td>- Firing rate (%)</td>
<td>12.2</td>
<td>3.87</td>
<td>10.6</td>
<td>4.9</td>
</tr>
<tr>
<td>- MIDI (us)</td>
<td>105.11</td>
<td>55.56</td>
<td>86.93</td>
<td>43.99</td>
</tr>
</tbody>
</table>

*** P< 0.001 (extremely significant)
** P< 0.01 (highly significant)
* P< 0.05 (significant)
Table 7. Correlates the electrophysiological parameters of regeneration in conventional EMG and conduction studies (% of polyphasic MUPs, % of degeneration, NET and SFEMG parameters (FD, mean jitter and percent blocking).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Single fiber EMG parameters</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>FD</td>
<td>Mean</td>
<td>Jitter</td>
<td>% blocking</td>
</tr>
<tr>
<td>A- EMG studies:</td>
<td></td>
<td>r</td>
<td>P</td>
<td>r</td>
<td>P</td>
</tr>
<tr>
<td>- % of polyphasic MUPs</td>
<td></td>
<td>-.25</td>
<td>.3</td>
<td>-.01</td>
<td>.9</td>
</tr>
<tr>
<td>B- NCV</td>
<td></td>
<td>-.04</td>
<td>.8</td>
<td>.14</td>
<td>.6</td>
</tr>
<tr>
<td>- % of degeneration</td>
<td></td>
<td>.66</td>
<td>0.004 **</td>
<td>.53</td>
<td>0.03*</td>
</tr>
</tbody>
</table>

*P<0.05 (significant), **P<0.01 (highly significant), r-value: Pearson correlation coefficient.

Table 8. Compares the clinical data in Group I (diabetic) and group II (non-diabetic).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I</th>
<th></th>
<th></th>
<th>Group II</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>T</td>
<td>P</td>
<td></td>
</tr>
<tr>
<td>1- Age(years)</td>
<td>47.8</td>
<td>11.21</td>
<td>27.46</td>
<td>16.91</td>
<td>-2.5</td>
<td>0.02*</td>
<td></td>
</tr>
<tr>
<td>2- Duration of illness</td>
<td>9.0</td>
<td>4.5</td>
<td>14.8</td>
<td>9.4</td>
<td>1.3</td>
<td>0.2</td>
<td></td>
</tr>
</tbody>
</table>

* P<0.05 (significant)

Table 9 I and II. Compares the electrophysiological parameters (Conventional EMG and NCV in Group I (diabetic) and group II (non-diabetic).

Table 9 (I):

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (5 cases)</th>
<th></th>
<th></th>
<th>Group II (13 cases)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>Z</td>
<td>P</td>
<td></td>
</tr>
<tr>
<td>A. EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1- At rest(signs of denervation)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>1</td>
<td>20%</td>
<td>1</td>
<td>7.7%</td>
<td>0.5</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>4</td>
<td>80%</td>
<td>12</td>
<td>92.3%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2- Mild contraction (Polyphasics %)</td>
<td>0</td>
<td>0%</td>
<td>4</td>
<td>30.8%</td>
<td>.2</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>3- Max. contraction (IPs)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mildly reduced</td>
<td>1</td>
<td>20%</td>
<td>7</td>
<td>53.8%</td>
<td>-1.8</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>Moderately reduced</td>
<td>1</td>
<td>20%</td>
<td>4</td>
<td>30.8%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grossly reduced</td>
<td>3</td>
<td>60%</td>
<td>2</td>
<td>15.4%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* P< 0.05 (significant)

Table 9 (II):

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group A Mean ± SD</th>
<th>Group B Mean ± SD</th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. NCV studies:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Latency (ms)</td>
<td>4.7±2.9</td>
<td>4.0±0.9</td>
<td>-.7</td>
<td>0.4</td>
</tr>
<tr>
<td>- Amplitude (uV)</td>
<td>0.5±0.4</td>
<td>0.6±0.7</td>
<td>0.3</td>
<td>0.7</td>
</tr>
<tr>
<td>- % of degeneration</td>
<td>48.6±30.8</td>
<td>65.3±27.2</td>
<td>1.1</td>
<td>0.2</td>
</tr>
<tr>
<td>- Duration (ms)</td>
<td>7.7±6.2</td>
<td>7.0±5.6</td>
<td>-.2</td>
<td>0.8</td>
</tr>
</tbody>
</table>

* P<0.05 (significant)
Table 10. Compares the parameters of single fiber EMG studies in Group I and group II.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I</th>
<th>Group II</th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single fiber EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- jitter(MCD)(us)</td>
<td>186.4</td>
<td>170.6</td>
<td>- .16</td>
<td>0.8</td>
</tr>
<tr>
<td>_ MISI(us)</td>
<td>1245.6</td>
<td>1777.2</td>
<td>- .51</td>
<td>0.6</td>
</tr>
<tr>
<td>- MSD(us)</td>
<td>223.6</td>
<td>398.0</td>
<td>.85</td>
<td>0.4</td>
</tr>
<tr>
<td>- Range</td>
<td>684.8</td>
<td>456.2</td>
<td>- .61</td>
<td>0.5</td>
</tr>
<tr>
<td>- blocking(%)</td>
<td>25.2</td>
<td>22.5</td>
<td>- .38</td>
<td>0.7</td>
</tr>
<tr>
<td>- R-RV(%)</td>
<td>59.8</td>
<td>45.8</td>
<td>- .63</td>
<td>0.5</td>
</tr>
<tr>
<td>- FD</td>
<td>1.6</td>
<td>2.0</td>
<td>1.1</td>
<td>0.2</td>
</tr>
<tr>
<td>- firing rate(%)</td>
<td>11.7</td>
<td>12.4</td>
<td>.35</td>
<td>0.7</td>
</tr>
<tr>
<td>- MIDI (us)</td>
<td>98.8</td>
<td>107.53</td>
<td>.29</td>
<td>0.7</td>
</tr>
</tbody>
</table>

* P< 0.05 (significant)

DISCUSSION

There have been no extensive studies in patients of the time course and dynamics of denervation and reinnervation following nerve trauma, particularly in the early stages.

Our study was designed to follow the course of reinnervation aiming at better management of patients afflicted by Bell's palsy.

Eighteen patients with acute facial palsy were studied, 7 cases had grade III, 8 cases had grade IV and 3 cases had grade V; according to House-Brackmann grading of facial palsy.

The process of degeneration and reinnervation of facial nerve fibers was monitored over one month from the onset of illness; five out of the 18 cases are diabetic under insulin therapy. Diabetics have 4.5 times more risk to develop Bell's palsy. Left/right side's affection was unequally involved in our sample as 11 cases had left sided affection, 5 cases had right one and the remaining 2 cases had bilateral affection.

Conventional EMG during the first two weeks gave little informative data, signs of denervation were detected in only 1 case, presence of polyphasic MUPs was found in only 4 (22.2 %) cases, noting the onset of reinnervation, the remaining cases showed broad MUPs. Although, this could have a definite value in identifying an incomplete lesion despite the clinical absence of facial function, needle EMG is not a reliable indicator of prognosis. The presence of some motor units in cases with paralysis that appears with visual inspection to be complete, is of particular prognostic value when a facial palsy follows surgery; the survival of some units indicates the nerve is in continuity.

By one month, the percentage of polyphasicty was increased, as it was detected in 11(61.1 %) cases. Polyphasic MUPs suggest desynchronized discharge or drop-off of individual fibers.

There was also a statistically significant improvement in the degree of reduction of the interference pattern (P<0.03) indicating maturation of new axon sprouts and that the muscle fibers now discharge more synchronously.

The reduced interference patterns due to loss of some of the motor fibers were found to be highly significantly correlated with the degree of paralysis. The recruitment interval is actually a sensitive index of weakness.

Nerve excitability test (NET) measures early evidence of nerve degeneration as the earliest evidence of degeneration of the nerves after injury is the failure of the nerve to respond to electrical stimulation below the site of injury. This test was performed as soon as possible after the
third day after the onset. Our cases were studied
within the first two weeks, 3 out of the 18 cases
before the 3rd day, and the test was impaired in
only 7 cases (38.8%). Some authors claimed that
in some patients (11%), nerve excitability was
unimpaired initially, and usually happened after
the tenth day, giving erroneous good prognosis, so
we must repeat the NET after the tenth day and
this may be due, in few patients to degeneration
of the facial nerve continuing within the first
week.

Most studies found that the CMAP amplitude
comparison is the most useful test for formulating
a prognosis.

In our study, a reduction of the amplitude of
the CMAP to 10 % or less of normal(90%
reduction) was found in 17 out of 18 studied cases
indicating poor prognosis and this in agreement
with most studies.

Eitersen2 found that in the acute stage of
Bell’s palsy, with a reduction to 10% or less of
the contralateral muscle, recovery took six months
to a year and the final result was poor.

Moreover, some authors claimed that
surgical procedures performed when axonal
degeneration has reached 95-100% within 1-14
days after onset, significantly improve the
recovery of facial movements.9

On monitoring the amplitude of CMAP over one
month, we noticed that it significantly increased
with substantial improvement in the percentage of
degeneration. This could be of prognostic value,
indicating the progression of reinnervation.

It also showed a significant negative
relation with the degree of paralysis. The
amplitude of CMAP is proportional to the number
of intact motor axons. The degree of axonal loss
has direct implications for assessing the degree of
damage and estimating the prognosis and the time
required for recovery.

The latency of the CMAP was found to be of
no prognostic value.

The duration is another factor that could
indicate the dynamics of reinnervation. It was
significantly increased over the first month.
Increased duration is a good index of the motor
unit territory. It probably results from usual
variability in length, conduction time of
regenerating axon terminals and membrane
excitability rather than the enlarged territory.

The use of SFEMG gives information about
changes in the topography of the motor unit and in
the function of transmission in terminal nerve,
motor end plate and muscle fiber. All of this
assists in drawing better management of patients
afflicted by such illness, that even may require
surgical interference in some cases with more than
90% degeneration of the facial nerve fibers. In
such cases, time factor plays a major role in
terminating a devastating problem.10

The parameters of fiber density, mean jitter,
and percent blocking must each be followed and
related to the type of injury to determine the stage
of reinnervation.

During the first two weeks of illness, mean
jitter was found to be increased in 13 cases ,it was
significantly positively correlated with the degree
of paralysis, however ,it showed a significant
reduction after the first month. These findings
indicate ongoing reinnervation, and the increased
jitter is probably because of the functional
immaturity of the newly formed motor-end-plates,
both pre-and post-synaptically.

The time to the peak of abnormality of jitter
values will be dependent upon many factors
including the number and rate of regrowth of
returning axons. The peak may be reached before
the second month but may require 6 months which
is about the time it takes for the motor endplate to
become histologically and histochemically
normal.11

The marked abnormality of jitter during
reinnervation suggests that newly formed
immature end-plates were not capable of
sustaining normal neuromuscular junction (NMJ).
The fall in FD and jitter after maximum function
has returned suggests that the original axons had
resumed function and re-established innervation to
the muscle, after which point collateral sprouting
at least partially disappeared resulting in
remodeling of the motor units.
Janice & Donald⁷ reported that the maximum increase in mean jitter following facial palsy occurred at 37 days, persisted until 67 days, then returned toward normal.

Muscle fiber propagation slows substantially upon firing because successive action potentials occur in the relative refractory period of the muscle. This delay may differentially affect the activation of two muscle fibers, depending on the lengths of their respective axon terminals. In general, a long IPI and rapid firing rates tend to increase jitter because these factors induce physiologic slowing that influences two muscle fibers differently.¹²

As a rule, the firing rate affects jitter if the IPI exceeds 4 ms, a computer can sort the IPI on the basis of the interdischarge interval (IDI) to calculate the corrected MCD, termed mean sorted interval difference (MSD). If firing rate has not affected jitter, MCD/MSD=1. If the ratio exceeds 1.25, one must use MSD instead of MCD, because the firing rate has influenced jitter.¹³

As in our sample, the MCD/MSD exceeded 1.25, so we used MSD to determine the jitter values. They showed a significant increase in 16 out of the 18 studied cases, eventually, by 30 days they had again reduced to about 50% of the value.

Firing rate seems to have also a significant value. The effects of firing rate on jitter may be an indicator of the type of underlying pathology.

In postsynaptic defect, the rapid firing rate increases jitter even with an IPI of less than 4 ms, on the other hand, in presynaptic disorders, jitter increases at slow firing rates and decreases at fast rates.

We found a concomitant increase in the firing rate and mean jitter at the onset of illness within the first 15 days, with subsequent reduction thereafter. This indicates the presence of a postsynaptic defect.

The mean interpotential interval (MIPI) was noted to be significantly increased (>1ms), then it showed a tendency toward a significant reduction over the first month of paralysis. Early with reinnervation, the newly formed axon sprouts with previously denervated muscle fibers, conduct impulses at slower speeds. As they recover, if their muscle fiber propagation velocity becomes variable, this can add to the increased MIPI recorded early in reinnervation that reverts back to normal with maturation of the new axon sprouts and new endplate structures.¹⁴

FD was found to be normal within the first two weeks and significantly increased thereafter. This in agreement with the previous serial single-fiber EMG studies carried by Stalberg¹⁰ in the frontalis muscle who reported increased jitter with normal fiber density 15 days after facial nerve trauma.

Fiber density is defined as the mean number of associated single fiber potentials that fire almost synchronously with the initially identified potential. It provides a measure of muscle fiber clustering, rather than the total number of muscle fibers within a motor unit. By definition, the lowest possible value is 1.0. An increase in fiber density usually indicates the presence of collateral sprouting. It rivals histochemical fiber grouping in identifying rearrangements within the motor unit.⁶

Increase in jitter without concurrent increase in FD 15 days after partial injury demonstrates that transmission in the peripheral portion of the motor nerve or the NMJ was abnormal during the period of denervation before reinnervation began. The increase in FD at 21 days indicates that functional contact has been made between collateral sprouts and muscle.

Jitter is defined as the degree of variability in the IPI. Sometimes after increases in the IPI, the second potential fails to appear. This phenomenon is referred to as blocking; blocking in more than one fiber or jitter values exceeding 55 us constitutes an abnormality in any muscle. Jitter values, typically beyond 80-100us, precede the transmission block.¹³
In our study, the percent blocking was increased at the onset of illness, in 13 out of the 18 studied cases and showed a significant reduction after one month. This is a sign of functional improvement and can serve as a guide when estimating the age of reinnervation potentials in different neuropathies. Our results are in agreement with Stalberg who recorded increased jitter and blocking at one month.

Early in reinnervation, blocking is frequently seen at the branching site of the new axon sprout from its parent axon. If transmission fails at this site, concomitant blocking or neurogenic blocking can occur if the new axon sprout has branched to contact more than one muscle fiber. With time and maturation of the new axon sprout and new enplate structures, the blocking stops and the jitter reverts towards normal.

FD and blocking were also found to be significantly positively correlated with the degree of paralysis.

On evaluating the effects of diabetes in patients with Bell’s palsy, we found only a significant difference regarding the age and degree of paralysis, however, it showed no statistically added abnormalities on the electrophysiological parameters. One possible explanation, supported by histochemical findings, is that, in diabetic neuropathy, the degenerative process is mainly restricted to the myelin sheath but in the mixed motor and sensory neuropathies both the myelin and the axons are destroyed (Wallerian degeneration), this axonal degeneration causes denervation followed by collateral reinnervation of the muscle fibers.

From this study, it appears that the percentage of degeneration, percent blocking and mean jitter seem to be the most sensitive indicators of the process of denervation and reinnervation as their abnormalities were recorded as early as 15 days after onset of paralysis. Fiber density (FD) will be elevated as early as four weeks after onset. These abnormalities can be used to note the onset of reinnervation and to allow better management of patients with Bell’s palsy.

**REFERENCES**


الملخص العربي

تعتبر إصابات العصب الساقع من أكثر الأمراض المقلقة من الناحية التجميلية للمريض، وهذا من المؤكد له تأثيره على المجتمع أيضا.

أجريت هذه الدراسة كمحاولة لتشخيص ومتابعة هذا المرض باستخدام أحد الطرق الحديثة والتقنية لتخطيط الكهربائي للعضلات عن طريق فحص اللياقة العضلية الأحادية خلال الشهر الأول من الإصابة حتى يمكن تحديد نسبة الإصابة ومتابعة مراحل إعادة بناء العصب.

أجريت هذه الدراسة على 18 مريضا بعوام من اتصلال العصب الساقع وقد أجريت لهم الفحوص الآتية:
1- فحص الكهربائي للجهاز العصبي مع تحديد نسبة الإصابة تعا تطبيق هاوس بريكمان للعصب الساقع
2- التخطيط الكهربائي للعضلات (فحص المفصلية وفحص للعصبية الأحادية)
3- مدى حساسية العصب للتنبيب الكهربائي
4- سرعة توصيل العصب (السليم والمصاب).

وقد أجريت هذه الفحوصات في جلستين الأولى خلال أول 15 يوم من الإصابة والجلسة الثانية بعد شهر من الإصابة.

وأوضحنا أخيرا إصابات كهربائية في اتصلالية العصبية الأحادية في تشخيص ومتابعة اتصلال العصب الساقع و من خلال تحليل النتائج وجد أن هناك تباين واضح في سرعة توصيل اللياقة العضلية الأحادية المتبعة ومتاوبة وزيادة نسبة الليفة المفصلية الأحاديةExpr. في أول 15 يوم من بداية الإصابة. تم بدأ التحسين تدريجيا بعد ذلك.

كما أظهرت النتائج أيضا حساسية ارتفاع الموضع الكهربائي المفصلية للعصبية ونسبة إصابتها مقارنة بالجهة السليمة في تشخيص توزيع نسبة الإصابة ومؤشرات إعادة بناء العصب.

ووجد أيضا أن هناك علاقة موجبة بين درجات الإصابة الإكلينيكية ونتائج الاستبانات السيرولوجية الكهربائية.

ويصبح أيضا مدى أهمية التشخيص المبكر وتحديد نسبة الإصابة في تحديد مدى استقامة المريض من الدخول الجراحي حيث أوضحنا عدد المريض من اتصلال العصب الساقع يستفيد من الدخول الجراحي في أول 15 يوم فقط من الإصابة.