Nerve Conduction Studies In Guillain Barre’ Syndrome
L D Parmar,, V Doshi, S K Singh

Citation

Abstract
BACKGROUND: NCS are objective methods for diagnosis, quantification and classification of polyneuropathies. Electrophysiology is most important to confirm GBS in all its forms.
Aim: To study early characteristics of NCS in GBS.
Research design: Cross sectional, analytical
Material & Methods: NCS performed on 49 GBS participants were retrospectively analyzed. Different parameters motor, sensory, late waves studied & compared with literature.
Statistics: Descriptive statistics, ANOVA, correlations.
Results: Age range 1-82 yrs. 76% males, AIDP form (93.88%) predominates, CMAP median bilaterally moderately associated with muscle grades p value 0.005 & 0.006, CMAP & F disturbance severe and predominant feature. 41.86 % H- reflex unrecordable. 34.69% showed sensory abnormalities. Age group ≤ 3 years showed similar pattern. 3 children showed in excitability. Abnormality in number of variables of nerves in combination is likely pattern.
Conclusions: Early NCS pattern emerged similar to several studies.

INTRODUCTION
Polio has been eradicated in most parts of the world and presently Guillain-Barré syndrome (GBS) is the most common cause of acute flaccid paralysis, severe generalized paralysis1, 2, 3,4,5,6, 8, 17, 30 Guillain-Barré syndrome (GBS), or better Guillain-Barré-Strohl syndrome to recognize the three authors responsible for the first description of this disorder. Georges Guillian, Jean Alexander Barre’ and Andre’ Strohl in 1916 related a benign acute poly-neuritis in three soldiers, with increase in protein levels without pleocytosis (albumin-cytological dissociation).9, 10, 11, 12
The annual incidence of GBS has been reported to be relatively uniform between 0.4 and 4 cases per 100000 per year throughout the world.1 3, 4, 5, 7, 12, 14, 15, 22, 24, 29, 30, 31 But the most recent and careful population based studies report an incidence of 1.2 – 1.9 per 100000 4, 13, 15, 20, 27, 28, 29. The lifetime likelihood of any individual acquiring GBS is 1:1000.

It is now appreciated that GBS is not a single disorder but again a syndrome of several types of acute immune-mediated polyneuropathies. The most commonly proposed mechanism for the development of autoimmune disease is molecular mimicry (Yuki, 2005). Molecular mimicry refers to the situation where the pathogen and host share nearly identical antigens, which induces an antibody and T cell immune response that is cross reactive.5,7,8,10,11,12,13,14,15,19,22,23,27,28.
The onset of symptoms can either be acute or sub acute. Gradual recovery takes place after a plateau phase. The disease usually progresses over 2-4 weeks.5 The condition reaches its nadir by 2 weeks in most cases and in 4 weeks in nearly all.15, 18, 22 Current diagnostic criteria include <4 weeks of progression to clinical nadir7, 47

Electrophysiology represents the most important laboratory study to confirm the diagnosis of GBS in all its forms. 5, 10, 11, 13, 15, 19, 21, 22, 25, 32, 33, 34, 35, 36, 37, 38,39,40,41,42,43,44 Electrophysiological testing must be done as early as possible after presentation and should be repeated on a weekly basis to further confirm diagnosis and for prognostic purposes.13 Another important aspect of studying the nerves early is that it provides the best chance to allow differentiation between demyelinating and axonal forms of GBS. In severe cases of demyelinating GBS, axonal loss will occur over time and, as it does, nerves become inexcitable. Presence of early motor nerve inexcitability represents a reliable marker of axonal polyradiculoneuropathy.13
AIM AND OBJECTIVE

AIM OF THE STUDY:
The aim of the study was to analyze the NCS data of the clients confirmed as GBS.

OBJECTIVE OF THE STUDY:
The objective was to study the abnormality in various variables of motor, sensory & late waves including:
- Motor nerves all four limbs / all that are accessible
- Sensory nerves / all that are accessible
- F- Waves
- H- Reflex

MATERIALS AND METHODS

The study was conducted at ‘ K M Patel Institute of Physiotherapy, Shree Krishna Hospital, Karamsad, approved by the Human Research Ethics Committee of the institute ‘ Charutar Arogya Mandal’.

RESEARCH DESIGN: Cross sectional, analytical.

INCLUSION CRITERIA:
- The patients with provisional clinical diagnosis of GBS referred for NCS
- The electrophysiological data of all the patients referred for NCS after they were confirmed as GBS was taken for analysis.

EXCLUSION CRITERIA:
- Diabetes
- Alcoholism
- Any trauma affecting muscles or nerves
- Renal or metabolic dysfunctions
- Peripheral vascular diseases
- Myopathy
- Motor neuron disorders
- Any genetic or other disorders affecting nerve and muscle.

METHODOLOGY:
The clients with symptoms of acute flaccid paralysis / weakness after preliminary investigations are referred for NCS immediately following their admission to our hospital. A RMS EMG EP Mark-II machine (picture) is used. Filters set at 2 Hz to 10 kHz and sweep speed was 10 ms per division for motor study and for sensory study, filters were at 20 Hz to 3 kHz and sweep speed was 2 ms per division. Duration for both motor and sensory study was at 100 µs, F-sensitivity was at 500 µs. F wave parameters for lower limb were same except Sweep 10ms/Div
Nerve conduction studies (NCS) for all accessible nerves of bilateral Upper and Lower limbs was performed on 49 subjects (including men, women & children) who satisfied the inclusion criteria. The procedure was described and informed consent obtained from relative of each participant. NCS was performed in the room where the temperature was maintained at about 30 degree Celsius. NCS was performed by placing the participants in supine position with the respective limb to be tested at side with adequate support in the standard procedure given in standard books. Universal precautions were followed regarding the electrodes hygiene and patient safety inclusive of electrical safety measures. Whenever possible participants were instructed about the sensory perception that they would have, and the study performed with utmost care so as complete the required data needed to be analyzed, if the participant reported any increased sensory perception or resisted the necessary data would be tried with extra support of the relatives, in case of intolerance to electrical stimulus the studies would be terminated. Recording of all accessible motor, sensory, F waves & H- reflex was done and hard and soft copies of recording taken.

The study includes the data analysis of total 49 cases that satisfied the inclusion / exclusion criteria.

The present study defines an electro diagnostic variable as abnormal if it fell outside our laboratory’s limits of normal ± 2SD at CI of 95%. These limits of normal were obtained in 59 healthy volunteers (tests for normality for all the variables done). Conduction velocities and amplitude were abnormal if less than lower limit of normal (mean -2SD), distal latencies and F response minimum latencies if more than upper limit of normal (mean +2SD). F-persistence was considered abnormal if it was < 7 in the upper limbs and < 5 in the lower limb nerves, F-chrono dispersion was abnormal if it exceeded 4ms & 8ms in upper and lower limbs respectively.

RESULTS

There were total of 49 cases of GBS analyzed. The analysis is in two groups
1) The total 49
2) The children in the age category ≤ 3 years
H/O fever / illness was reported in total 26 of 49

Almost all 46 / 49 were referred for NCS in the first 10 days except three (3) who were referred on 14th, 18th & 20th day for NCS.
Table 1
Age of patients (in years) with the mean, median and Standard Deviation with the graph showing the distribution.

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Range (years)</th>
<th>Mean</th>
<th>Median</th>
<th>SD ±</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.00 – 2.00</td>
<td></td>
<td>2.05</td>
<td>2.03</td>
</tr>
<tr>
<td>2</td>
<td>&lt; 2yrs group</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1
Histogram showing the distribution of age (table 1)

Figure 2
Gender wise distribution males 37 (75.5102 %) and females 12 (24.4898 %) of total 49.

Figure 3
GBS type

TOTAL NOS WITH MUSCLE GRADES (MRC) = 0 ------- -- 05 (10.20408 %)
TOTAL NOS WITH MUSCLE GRADES(MRC) ≤ 3 ------- --- 25 (51.02041 %)
TOTAL NOS WITH MUSCLE GRADES (MRC) = 4/5 ------ --- 08 (16.32653 %)

One way : Comparison of Ankle muscle power grades (MP) with CMAP of peroneal and tibial did not show statistically significant correlation [right & left peroneal r = 0.38, 0.055 with p-value 0.820&0.0741 and for right & left tibial r = 0.381, 0.275 with p-value 0.015 & 0.090 resp]

Similar comparison of Wrist MP (muscle power grades) with CMAP (Descriptives) shows moderate statistical significance between wrist muscle grades with CMAP amplitude of median right & left  r = 0.488, 0.435 with p-value 0.005 & 0.006; whilst with right & left ulna r = 0.375, 0.355 with p-value 0.041 & 0.039

Figure 4
Graphical representation of blocks in each nerve when cmap amplitude was < -2 SD or > +2 SD

rmm & lmm right & left median motor; rum & lum right &
Nerve Conduction Studies In Guillain Barre' Syndrome

left ulna motor; rpm & lpm right & left peroneal motor; rtm & ltm right & left tibial motor

**Figure 5**
Representation of % F unrecorded and % age reduction in persistence of each nerve

![Figure 5](image)

rmf & lmf right & left median F; ruf & luf right & left ulna F; rpf & lpf right & left peroneal F; rtf & ltf right & left tibial F

**Figure 6**
% age of subjects with chronodispersion in upper & lower limb nerves of both sides, abnormal F- dispersion (> 4 in median & ulna and > 8 ms in peroneal & tibial nerves

![Figure 6](image)

rmm & lmm right & left median motor; rum & lum right & left ulna motor; rpm & lpm right & left peroneal motor; rtm & ltm right & left tibial motor

**Figure 7**
% age of subjects showing abnormalities of H- reflex, unrecordable and % age in whom it was prolonged.

![Figure 7](image)

There was no association seen between the reduction in CMAP with reduced persistence of the F response.

---

Table 2

<table>
<thead>
<tr>
<th>Nerve</th>
<th>No. out of 49 cases</th>
<th>% Nerve UR</th>
<th>% Nerves with DL &gt; +2 SD</th>
<th>% Nerves with CV &lt; -2 SD</th>
<th>% Nerves with CMAP &lt; -2SD</th>
<th>% Nerves showing BLOCKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>unu</td>
<td>40</td>
<td>3(7.5%)</td>
<td>10(25.0%)</td>
<td>13(32.5%)</td>
<td>19(47.5%)</td>
<td>14(35.0%)</td>
</tr>
<tr>
<td>mmm</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
<tr>
<td>mmu</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
<tr>
<td>umu</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
<tr>
<td>mrm</td>
<td>34</td>
<td>3(8.8%)</td>
<td>8(23.5%)</td>
<td>12(35.3%)</td>
<td>20(58.8%)</td>
<td>16(46.4%)</td>
</tr>
<tr>
<td>rum</td>
<td>32</td>
<td>3(9.3%)</td>
<td>8(25.0%)</td>
<td>12(37.5%)</td>
<td>20(62.5%)</td>
<td>16(48.4%)</td>
</tr>
<tr>
<td>lmm</td>
<td>36</td>
<td>3(8.3%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(55.6%)</td>
<td>16(44.4%)</td>
</tr>
<tr>
<td>lmu</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
<tr>
<td>rum</td>
<td>32</td>
<td>3(9.3%)</td>
<td>8(25.0%)</td>
<td>12(37.5%)</td>
<td>20(62.5%)</td>
<td>16(48.4%)</td>
</tr>
<tr>
<td>lmm</td>
<td>36</td>
<td>3(8.3%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(55.6%)</td>
<td>16(44.4%)</td>
</tr>
<tr>
<td>lmu</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
<tr>
<td>rmm</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
<tr>
<td>rmu</td>
<td>34</td>
<td>3(8.8%)</td>
<td>8(23.5%)</td>
<td>12(35.3%)</td>
<td>20(58.8%)</td>
<td>16(46.4%)</td>
</tr>
<tr>
<td>lmm</td>
<td>36</td>
<td>3(8.3%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(55.6%)</td>
<td>16(44.4%)</td>
</tr>
<tr>
<td>lmu</td>
<td>35</td>
<td>3(8.6%)</td>
<td>8(22.2%)</td>
<td>12(33.3%)</td>
<td>20(57.1%)</td>
<td>16(45.7%)</td>
</tr>
</tbody>
</table>

rmf & lmf right & left median F; ruf & luf right & left ulna F; rpf & lpf right & left peroneal F; rtf & ltf right & left tibial F

There was no association seen between the reduction in CMAP with reduced persistence of the F response.
Nerve Conduction Studies In Guillain Barre’ Syndrome

Table 2a
% age of subjects showing abnormal F- variables within nerves stimulated (same nerves may show combination of abnormalities)

<table>
<thead>
<tr>
<th>Nerve</th>
<th>No. stim out of 49 cases</th>
<th>% F-UR</th>
<th>% with F-LAT prolonged (%)</th>
<th>F-DISP &gt; 4 in UL &amp; &gt; 6 in LL</th>
<th>% nerves with odd F-PERSIST</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm</td>
<td>38</td>
<td>18(51.3)</td>
<td>8(21.6)</td>
<td>7(18.4)</td>
<td>23(60.5)</td>
</tr>
<tr>
<td>lmm</td>
<td>38</td>
<td>18(47.3)</td>
<td>5(13.16)</td>
<td>19(48.72)</td>
<td>25(65.79)</td>
</tr>
<tr>
<td>mm</td>
<td>32</td>
<td>12(37.5)</td>
<td>6(18.75)</td>
<td>4(12.5)</td>
<td>18(56.29)</td>
</tr>
<tr>
<td>lmm</td>
<td>36</td>
<td>20(55.6)</td>
<td>4(11.11)</td>
<td>8(22.22)</td>
<td>16(44.44)</td>
</tr>
<tr>
<td>rmm</td>
<td>42</td>
<td>26(61.9)</td>
<td>12(33.33)</td>
<td>12(33.33)</td>
<td>22(55.88)</td>
</tr>
<tr>
<td>lmm</td>
<td>43</td>
<td>29(67.4)</td>
<td>7(19.15)</td>
<td>9(25.64)</td>
<td>14(32.56)</td>
</tr>
<tr>
<td>rmm</td>
<td>43</td>
<td>29(67.4)</td>
<td>7(19.15)</td>
<td>9(25.64)</td>
<td>14(32.56)</td>
</tr>
<tr>
<td>lmm</td>
<td>43</td>
<td>31(72.1)</td>
<td>11(29.20)</td>
<td>11(29.20)</td>
<td>20(46.51)</td>
</tr>
</tbody>
</table>

Table 3
Number of nerves showing abnormal variables within sensory nerves stimulated (same nerves may show combination of abnormalities)

<table>
<thead>
<tr>
<th>Sensory Nerve</th>
<th>No. stim out of 49 cases</th>
<th>Nerve UR</th>
<th>Lat &gt; 2SD</th>
<th>CV range m/s</th>
<th>CV range m/s</th>
<th>SNAP range mV</th>
<th>SNAP range mV</th>
<th>mm &amp; lmm right &amp; left median motor; rum &amp; lum right &amp; left ulna motor; rpm &amp; lpm right &amp; left peroneal motor; rtm &amp; ltm right &amp; left tibial motor</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm</td>
<td>33</td>
<td>2</td>
<td>1</td>
<td>1.08 - 3.17</td>
<td>8</td>
<td>43.86 - 66.31</td>
<td>16</td>
<td>9.2 - 136</td>
</tr>
<tr>
<td>lmm</td>
<td>32</td>
<td>2</td>
<td>0</td>
<td>0.81 - 2.29</td>
<td>6</td>
<td>33.52 - 65.1</td>
<td>15</td>
<td>6.3 - 114</td>
</tr>
<tr>
<td>mm</td>
<td>28</td>
<td>0</td>
<td>1</td>
<td>1.04 - 2.29</td>
<td>7</td>
<td>39.2 - 69.77</td>
<td>12</td>
<td>3.8 - 98.9</td>
</tr>
<tr>
<td>lmm</td>
<td>36</td>
<td>4</td>
<td>1</td>
<td>1.17 - 3.38</td>
<td>12</td>
<td>38.66 - 67.31</td>
<td>11</td>
<td>17 - 125</td>
</tr>
<tr>
<td>mm</td>
<td>36</td>
<td>2</td>
<td>1</td>
<td>0.96 - 3.75</td>
<td>12</td>
<td>38.25 - 87.6</td>
<td>10</td>
<td>9.6 - 128</td>
</tr>
<tr>
<td>lmm</td>
<td>28</td>
<td>2</td>
<td>1</td>
<td>0.96 - 2.04</td>
<td>12</td>
<td>33.06 - 83.33</td>
<td>14</td>
<td>7.9 - 64.8</td>
</tr>
<tr>
<td>mm</td>
<td>40</td>
<td>3</td>
<td>0</td>
<td>1.08 - 2.96</td>
<td>4</td>
<td>28.72 - 61.64</td>
<td>10</td>
<td>3.3 - 74</td>
</tr>
<tr>
<td>lmm</td>
<td>39</td>
<td>3</td>
<td>1</td>
<td>1.04 - 2.79</td>
<td>5</td>
<td>20.94 - 62.6</td>
<td>7</td>
<td>3.3 - 61.6</td>
</tr>
</tbody>
</table>

Figure 8
Shows the nos. of motor nerves stimulated in age ≤ 3 years and nos. in which CMAP amplitude were markedly low.

Figure 9
Represents no. of motor nerves stimulated in age ≤ 3 years, no. of them un recordable, with blocks, & F-un recordable.

ANALYSIS CASES WITH AGE LESS THAN OR EQUAL TO 3 WERE 10 / 49 (20.41 %)
All were males except one
None had any serious respiratory problems, and 6 of 10 presented with history of fever
All the 10 had the muscle strength on mrc grade less than or equal to 3, except one who had proximal 4 and only distal
Figure 10
Shows the number of sensory nerves stimulated of the 10 children in age ≤ 3 years and the nos. un recordable

![sensory nerves status](image)

rms & lms right & left median sensory; rus & lus right & left ulna sensory; rss & lrs right & left radial sensory; rss & lss right & left sural sensory

One 3year old had all sensory UR, with motor showing blocks (fall in amplitude) 60% in both left median & ulna and 50% & 40% in right & left peroneal respectively. The other one had motor complete un recordable with sensory in upper limbs well preserved BUT sensory & motor UR (lost) in both lower limbs.

**DISCUSSION**

In agreement with several reports the present study also agrees that GBS can occur at any age. In the present study the age range was 1 year - 82 years. The maximum number affected in this series was in the age group 1-25 years 91.84%; 57.14% were in the age group ≤ 10 yrs, and 20.41% were of the age ≤ 3 years, almost similar to reported by Lyu, Tang, Cheng, Hsu, Chen. Several studies report the maximum occurrences in varying age groups; 1-5 years, 5-9 years its highest incidence is at 30-50 years of age. In childhood it usually occurs af¬ter the age of 3 years, with reports of incidence peaks; in one series the age varied from 7 months to 13 years with an uniform distribution.

Although it is a disease that occurs at any age, most series report an incidence of two peaks, the first in late adolescence and young adults, and the second in the elderly. It is rare in children under one year of age, in present study too it was 1 year after. Some studies show an incidence increase linearly with age with age, especially in the older age group.

GBS is equally common in men and women. Men are affected slightly more often than women. The male to female ratio varies in different studies, the present study agrees with reports showing male predominance, as this study showed males 76% compared to females which were 24%.

Associated preceding illness in GBS is reported commonly in varying percentages, almost in two-thirds of cases. In the present study 53.06% reported history of fever, majority patients presented with symmetrically ascending paralysis that rapidly progressed within 24 -72 hours to involve all limbs. About 20% of survivors have residual permanent severe disability especially in ambulation. Similar findings agreed by several studies.

Clinical, laboratory, and electrophysiological findings in children with GBS are similar to those found in adults. The clinical presentation of GBS is often quite characteristic but far from uniform. The speed of progression of the weakness is a good predictive marker of the subsequent severity of the disease. Hence, the risk of developing severe muscle paralysis and possible respiratory failure correlates with the rate of progression of weakness.

In this study the number of cases which showed muscle power grade 0 / 1 on MRC scale 26.53%; ≤ 3 were 51.02%; and those with grade 4 / 5 on MRC scale were 16.33%. The present study tried to find the association between the muscle power grades on MRC scale and the reduction in CMAP amplitude, there was moderate to weak correlation between the median (p-value 0.005 & 0.006) and ulna (p-value 0.041 & 0.039) CMAP amplitude to the MRC grades of wrist and hand muscles, but the same was not significant with CMAP amplitudes of peroneal (p-value 0.820& 0.741) &tibial (p-value 0.015 & 0.090). In one study no correlation was found between the nerve conduction velocities or distal motor latencies and clinical severity of the neuropathy, although distal CMAP amplitudes less than 10-20% of normal are associated with a poorer prognosis. Pattern of limb weakness with regards to proximal or distal or equal are reported in varied proportions by different studies.
Predominant distal weakness is common. In 1/3 of cases, the degree of weakness in the arms and legs is roughly equal. The present study too weakness was bilateral symmetrical, distal more than proximal more commonly distal almost equal to proximal.

Electrophysiological examinations in various studies have ranged from 4 to 32 days of illness. From the first days of the disease, the electro diagnostic results are helpful in the diagnosis of the disease which is in accordance with the findings of the study by Delanoe and others. In the present study (46 of 49) 93.88% had electrophysiological testing in the first 10 days of the onset of symptoms. The median was 3 (min.1 & max. 20 days). Electrophysiology usually should include data from at least 3 sensory nerves, 3 motor nerves, with multisite stimulation, and F wave and H-reflex study recommended to assess more nerves, all four limbs. The present study assessed all the accessible nerves in each subject. The motor & sensory variables viz. latency, evoked potentials amplitude (% age of block in motor nerves), conduction velocity along with detail F-wave study (inclusive of presence, latency, persistence & chronodispersion) and H-reflex were assessed supported by several authors. However, unlike the clinical diagnostic criteria, which have been agreed on, there is no consensus on the neurophysiological criteria for classification.

GBS can be divided into three subtypes that can be differentiated through electro diagnostic techniques (Kuwabara, 2004). AIDP is the most common form of GBS in developed countries accounting for 90% of cases (Moran et al., 2005). As shown in most of the population based studies our study has also shown highest number of AIDP patients. This series also well in agreement with the literature showed that the AIDP [93.88%] was the commonest form. Typical AMAN occurs in only 5% of GBS cases in western countries. A study from Argentina about 30%. It also stated that in their study usually near the peak of the illness three patterns emerged: 1) (52.5%) patients the demyelinating type of GBS, 2) (20%) patients had the demyelinating type of GBS with secondary axonal loss, and 3) (27.5%) patients had axonal type GBS. In European and US studies the AIDP is the most prevalent form with an incidence between 85% to 90%, in spite of Asian studies which report it about 53%. Where as in Northern China AMAN was the highest incidence. Rees JH, et al (1995), England study shows 85% AIDP and MFS 5%. Ho TW, et al in Western China study show AIDP – 19%, AMAN 65%, AMASAN 10-15%. Udaya Senevaratne, Sri Lanka Study (2000) shows, 76% AIDP, 24% AMAN and 10% comprises other subtype of GBS. Rong KL, et al (1997) Taiwan study showed AIDP (49%), AMAN (4%), MFS (18%) and others 29%. Emilia Romagna, Italy study (1992-93) showed AIDP (36%), AMAN (14%) and MFS (13%). In another study7 electrophysiological diagnosis 74.2 per cent classified as acute inflammatory demyelinating poly neuropathy (AIDP), of which three showed evidence of secondary axonal degeneration (denervation changes on electromyography). (12.9 per cent) had changes of a primary axonal neuropathy, of which two had pure motor involvement electro physiologically, thus fulfilling the criteria of AMAN. These findings suggest that the incidence of the demyelinating type of GBS varies considerably among countries. It may be due to different genetic background and environmental exposures as stated.2

The present study also reports the AIDP form to be the most predominant, with AMSAN (4.08%), & AMAN (2.04%). Of the total 49; 3 patients had presence of early motor nerve inexcitability which represents a reliable marker of axonal poly radiculonueopathy. One was classical AMAN, and of them 2 had even the sensory unrecordable and/or markedly reduced SNAP amplitude similar to as AMSAN. The finding of inexcitability of nerves was an indicator of poor outcome, and previous reports referred to in this study. Inexcitability can result either from axonal degeneration or dysfunction or demyelination causing complete conduction block. Other factors predicting poor outcome include, a protracted plateau time, change to chronic progressive or relapsing neuropathy or concurrent myelitis. One study, suggested that the finding of low amplitudes on nerve conductions may not necessary imply a bad outcome.

Serial electrophysiological examinations, within 2–7 days, showed much reduced or absent evoked responses on distal supra maximal stimulation of motor and sensory nerves, progressing rapidly to total loss of electrical excitability. This pattern was most consistent with findings observed in...
nerve fibers undergoing acute axonal degeneration. Accordingly, patients showed severe, generalized muscle atrophy with delayed and very poor recovery. The disorder was notable for the fulminant onset of severe paralysis and sensory deficits. Griffin and colleagues introduced the descriptive term now generally used: acute motor-sensory axonal neuropathy (AMSAN).23

Albers and colleagues proposed that the diagnosis of acute demyelinating poly neuropathy be based on one classic criterion for primary demyelination in only two nerves21, 33, 45 during the first 2 weeks of illness. Farhad Mahvelati Shamsabadi2, suggest that during the first week of an acute illness in a child without any other previous neurological deficit, if three nerves show signs of demyelination with at least four abnormal variables are highly suggestive of acute demyelinating poly neuropathy.2, 33. These abnormal variables include: reduction in motor conduction velocity13, conduction block, prolonged distal latency, absent F wave or prolonged minimum F wave latencies, reduction in sensory nerve conduction velocity, diminution of CMAP and/or SNAP amplitudes, all parameters usually show symmetrical and multifocal involvement.2,5,33 It is worth noting that the absence of electrophysiological findings during this period does not rule out the hypothesis of GBS.5,22,43

Electrodiagnostically a nerve can be defined as abnormal if a minimal number of variables exceed the normal limits; however, no consensus exists on the number of required abnormal variables per nerve and the required number of abnormal nerves with regard to the electrodiagnostic definition of poly neuropathy. Due to the nature of poly neuropathy a minimal number of two abnormal nerves would seem to be required.43 A consensus for early diagnosis of GBS has not been established.5, 33 Controversy surrounds early electrophysiological findings in GBS. Some authors suggest that conduction block is the earliest demonstrable change, whereas others report that prolonged distal latencies and prolongation or absence of F-wave and H-wave latencies are the earliest findings, that reflect the early predilection for involvement of the proximal spinal roots and distal motor never terminals in AIDP. 5, 7,11

The author Amato AA1 always perform F-wave studies in both the upper and lower limbs in patients suspected of having AIDP because of the early predilection for the proximal nerve segments and spinal roots, is supported by several others.5, 13, 20, 37, 42, 44 Based on studies of the F-response, conduction slowing across proximal segments of motor nerves in excess of more distal segments has been shown, as well as proximal conduction block early in the disease, findings confirmed by Brown and Feasby and Berger et al.42 Prolonged or absent F-waves and H-reflexes are found in 80-90% of patients during the course of AIDP.11 It is conduction block of nerve impulses that result in actual weakness and sensory loss. Abnormalities in late responses, either absent responses or prolonged responses, are common, occurring in up to 46% of individuals studied in the first month, and reflecting the predilection of the disease for proximal nerve segments.32

The present study too, as reported in the literature saw motor fibers clinically involved more than sensory fibers. In one study, 90% of GBS patients had motor nerve conduction abnormalities,7,34 this is common in the first two weeks of illness and this figure rises to 96% by the third week of illness.32

The present study also in agreement with literature saw abnormalities in number of variables of multiple nerves, and in agreement to several authors it was observed that in a small number of patients in whom still the CMAP’s amplitudes were preserved the F-waves were lost, as was the H-reflex. In few we saw that with clinical signs only ‘F’ was lost with motor and sensory showing changes but still remaining within mean ±2 SD. This of course did accompany predominantly with abnormal several other variables assessed in the rest. The occurrence of F-disturbance was reduced persistence, F-un recordable / absence, increased chrono dispersion, delayed F-min. latency almost in descending order. (fig 5, 6). The F-waves were invariably the hallmark consideration when reporting interpretation.

In early GBS, the F-waves may be absent or may have a decreased persistence (frequency of occurrence) and a prolonged minimum latency; these alterations are caused probably by an axonal dysfunction in proximal nerve segments or by axonal degeneration at the level of nerve roots (Cornblath et al., 1988; Kuwabara et al., 2000).44

The present study too, we saw either F un recordable, reduced in persistence and or showing increased CD, sometimes prolonged significantly in isolation or in combination with regards to other F-variables. Fig.5, shows the high %age of subjects with F-UR both sides (median 28%, ulna 34-45% , peroneal 45-54%, tibial 26-37%) and F-reduced persistence was significantly higher (median 65%,
thus, low amplitude CMAPs does not necessarily imply earliest electrophysiological abnormalities noted in AIDP, days of onset.11 Low amplitude CMAPs are one of the amplitudes or absent CMAPs 7, 11 and SNAPs within 7-10 days of onset.11 Nerve conduction studies reveal markedly diminished sole abnormality in about 30-50% of cases studied 22. Reflected latencies. Prolonged or absent F waves may be initial abnormalities of F waves and H reflex, typically there is multifocal demyelination affecting proximal and distal nerve segments. Earliest findings may be abnormalities of F waves or the increase of their latency later in the progression of the disease; this suggests that the presence of repeaters may be a transient and a very early electrophysiological sign of GBS that should be considered in the differential diagnosis of this disease44.

A particularly interesting parameter of the F-waves is the presence of repeater F-waves (defined as recurrent identical waveforms).44 In the present study too, repeater waves were clearly identifiable and seen in 3-4 subjects nerves more commonly were peroneal and tibial. The isolated presence of repeater F-waves in some nerves of the legs during the initial phases of the GBS precede the abolition of the F-waves or the increase of their latency later in the progression of the disease; this suggests that the presence of repeaters is a reliable and constant abnormality in patients with 41 Miller Fisher syndrome (MFS), when examined. Given the inconsistent findings with F waves, it is more likely that the absence of H-reflex is related more to the sensory portion of the reflex, rather than the motor portion. As mentioned earlier, it was postulated that group Ia muscle spindle afferents are preferentially affected in MFS and may be responsible for the clinical signs of ataxia and areflexia, and the absence of H-reflex. 41 Typically, there is multifocal demyelination affecting proximal and distal nerve segments. Earliest findings may be abnormalities of F waves and H reflex latencies. Prolonged or absent F waves may be initial sole abnormality in about 30-50% of cases studied 22.

Nerve conduction studies reveal markedly diminished amplitudes or absent CMAPs 7, 11 and SNAPs within 7-10 days of onset.11 Low amplitude CMAPs are one of the earliest electrophysiological abnormalities noted in AIDP, thus, low amplitude CMAPs does not necessarily imply axonal degeneration. Distal conduction block with or without demyelination also leads to low amplitude distal CMAPs.11 In the present study too markedly diminished CMAP amplitudes were seen in significantly high %age of subjects both sides (median 54-57%, ulna 72-75%, peroneal 38-42%, tibial 65-62%), as seen in Table 2. This was lesser than reported by a study with 96% & 83% showing CMAPs reduced in peroneal and median respectively. As reported in the literature later, weakness and sensory loss may occur due to secondary axonal degeneration. Conduction block is noted in 74% of patients within the first two weeks. Conduction block is often appreciated at common sites of entrapment or compression such as the carpal tunnel (median nerve), cubital tunnel (ulnar nerve), and fibular head (peroneal nerve)11.

Accordingly along with F & H abnormalities, Conduction block in about 1/3 of cases is seen early in GBS; conduction slowing and temporal dispersion reflect demyelination. 22.

Conduction block and temporal dispersion are considered hallmarks of multifocal demyelination. 32, 34 These occur in up to 75% of individuals,32 and may be present even when there is little abnormality of maximum conduction velocity. 22, 37 Feasby et al were able to relate conduction block to pathological demyelination in nerve biopsies. Also, experimental conduction block-whether induced mechanically or immunologically is associated with demyelination. 35 Conduction block (50% drop in the proximal amplitude compared with the distal CMAP amplitude) was present in posterior tibial nerves in two patients and in the peroneal nerve in one patient.45.

The present study too, early in the series, we see the conduction block is present in 20.93% to 44.44% of patients amongst the various nerves assessed and 57.14% patients in total showed blocks and almost all had blocks in more than two nerves in upper and / or lower limbs. Fig. 4b shows the %age of patients showing blocks when the CMAP amplitude was ± 2SD. In the present study we see that the % age of patients showing blocks, when CMAP amplitude was abnormal (< mean -2SD) is greater than the those in whom the CMAP amplitude had not regressed (within mean ±2SD). The present study also saw that those cases with conduction blocks presented with inability to ambulate, thus agreeing to literature that conduction block of nerve impulses results in actual weakness.11 Motor CB has been documented in only 2 to 15% of patients with GBS within 3 weeks from disease onset.39 Peroneal nerve conduction block and age above 40 years were independent predictors of
disability at 6 months.12

This contrasts with the lower percentage of abnormalities in conduction velocity apart from sites of “nerve entrapment” (20%), in distal latency (33%), and in temporal dispersion (20%). Evidence of conduction slowing is more common at usual sites of “entrapment” (60%): Conversely, sensory conduction studies are abnormal in only 25% of individuals in the first week, rising to 73% of individuals by the third week. This is usually manifest as a reduction in evoked amplitude.32 As seen in the present study too, the %age of subjects showing significant decrease in conduction velocity (< -2SD) early in the condition are less (28-34% in upper limbs and 20-30% in lower limbs. Prolonged distal latency was in 10-20% in upper limbs and 25-35% in lower limbs)

This was again different, reported by Farhad Mahvelati Shamsabadi 2 with 8.5 %CV , 61% CV of median, and peroneal respectively & 39 %, 74% showing DL prolonged of median, and peroneal respectively

Slow motor nerve conduction velocity is an early and characteristic finding of patients with AIDP. Conduction blocks (drop in the muscle action potential amplitude when a site of proximal simulation is compared to distal stimulation of the same nerve) appear later, but are more specific of this disorder.13 In early GBS, prolonged distal latency of median, 30-40% ulna, 40-60% peroneal and temporal dispersion are more commonly demonstrated than are slow motor conduction velocities and conduction block.7

The Table 2 & 2a gives the details of %age of subjects showing abnormal motor & F- variables within nerves stimulated (same nerves may show combination of abnormalities) In the present study all parameters showed symmetrical and multi focal involvement.

Motor fibers are clinically involved more frequently than sensory fibers,” and sensory evoked responses may be entirely normal in patients having prominent motor abnormalities. Nevertheless, median or ulna SNAPs have been reported to be absent in 58% of patients," with abnormalities of sensory conduction (abnormal SNAP amplitude or evidence of demyelination) reported in 76% of patients.5,34 The largest electro diagnostic discrepancy between motor and sensory abnormalities occurs during the first 2 weeks after disease onset, as judged by either evoked response amplitudes or percentage of patients having abnormal motor or sensory studies. Interestingly, SNAPs may be abnormal or absent45in certain nerves (e.g., median), yet normal in others (e.g., sural) (Kornhuber et al., 1999; Gordon and Wilbourn, 2001). The relative sparing of sural responses in the presence of abnormal median sensory responses is atypical of any diffuse poly neuropathy. This finding, in association with an appropriate clinical syndrome, suggests the diagnosis of AIDP.13, 34, 44 This characteristic pattern is abnormal reflecting random, multifocal demyelination22 In sensory nerve conduction, about 40-60% of patients have some abnormality of both conduction velocity and amplitude (more often) of several sensory nerve conduction potentials; these findings may be absent during the first weeks of the disease. Up to 4-6 weeks may be necessary until changes in these potentials can be easily detected.5The existence of a purely sensory form of Guillain-Barre syndrome is still subject to controversy, although the criteria for its diagnosis have been established.46

Table 3 shows the sensory nerve data, 34.69% number of subjects who showed abnormality in the various variable assessed of sensory nerves, the maximum number show SNAP affected early in GBS more than the conduction velocity which immediately follows in almost all nerves. >75% of sural were unaffected in the present series too, the pattern of median abnormal with sural preserved was on an average common, insignificant number showed delay in the latency. Several possibilities exist that can explain the discrepancy between motor and sensory studies, as well as the discrepancy between sural and median sensory conduction studies.34

The data of 10 children age group ≤ 3 years was analyzed separately. As seen (fig.8) the reduced CMAP amplitudes were predominant in most of them of both sides (20-60% median, 30-40% ulna, 40-60% peroneal and 30-60% in tibial). Slowing of conduction velocity followed, distal latency was affected in least. Fig. 9 shows the abnormal variables in the age group ≤ 3 years, as seen the abnormal F predominates, F-un recordable ranges in 30 -60%ages of subjects maximum seen in the peroneal (60% bilaterally). The duration in this age group of electrophysiological testing was within 8 days except in one child. Fig.10 shows the un recordable sensory nerves two showed sural lost, in one of these there was complete in excitability of both lower limbs and in one all sensory were in excitable whereas the motor showed blocks in three nerves. Sensory nerve details also indicated the maximal values of SNAP and the CV well compatible with adults in some.

Electro diagnostically a nerve can be defined as abnormal if a minimal number of variables exceed the normal limits; however, no consensus exists on the number of required
abnormal variables per nerve and the required number of abnormal nerves with regard to the electro diagnostic definition of poly neuropathy. Due to the nature of poly neuropathy a minimal number of two abnormal nerves would seem to be required.43 The most commonly encountered neuro physiological abnormalities were absent or prolonged F wave: 90%, prolonged distal latency: 80%, absent H reflex: 76%, delayed nerve conduction velocity: 73%, partial or complete conduction block: 63%, abnormal blink reflex: 60%, reduced compound muscle action potentials (CMAP): 38% and abnormal sensory nerve action potentials (SNAP): 28%. Demyelination was the predominant type in the neuro physiological work-up followed by axonal type.20

The present study agrees that a variety of electro diagnostic findings have been reported for patients with Guillain-Barre syndrome, the reported abnormalities are thought to reflect both the multifocal nature of the disorder and the combination of demyelination with varying amounts of secondary axonal degeneration. In addition, the various electro diagnostic findings unquestionably reflect the time at which studies are performed relative to disease onset, recognizing that temporal changes occur in response to cumulative demyelination and axonal degeneration.34. Thus even early therefore one sees a combination of variables which may be abnormal stressing the importance of testing the maximal number of nerves accessible.

CONCLUSION
Well supported by the literature the present study also as reported by Alan R. Berger, M.D.22 saw;

- Electrophysiological studies diagnostic in >95% cases early in GBS
- Nature and severity of physiologic findings dependent on timing of study, number of nerves studied, and whether proximal nerve segments investigated.
- Typically, there is multifocal demyelination affecting proximal and distal nerve segments.
- Abnormal or absent F waves may be initial sole abnormality and in the present study F- un recordable was seen in about 40-60% of cases studied.
- In 41.86% H reflex were un recordable and 23.26% showed prolonged latencies
- Also markedly reduced CMAP amplitudes were seen in high %age (40-70%) of participants in almost all the nerves;
- Conduction block was seen in about 57.14% of cases; with conduction slowing and temporal dispersion reflect demyelination.
- Evidence of SNAP or CV abnormality seen in about 34.69% of cases
- 03 patients that showed in excitable nerves early.
- Weak to moderate correlation between the degrees of weakness was seen in the hand with CMAPs of median & ulna.

ACKNOWLEDGEMENTS
Authors acknowledge the immense help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed. The authors are also grateful to the trust “Charutar Arogya Mandal” for all the support provided.

References
02. Farhad Mahvelati Shamsabadi, MD; Parvaneh Karimzadeh1, MD; SeyedHasan Tonekaboni1, MD; Javad Ghorobi, MDAcute Inflammatory Demyelinating Polyneuropathy in Children: Clinical and Electrophysiologic Findings Iran J Pediatr Mar 2009; Vol 19 (No 1), Pp: 52-58
09. Vanessa van der Linden, José Albino da Paz, Erasmo Barbante Casella, Maria Joaquina Marques-Dias, Guillain-Barré syndrome in children Arq Neuropsiquiatr 2010;68(1):12-17
Nerve Conduction Studies In Guillain Barre' Syndrome


n0550.pdf


27. JAMES W. ALBERS, MD, PhD, PETER D. DONOFRIO, MD, and TIMOTHY K. MCGONAGLE, MD SEQUENTIAL ELECTRODIAGNOSTIC ABNORMALITIES IN ACUTE INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY MUSCLE & NERVE 1985: 8:528-539


33. Farhad Mahvelati Shamsabadi, MD; Parvaneh Karimzadeh, MD; SeyedHasan Tonekaboni1, MD; Javad Ghoriob2, MD Acute Inflammatory Demyelinating Polyneuropathy in Children; Clinical and Electrophysiologic Findings Iran J Pediatr 2009; Vol 19 (No 1), Pp:52-58


35. Mahmoud Reza Ashrafifar, MD, Setareh Sagheb, Mahmoud Mohammadital, Anoushiravan Vakili, MD, Abolfazl Nasirian1, MD, Gholam Reza Zaman1, MD. Clinical Short Term Outcome of GuillainBarré Syndrome in Children Iran J Pediatr Mar 2008; Vol 18 (1 No 1), Pp:11-19


37. 2Arthur K. Ashbury, MD, and David R. Cornblath, MD Assessment of Current Diagnostic Criteria for Guillain-Barré Syndrome Ann Neurol 1990;27(suppl):S1-524


41. M. Capasso, MD; C.M. Caporale, MD; F. Pomilio, DVM; P. Gandolfi, DSc; A. Lugaresi, MD, PhD; and A. Uncini, MD Acute motor conduction block neuropathy Another Guillain–Barré syndrome variant NEUROLOGY 2003;61:617–622


43. William F Brown, Robert Snow Patterns and severity of conduction abnormalities in Guillain-Barré syndrome
46. Shin J. Oh, MD; Chris LaGanke, MD; and Gwen C. Claussen, MD Sensory Guillain-Barré syndrome Neurology 2001;56:82 DOI 10.1212/WNL.56.1.82
Author Information

Lata D Parmar, M.Sc. (PhD)
Principal, College of Physiotherapy, Sumandeeep Vidyapeeth, Piparia, Waghodia, Vadodara, Gujarat, India
latashroff@yahoo.com

Vikas Doshi, Ph.D. Assistant Professor
Statistics, Department of Preventive & Social Medicine, Medical College Vadodara, India

S. K. Singh, Professor MD,
Physiology Department, Pramukh Swami Medical College Karamsad, HOD, Anand, Gujarat, India