CHAPTER IV

RESULTS
The results presented below have been divided into three sections: they are with respect to the three components of AEPs, viz., P₁, N₁ and P₂, each component has been dealt with separately. However, the integrated data of all the three components has been illustrated towards the end.

Three measures were considered for comparative analysis. These three measures were amplitude, latency and the absence of the potential. (Details are given in Chapter III).

The comparisons were done with regard to the T₄-Cz and T₃-Cz leads. These comparisons were carried out only on those patients in whom these components were identifiable. Those patients who did not produce these components have been dealt with separately.

P₁ COMPONENT:

NORMAL CONTROL GROUP:

It was possible to neurologically normal subjects with the exception of 5 on T₄-Cz and 6 on T₃-Cz leads. The mean latency obtained on T₄-Cz was 63.3 (SD = 10.6) and on T₃-Cz it was measured to be 64.4 (SD= 12.8). No difference was observed between the mean values
of both the leads. The mean amplitude of this group was found to be 5.7 (SD = 4.4) on T4-Cz and 5.82 (SD 4.8) on T3-Cz lead, which show high variability within the normal group. Evidently, there was no significant difference between the two electrode sites ($P > .05$) (See fig. A-1 and A-nx).

**COMPARISON BETWEEN MCG AND SOLG:**

Patient group with SOL, in general, (regardless of the consideration of the site of lesion) produced the mean latency of 91.23 (SD : 20.8) on T4-Cz and, 87.6 (SD :17.45 on T3-Cz leads. Obviously, both the groups differed significantly on both on T4-Cz lead ($X^2 = 20.5, P < .001$) and on T3-Cz ($X^2 = 17.9, P < .001$) leads. This mean difference has been illustrated in fig A-nx. No significant difference was observed between the two electrode sites with respect to the latencies ($P > .05$). (See Fig.A-I).

It was noteworthy that 28% of patients on T4-Cz and 34% patients on T3-Cz lead failed to produce this component as compared to negligible absence in the normal control group. No statistically significant difference emerged between the two groups regarding the amplitudes on both the leads where SOLG produced the mean amplitude of 4.8 (SD = 1.72) and 4.03 (SD 1.59)
With respect to T4-Cz and T3-Cz, the statistical values
\( x^2 = 0.87, \ p > 0.05 \) on T4-Cz and \( x^2 = 0.9, \ p > 0.05 \)
on T3-Cz leads did not demonstrate a statistically significant,
difference between the two groups. Also, within the SOLG, no
significant difference was observed between the amplitudes
obtained at both the leads \( (x > 0.05) \) (See Fig IX).

**COMPARISON BETWEEN ASOLG AND PSOLG**

ASOLG produced a mean latency of 87.3 (SD = 20.3) on
T4-Cz and 86.3 (SD = 15.8) on T3-Cz leads as compared to
PSOLG, which produced a mean latency of 104.8 (SD = 17.11)
on T4-Cz and 99.5 (SD = 1.8) on T3-Cz leads, and this differ-
ence reached the level of statistical significance \( x^2 = 6.69 \)
\( p < .01 \) on T3-Cz lead, though it was not significant
at T4-Cz lead \( (x^2 = 3.14 , \ p > .05) \)

The posterior lesions involving temporo-parietal
lobes seem to prolong the mean latency as compared to the
lesion in the anterior region, which might suggest the impor-
tant role of posterior structures in the production of this
component. It may also be pointed out that no significant
difference emerged between the two groups (See Fig A-X). It
may also be mentioned that across both the leads of ASOLG, no
significant difference was observed. Similar findings were
observed with regard to the PSOLG. Thus within each of these
groups no topographical variation was observed. The amplitudes
also did not show a significant difference (See Fig A-X).
COMPARISON BETWEEN ASOLG AND NCG:

A comparison between ASOLG and NCG groups showed a statistically significant difference on both T3-Cz ($X^2=17.9, P<.001$) ($X^2=25.4, P<.001$) leads which showed that ASOLG obtained longer mean latency as compared to the NCG. With regard to amplitudes, no significant difference was found between the two groups on T4-Cz ($t^2=1.9, P>.05$) and T3-Cz ($X^2=1.6, P>.05$) leads (See fig A-X).

COMPARISON BETWEEN PSOLG AND NCG:

A comparison between PSOLG and NCG showed a statistically significant difference between the two groups. On both the T4-Cz and T3-Cz leads, a statistically significant difference was observed between the two groups ($X^2=19.8, P<.001$) on T4-Cz ($X^2=22.8, P<.001$) on T4-Cz lead. This points out the role of PSOLG in the mediation of this component (See Fig A-II).

With regard to amplitudes, the difference between the two groups did not reach the level of statistical significance ($X^2=1.4, P>.05$) at T4-Cz, and ($X^2=1.8, P>.05$) at T3-Cz leads (See fig A-X).
COMPARISON BETWEEN RSOLG AND LSOLG:

The LSOLG and RSOLG produced the mean latency of 95.2 (SD = 9.43) and 84.04 (SD = 20.6) respectively on T4-Cz lead and on the T3-Cz lead, a similar trend was observed, where mean latency of these groups was 89.5 (SD = 19.4) and 95.8 (SD = 20.4). It is to be noted that no significant difference was observed between the two groups at T4-Cz ($t^2 = 1.2$, $p > .05$) and T3-Cz ($t = .08$, $p > .05$) leads.

(See fig. A-VI).

The trends on both the leads indicate the larger effect of left sided lesion on the generation of this component. It suggests that the left hemisphere might be playing an important role in the genesis of this potential. With regard to amplitudes, the RSOLG obtained the mean amplitude of 4.96 (SD = 2.00) and 4.00 (SD = 1.62) on T4-Cz and T3-Cz leads respectively. Whereas, the LSOLG obtained the mean amplitude of 5.32 (SD = 1.7) and 3.68 (SD = 1.15) with respect to T4-Cz and T3-Cz leads respectively. Evidently, no significant difference was observed between the two groups at T4-Cz ($t = 0.55$, $p > .05$) and T3-Cz ($t = 0.46$, $p > .05$).

COMPARISON BETWEEN RFSOLG AND LFSOLG:

The left sided lesion of frontal lobe seems to be playing
The absence of the component to heighten in case of component. On the other hand, in LPSOLC, 53.81 and 38.44% of participants were observed that 17.89 and 23.56% of participants were not observed to have a group on the basis of mean amplitude.

The interaction between the left and right trapezoid lobes did not reach the level of statistical significance, with regard to amplitude, the difference between the right trapezoid lobe and the generation of the AEP component. This further suggests an activity between 12.84 (9.7) and 13-cz 88.9 (SD = 19.8) and 13-cz 92.34 (SD = 18.99) of LPSOLC, 6.96% of participants. As compared to the mean latency of LPSOLC at 12-cz 69.21, 9.95 (SD = 9.19) and 13-cz 92.34 (SD = 18.99) of LPSOLC, is evidenced by larger mean latency and produced at 4-cz.
The mean latencies of each group have been given in the table:

<table>
<thead>
<tr>
<th></th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AFSOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>T4-Cz</strong>: Mean</td>
<td>63.3</td>
<td>77.63</td>
<td>94.34</td>
<td>91.23</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>10.66</td>
<td>19.45</td>
<td>19.94</td>
</tr>
<tr>
<td><strong>T3-Cz</strong>: Mean</td>
<td>64.4</td>
<td>87.38</td>
<td>97.9</td>
<td>94.3</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>12.9</td>
<td>24.15</td>
<td>18.8</td>
</tr>
</tbody>
</table>

The above table shows strikingly higher mean latencies in AFSOLG and PFSOLG. The results indicate the important role of anterior and posterior frontal regions in the production of this component. Similarity of findings with respect of T3-Cz further strengthened this result. However, it is to be noted that a comparison between DFSOLG and AFSOLG ($z=0$, $P>.05$) at T3-Cz lead did not show a significant difference, but this difference was significant at T4-Cz lead ($t=2.14$, $P<.05$). On the other hand, the comparison between PFSOLG and AFSOLG at both T4-Cz ($z=0.33$, $P>.05$) and T3-Cz ($z=.34$, $P>.05$) leads did not show a statistically significant difference.
A comparison between DFSOLG and PFSOLG reached the level of statistical significance at T4-Cz (Z = 1.96, \( p < .05 \)) lead, but not at T3-Cz (Z = 0.37, \( p > .05 \)) lead. (See fig A-V)

With regard to amplitudes, no significant difference was observed at any level of analysis at either of the electrode sites. The mean values have been given in the table below:

<table>
<thead>
<tr>
<th></th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AF SOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td>T4-Cz</td>
<td>Mean: 5.5</td>
<td>5.49</td>
<td>6.54</td>
<td>5.37</td>
</tr>
<tr>
<td></td>
<td>SD : 1.5</td>
<td>1.51</td>
<td>1.03</td>
<td>0.68</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>T3-Cz</th>
<th>Mean: 4.32</th>
<th>4.32</th>
<th>4.85</th>
<th>3.59</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SD : 1.9</td>
<td>1.9</td>
<td>0.88</td>
<td>1.54</td>
<td></td>
</tr>
</tbody>
</table>

With regard to amplitudes, a comparison between DFSOLG and AF SOLG (Z = 0.91, \( p > .05 \)) and PFSOLG and AF SOLG (Z = 1.05, \( p > .05 \)) and AF SOLG and DFSOLG at T4-Cz lead did not yield a statistically significant
difference. Likewise, no significant difference between DFSOLG and AFSOLG ($z = 0.68, P > 0.05$), DFSOLG and AFSOLG ($z = 0.0, P > 0.05$) DFSOLG and AFSOLG ($z = 0.86, P > 0.05$) was observed at T3-Cz lead. Thus, the results mainly reveal the trends rather than a clear cut difference. (See fig. A-XI).

**COMPARISON BETWEEN TEMPORAL AND PARIETAL SOLGS:**

A comparison between the temporal and parietal SOLGS did not show difference between the mean latency of temporal SOLG 109.8 (SD = 13.6) and parietal SOLG 95.7 (SD = 9.21) on T4-Cz lead ($z = 1.15, P > 0.05$). A similar trend was seen at T3-Cz ($z = 1.15, P > 0.05$) lead where temporal SOLG obtained the mean latency of 118.7 (SD = 10.3) and parietal SOLG 97.9 (SD = 15.9) (See fig. A-VII).

In another comparison on T3-Cz lead the left temporal SOLG obtained mean latency of 115.7 (SD = 12.6) and the right temporal SOLG obtained 124.6 ($N = 2$). Similarly, on T4-Cz, lead the latency of both the groups were 111.25 ($N = 2$) of left temporal and 86.03 (SD = 13.59) of right temporal SOLGS.

In case of parietal SOLG, a similar trend was seen
the left parietal SOLG obtained the mean latency of 111.25 (N=2) and 86.03 (N=3) on T4-Cz and T3-Cz electrodes sites respectively. In comparison to this, the right parietal SOLG produced the mean latency of 100.13 (SD = 4.5) and 91.2 (SD = 11.2) on T4-Cz and T3-Cz leads respectively. The comparison between the groups with unilateral lesion of right or left hemispheres pointed out the role of the left temporal parietal regions, in the regulation of this component.

With respect to the amplitudes, the temporal SOLG and parietal SOLG did not show difference in their mean amplitudes. The parietal SOLG obtained the mean amplitude of 4.75 (SD = 2.5) and 4.6 (SD = 1.6) on T4-Cz and T3-Cz leads respectively, and temporal SOLG produced the mean amplitude of 3.8 (SD = 1.84) and 2.8 (SD = 1.4) on T4-Cz and T3-Cz leads respectively. No significant difference emerged from this comparison. In another level of comparison between the left and right temporal SOLGs, the LTSOLG obtained the mean amplitude of 3.4 (SD = 0.39) and 3.7 (SD = 1.46) on T4-Cz and T3-Cz leads whereas the right temporal SOLG produced the mean amplitude of 4.2 (SD = 2.3) and 1.85 (SD = 0.42) at T4-Cz and T3-Cz leads respectively.
On the other hand, the left parietal SOLG obtained the mean amplitude of 5.4 (SD = 2.12), the right parietal SOLG obtained the mean amplitude of 4.11 (SD = 2.44) at T4-Cz lead, whereas at T3-Cz lead the left and right parietal SOLG obtained the mean amplitude of 4.00 (SD = 2.55) and 4.9 (SD = 1.3) respectively. Evidently, no significant difference was observed at any level. It was noted that 50% patients of temporal SOLG on each lead failed to produce this component while in the parietal SOLG, 57.1% and 42.8% of the patients on T4-Cz and T3-Cz leads respectively failed to produce this component.

N1 component of AEPs:

N1 component of AEPs was identified in all the neurologically normal subjects. The mean latency with respect of T4-Cz lead was measured to be 103.06 (SD = 17.6) and at T3-Cz lead, it was found to be 107.48 (SD = 15.06)

The mean amplitude however was 6.79 (SD = 3.81) at T4-Cz lead, and 5.6 (SD = 2.7) at T3-Cz lead. These results do not demonstrate a striking difference between the values obtained at both the electrode sites. It may be noted that both with respect to amplitudes ($P > .05$) and latencies ($P > .05$), no marked variation in the topographical distribution was observed. (See fig A-I).
A comparison between the NCG and SOLG was carried out by utilising median test both with respect to the latencies and amplitudes across both the electrode sites. SOLG obtained the mean latency of 144.21 (SD = 30.35) at T4-Cz and 142.18 (SD = 25.4) at T3-Cz leads. No topographical differences were observed with respect to both the leads (P > .05). However, a statistically significant difference between NCG and SOLG was observed with respect to T4-Cz ($X^2 = 20.8 , P < .001$) as well as T3-Cz leads ($X^2 = 29.5, P < .001$). This comparison has been illustrated in Fig A-I. The prolonged latencies of N1 component show the dysfunction of the selection attention process, because N1 represents the processing negativity.

No significant difference was observed between the two groups at either of the leads with respect to the mean amplitude of this component. As compared to NCG, SOLG obtained the mean amplitude of 5.55 (SD = 2.9) and 6.04 (SD = 2.9) with regard to T4-Cz and T3-Cz leads respectively. These results point out that the pathophysiological condition adversely affect the latencies, but the amplitudes remain unaffected. The above comparative results have also been shown in Fig A-IX.

In 36% of the patients (SOLGs), N1 component was
observed to be absent. It may be restated that this component could be identified in all the neurologically normal subjects. The absence of this component might signify the cortical inhibition at various levels due to pathology, which might have affected the generation of this potential.

**COMPARISON BETWEEN ASOLG AND PSOLG:**

In order to examine the effect of the caudality of lesion on the resultant changes in the electrophysiological responses, a comparison was carried out between ASOLG and PSOLG with respect to both the electrode sites. ASOLG obtained the mean latency of 143.65 (SD = 21.9) in comparison to the mean latency of 140.33 (SD = 28.64) at T4-Cz lead. No statistically significant difference was observed between the two groups. Likewise at T3-Cz lead, the ASOLG obtained the mean latency of 149.3 (SD=30.8) as compared to PSOLG which scored the mean latency of 148.76 (SD=26.9) which also did not demonstrate a statistically significant difference. These values have also been illustrated in Fig. A-II.

The results thus reveal the almost equal effect of anterior and posterior lesions on N1 components of AEPs. It
may also be pointed out that within each of these two groups, no marked variation was observed, across the two leads. The statistical values did not show the difference between T4-Cz and T3-Cz leads of PSOLG \( (P > .05) \) and the same was true with respect to the PSOLG \( (P > .05) \).

An analogous trend was observed with respect to the amplitudes. The ASOLG obtained the mean amplitude of 5.87 \( (SD = 2.9) \) at T4-Cz and 6.00 \( (SD = 2.7) \) at T3-Cz lead as compared to the PSOLG, which scored the mean amplitude of 4.8 \( (SD = 2.09) \) at T4-Cz and 6.3 \( (SD = 3.2) \) at T3-Cz leads. Evidently, no significant difference was observed at T4-Cz \( (t = 0.15, P > .05) \) and T3-Cz \( (T = 1.09, P > .05) \) leads between the two groups. (See Fig.A-III).

It was noted that 10% of the patients at both leads failed to produce this component. Similarly in 30% of the patients at T4-Cz lead and 10% at the T3-Cz lead of PSOLG, this component could not be detected. It appears that the proportion of patients who failed to produce this component is higher in case of PSOLG as compared to ASOLG especially with regard to the T4-Cz lead. (See fig.A-XIII)
A separate comparison was carried out between the \text{CG} and ASOLG in order to examine the difference between the two groups with respect to the latencies and amplitudes. This comparative analysis showed a statistically significant difference between the two groups at T4-Cz ($x^2=15.6, p=.001$) but not at T3-Cz ($x^2=0.03, p=.05$) with regard to the latencies. (See Fig. A-II) However, no such difference was observed with regard to the amplitudes with respect to T4-Cz ($t=0.03, p>.05$) and T3-Cz ($t=0.8, p>.05$) leads. The findings reveal that the lesion in the anterior structures of the cortex do prolong the latencies of this component, but the amplitudes remain unaffected. (See Fig A - X)

\textbf{COMPARISON BETWEEN RFSOLG AND LFSOLG:}

In order to understand the effect of unilateral frontal lesions on the N1 component of AEP, patients with frontal lesions were divided into the left and right sides SOLGs. The patients with bilateral lesions were dropped from this comparison. The RFSOLG obtained the mean latency of 141.6 (SD = 31.03) and 141.5 (SD = 19.8) with respect to T4-Cz and T3-Cz leads respectively. Evidently, no statistically significant differences was observed between the two groups with respect to T4-Cz ($t = 0.13, p>.05$) and T3-Cz leads ($x^2=1.2, p>.05$) but
a trend in terms of prolonged latencies of LFSOLG was apparently present. The mean values have been illustrated in fig A-III.

The mean amplitude of 5.7 (SD = 2.9) and (SD = 2.7) were produced by RFSOLG with respect to T₄-Cz and T₃-Cz leads. Similarly, the RFSOLG produced the mean amplitude of 5.7 (SD = 2.08) on T₄-Cz and T₃-Cz and T₃-Cz leads. No statistically significant difference was observed between the two groups on T₄-Cz (t= 0.08, p > .05 ) and T₃-Cz (t=0.24) p > .05) leads.

The 20% of the patients of LFSOLG group, 5% of the RFSOLG at T₄-Cz lead failed to produce N₁ component of AEPs. Whereas, with respect to T₃-Cz the 20% of LFSOLG and 5% of RFSOLG failed to produce this component. The values however, do not yield a very striking difference between the two groups, but the number of patients who evinced the absence of N₁ is higher in case of LFSOLG.

Thus both mean latencies, and the number of patients showing absence of this component suggest an important role of left frontal region in the regulation of N₁ component.
COMPARISON BETWEEN PSOLG AND NCG:

A comparison between PSOLG and NCG was not found to be statistically significant with respect to both T4-Cz ($X^2 = 2.4 \quad P > .05$) and T3-Cz ($X^2 = 0.063 \quad P > .05$) leads. The amplitudes also showed the similar trend on T4-Cz ($X^2 = 1.8 \quad P > .05$) and T3-Cz ($X^2 = 1.4 \quad P > .05$) leads. These results could suggest that the posterior structures have only a minimal role in the generation of this component. (see fig. A-II and A-X).

COMPARISON BETWEEN LSOLG AND RSOLG:

The LSOLG obtained the mean latency of 144.93 (SD = 43.8) as compared to RSOLG, which gained the mean latency of 137.67 (SD = 27.9) with respect to T4-Cz lead. No statistically significant difference was observed between the two groups ($X^2 = 0.90 \quad P > .05$). On the other hand, with respect of T3-Cz the LSOLG obtained a mean latency of 152.84 (SD = 23.8) and RSOLG gained a mean latency of 148.2 (SD=26.3), the t-test of statistical significance did not demonstrate a significant difference between the two groups ($t = 0.58 \quad P > .05$). It is to be noted that the LSOLG generally obtained prolonged mean
latency which might suggest a relatively important role of left hemisphere in the mediation of N1 component of AEP (See fig A-VI).

An analysis of mean amplitudes did not yield a statistically significant difference between the two groups. The LSOLG obtained the mean amplitude of 5.9 (SD = 2.9) and 5.8 (SD = 2.7) at T4-Cz and T3-Cz leads respectively. Whereas the RSOLG gained the mean amplitude of 5.3 (SD = 2.6) and 5.8 (SD = 2.9) and 5.8 (SD = 2.7) at T4-Cz and T3-Cz leads respectively.

It was noted that at least 2 and 3 subjects of LSOLG and RSOLG at both the leads failed to produce identifiable N1 component.

**COMPARISON BETWEEN DFSOLG, AFSOLG, PFSOLG and NCG:**

The mean latencies of each group have been shown in the table below:

<table>
<thead>
<tr>
<th></th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AFSOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3-Cz:</td>
<td>Mean</td>
<td>107.48</td>
<td>139.19</td>
<td>156.6</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>15.06</td>
<td>20.19</td>
<td>14.89</td>
</tr>
<tr>
<td>T4-Cz :</td>
<td>Mean</td>
<td>103.06</td>
<td>146.63</td>
<td>151.3</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>17.6</td>
<td>31.7</td>
<td>28.13</td>
</tr>
</tbody>
</table>
The table indicates that as compared to NCG, all the remaining three groups have delayed latencies. The PFSOLG and AFSOLG seem to have performed less well than DFSOLG in terms of their prolonged latencies.

Comparison between DFSOLG and APSOLG did not show a statistically significant difference at both T4-Cz ($Z = 0.38$, $P > .05$) and T3-Cz ($Z = 1.92$, $P > .05$) leads. Similarly, a comparison between APSOLG and DFSOLG also did not demonstrate a statistically significant difference at both T4-Cz ($Z = 0.54$, $P > .05$) and T3-Cz ($Z = 1.62$, $P > .05$) leads (See fig. A-V).

Similarly, with regard to the amplitudes, a comparison between DFSOLG and APSOLG ($Z = 0$, $P > .05$), PFSOLG and APSOLG ($Z = 0.33$, $P > .05$) at T4-Cz lead did not yield a statistically significant difference. Also with regard to T3-Cz lead, a comparison between DFSOLG and APSOLG ($Z = 0.02$, $P > .05$), PFSOLG and APSOLG ($Z = 0.35$, $P > .05$) and DFSOLG and PFSOLG ($Z = 0.36$, $P > .05$) did not demonstrate a statistically significant difference. PFSOLG had generally performed better than the other two groups (See Fig.XI).
### Table Showing Mean Amplitude of DFSOLG, AF_SOLG, PFSOLG and NCG:

<table>
<thead>
<tr>
<th></th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AF_SOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td>T4-Cz; Mean</td>
<td>6.8</td>
<td>6.21</td>
<td>7.39</td>
<td>6.88</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>3.8</td>
<td>3.2</td>
<td>4.83</td>
</tr>
<tr>
<td>T3-Cz; Mean</td>
<td>5.6</td>
<td>5.63</td>
<td>6.68</td>
<td>5.88</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>2.7</td>
<td>2.85</td>
<td>2.54</td>
</tr>
</tbody>
</table>

The mean values do not indicate a significant difference between all the three groups.

### Comparison Between Temporal and Parietal SOLGs:

A comparison between parietal SOLG and temporal SOLG showed an important trend; the prolonged latencies were observed in temporal SOLG where mean latency was 163.8 (SD = 27.2) as compared to parietal SOLG which obtained mean latency of 137.9 (SD = 30.9) on T3-Cz lead (Z = 1.11, P > .05) and 166.8 (SD = 22.3) and 141.5 (SD = 26.02) respectively at T4-Cz lead (Z=1.18, P > .05). In an other comparison, the left temporal SOLG obtained the mean latency of 175.03 (SD = 22.4) as compared to right temporal SOLG which gained the mean latency of 146.9
(SD = 31.5) on T4-Cz and a similar trend was observed on T3-Cz lead. (See fig. A-VIII)

The comparison between the left and right parietal SOLGs shows the mean latency of left parietal SOLG has 166.13 (SD = 10.3) and right parietal SOLG 130.9 (SD = 23.4) at T4-Cz lead and 162.43 (SD = 21.03) of left and 121.04 (SD = 28.6) of right parietal SOLG at T3-Cz lead.

This points to the general trend of more important role of the left parietal and temporal lobes in the regulation of N1. No such trend was observed with regard to the amplitudes. (See fig. A-VII). It was observed that 20% of the patients of temporal SOLG in each electrode failed to produce this component. 35% of the patients of parietal SOLG failed to produce this component. Thus, there was a slightly greater number of patients in parietal SOLG who did not produce N1 component of AEPs.

P2 COMPONENT OF AEPs:

NORMAL CONTROL GROUP:

P2 component was identified in all the neurologically normal subjects. The mean latency of 177.1 (SD = 228.8) on T4-Cz and 170.8 (SD = 7.3) on T3-Cz leads were recorded. No
the two electrode sites (2 > .05). The mean latency of this group was measured to be 7.56 (SD = 3.9) on T4-Cz and T3-Cz leads respectively (SD = 3.9) on T4-Cz and T3-Cz leads respectively (SD = 3.9) on T4-Cz and T3-Cz leads respectively. A comparison between the two groups demonstrated a statistically significant difference (x² = 25.2, P < .001) on T4-Cz lead and (x² = 30.4, P < .001) on T3-Cz lead. This difference has been illustrated in Fig A-I.

Within the SOLG, no marked difference was observed between the two leads (P > .05). With respect to amplitudes no significant difference was found between the two groups at T4-Cz (t² = 1.9 , P > .05 ) and T3-Cz (t= 1.3 , P > .05 ) leads. The SOLG obtained the mean amplitude of 5.2 (SD =2.5) and 5.4 (SD = 2.6) with respect to T4-Cz leads and T3-Cz leads respectively (See fig A-IX).

It was noted that in 40% of the patients this component was not detected at both the electrode sites.
**Comparison Between ASOLG and PSOLG:**

A comparison between the ASOLG and PSOLG with respect to the P200 component of AEPs did not yield a statistically significant difference on both the leads. The ASOLG gained the mean latency of 220.1 (SD = 26.7) and 217.2 (SD = 28.5) at T4-Cz and T3-Cz leads respectively. The PSOLG, on the other hand, obtained the mean latency of 227.9 (SD = 30.9) and 222.4 (SD = 31.7) with respect to T4-Cz and T3-Cz leads respectively.

The results did not demonstrate the differential effect of lesion locale on the P2 component at T4-Cz \( (t = 0.28, P > .05) \) and T3-Cz \( (t = 0.39, P > .05) \) leads. (See Fig. A-II)

Likewise with regard to amplitudes, no statistically significant difference was observed between the two groups. The ASOLG produced the mean amplitudes of 4.9 (SD = 2.2) and 5.6 (SD = 2.7) with respect to T4-Cz and T3-Cz leads respectively. Whereas, the PSOLG produced the mean amplitudes of 5.1 (SD = 2.2) and 4.6 (SD = 2.8) at T4-Cz and T3-Cz leads respectively. The difference with respect to T4-Cz \( (t = 0.98, P > .05) \) and T3-Cz \( (t = 0.89, P > .05) \) leads was not significant. (See Fig. A-X)

P2 component was found to be absent in at least 40% of the patients in ASOLG at each electrode site and 50% of the PSOLG at each electrode site. Thus, the absence was
slightly higher in the PSOLG.

**COMPARISON BETWEEN ASOLG AND NCG:**

A statistically significant difference was observed between the ASOLG and NCG at T4-Cz ($X^2 = 12.7, P < .001$) and T3-Cz ($X^2 = 22.7, P < .001$) and T3-Cz ($X^2 = 22.7, P < .001$) (See fig A-II). This comparison further demonstrates the marked impairment in terms of prolonged mean latency in ASOLG. However, no statistically significant difference was observed between the two groups on either of the leads with respect to amplitudes at T4-Cz ($X^2, P > .05$) and T3-Cz ($X^2 = 21, P > .05$) leads. (See fig A-X.)

**COMPARISON BETWEEN PSOLG AND NCG:**

The comparative analysis between PSOLG and NCG also demonstrated a statistically significant difference on T4-Cz ($X^2 = 11.6, P < .001$, and T3-Cz ($X^2 = 12.6, P < .001$) leads respectively. (See Fig A-II) with regard to amplitudes, no such difference was observed at T4-Cz ($X^2 = 1.7, P > .05$) and T3-Cz ($X^2 = 1.3, P > .05$) leads (see fig. A-X).
COMPARISON BETWEEN LSOLG AND RSOLG

The LSOLG obtained the mean latency of 213.6 (SD = 23.7) on T4-Cz lead and 207.6 (SD = 19.9) on T3-Cz lead. The mean latency of RSOLG was found to be 218.9 (SD = 31.9) on T4-Cz lead and 222.5 (SD = 27.7) on T3-Cz lead. On statistical comparison both the groups did not differ significantly at T4-Cz ($\chi^2 = 0.15$, $P > .05$) and T3-Cz ($\chi^2 = 2.7$, $P > .05$) leads (see fig. A-1).

With respect to amplitudes, the mean amplitudes of the LSOLG were found to be 6.4 (SD = 1.5) and 6.8 (SD = 2.9) on T4-Cz and T3-Cz leads. The RSOLG on the other hand obtained the mean amplitudes of 4.5 (SD = 2.2) and 4.8 (SD = 2.6) on T4-Cz and T3-Cz leads respectively. No significant difference was observed at T4-Cz ($t = 2.1$, $P > .05$) and T3-Cz ($t = 1.3$, $P > .05$) leads.

COMPARISON BETWEEN RFSOLG AND LFSOLG:

The RFSOLG obtained the mean latency of 223.3 (SD = 32.3) and 220.9 (SD = 24.3) on T4-Cz and T3-Cz leads respectively. On the other hand, the LFSOLG attained the mean latency of 205.6 (SD = 21.9) and 212.9 (SD = 18.2) on T4-Cz and T3-Cz leads respectively. No statistically significant difference was found between the two groups at T4-Cz ($t = 0.019$, $P < .05$) and T3-Cz
\( x^2 = 1.77 \ , \ P > .05 \) leads (See Fig.A-III).

With respect to the amplitudes, the RFSOLG obtained mean amplitude of 4.33 (SD = 2.2) and 6.00 (SD = 2.6) on T4-Cz and T3-Cz leads. The LFSOLG, on the other hand, produced mean amplitude of 6.34 (SD = 1.9) and 4.92 (SD = 2.03) on T4-Cz and T3-Cz leads respectively as is also illustrated in the Fig A-III. Here also, no statistically significant difference was observed at T4-Cz \( t = 1.72 \), \ P > .05 \) and T3-Cz \( t = 0.08 \), \ P > .05 \) leads.

It was noted that 23.5\% and 29\% of patients of RFSOLG at T4-Cz lead and T3-Cz leads respectively failed to produce this component. Whereas, with respect to LFSOLG, this component was found to be absent in 53.8\% and 61.5\% of the patients on T4-Cz and T3-Cz leads respectively. The absence is slightly higher in case of LFSOLG (see fig

**Comparison between DFSOLG, AF SOLG and PFSOLG**

DFSOLG, AF SOLG and PFSOLG obtained the mean latency of 215.6 (SD = 29.1) 224.7 (SD = 36.6) and 226.9 (SD = 33.7) on T4-Cz lead respectively, and on T3-Cz lead, the mean latency of each group was 221.2 (SD = 27.3) of DFSOLG, 234.3 (SD = 28.2) of AF SOLG, and 213.6 (SD = 37.7) of PFSOLG.
evidently on both the electrode sites, the AFSOLG and PFSOLG have comparatively more delayed mean latencies. This signifies the possible role of all the three subdivisions of frontal cortex, but more so, of anterior and posterior structures in the mediation of this component. Though statistically no significant different was observed between DFSOLG and AFSOLG at both T4-Cz (z=1.16, p > .05) leads; a comparison between PFSOLG and AFSOLG also did not yield a statistically significant difference at both T4-Cz (z = .74, p > .05) and T3-Cz (z = 1.14, p > .05). Similarly, a comparison between DFSOLG and PFSOLG also did not demonstrate a statistically significant difference between the two groups at T4-Cz (z = 0.21, p > .05) and T3-Cz (z = 0.50, p > .05) leads (see Fig A-V).

with respect to amplitudes DFSOLG, AFSOLG and PFSOLG obtained the mean amplitude of 5.4 (SD = .3), 3.9 (SD = 1.8) and 5.5 (SD = 4.07) on T4-Cz lead respectively, whereas on T3-Cz lead, DFSOLG obtained the mean amplitude of 6.4 (SD=2.9) AFSOLG, 4.9 (SD = 1.8) and PFSOLG, 4.7 (1.9) (see fig A-XI)

**COMPARISON BETWEEN PARIETAL AND TEMPORAL SOLGS:**

A comparison between parietal and temporal SOLGs show that the latter obtained prolonged mean latency as compared to the former. TSOLG obtained the mean latency of 231.3 (SD=38.9) and 231.2 (SD=62.7) at T4-Cz and T3-Cz leads respectively. PSOLG obtained the mean latency of 193.01 (SD=67.8) and 226 (SD = 21.1) on T4 Cz at T3-Cz leads respectively. The difference between the two groups was non significant at T4-Cz. (z = 0, p > .05) and at T3-Cz (z = .24, p > .05)

The comparison between the two groups based upon unilateral
obtained a mean latency of 234 (SD = 25.17) on T4-Cz lead, on the other hand, the RTSOLG produced a mean latencies of 231.2 (SD = 62.6) and 231.2 (SD + 62.6) on T4-Cz and T3-Cz leads respectively. The left PSOLG obtained mean latency of 226.8 (SD = 18.7) on both T4-Cz and T3-Cz electrode sites. The RTSOLG obtained mean latencies of 215.08 (SD = 33.02) and 225.4 (SD = 24.23) on T4-Cz leads respectively (See fig.A-VIV).

With regard to amplitudes, the PSOLG obtained the mean amplitude of 5.28 (SD = 2.04) on both T4-Cz and T3-Cz leads. On the other hand, the TSOLG produced mean amplitude of 6.38 (SD = 4.02), and 3.41 (SD = 1.62) on T4-Cz and T3-Cz leads.

The left right comparison on the basis of the site of lesion showed that the LTSOLG obtained mean amplitudes of 6.05 (SD = 1.06) and 4.2 (SD = 2.26) on T4-Cz and T3-Cz leads respectively. Whereas, the RTSOLG obtained a mean amplitude of 5.5 (SD = 2.3) and 5.63 (SD = 3.93) on both T4-Cz and T3-Cz leads respectively. The left parietal SOLG obtained mean amplitudes of 4.6 (SD = 2.4) and 3.41 (SD = 1.61) on T4-Cz and T3-Cz leads respectively. Whereas, the right parietal SOLG obtained a mean amplitude of 3.15 (SD = 1.48) on T4-Cz lead— and this component was absent in case of T3-Cz lead.
It was important to note that in general 16.6% and 3.3% of patients of TSOLO failed to produce this component on T4-CZ and T3-CZ leads. Whereas, 42.8% of PSOLO on each electrode failed to produce this component. Thus absence of his component was generally higher in parietal SOLG. (See fig A-14)

The results obtained from the ideometric interpretation of six tests related to attention reveal a striking impairment in SOLG on all the tests. The most significant impairment as seen with respect to the test of attention and mental set and a minimum impairment in terms of lesser number of patients showing this deficit was observed in case of the test of preservation. This profile is shown in fig C 11).

A comparative analysis of the impairment of ASOLO and SOLG revealed a greater impairment in case of ASOLO on all tests, which possibly indicate the greater role of anterior structures in the attentional processes (See fig 8,9)
FIG A-I-MEAN LATENCY (± SD) OF AEP COMPONENTS (P1, N1, AND P3) OF NORMALS AND PATIENTS WITH SPACE OCCUPYING LESIONS AT T6-Cz AND T3-Cz LEADS

FIG A-II - MEAN LATENCY (± SD) OF AEP (P1, N1, P3) OF NORMALS AND PATIENTS WITH ANTERIOR AND POSTERIOR CORTICAL LESIONS AT T6-Cz AND T3-Cz LEADS
Figure 1: Mean latency (± SD) of AEP components (P1, N1, and P3) of patients with the right and left frontal SOL at T4-Cz and T5-Cz leads.

Figure 2: Mean latency (± SD) of AEP components of normals and patients with frontal dorsolateral anterior and posterior space-occupying lesions at T4-Cz and T5-Cz leads.

- **N1 Component**
- **P1 Component**
- **P3 Component**
NUMBER OF PATIENTS WHO DID NOT PRODUCE AEP COMPONENT:
( P1, N1 AND P2 )
FIG. 2: MEAN LATENCY (± SD) OF AEP COMPONENTS (P₁, N₁, AND P₂) OF PATIENTS WITH RIGHT AND LEFT SOLS AT T₄-C₃ AND T₃-C₂ LEADS.
FIG. A-X: MEAN AMPLITUDES (± SD) OF AEPs (P1, N1, AND P2) OF NORMALS AND PATIENTS WITH SPACE OCCUPYING LESIONS AT T4 - Cz AND T3 - Cz LEADS

FIG. A-X: MEAN AMPLITUDE (± SD) OF AEPs (P1, N1, AND P2) OF NORMALS AND PATIENTS WITH ANTERIOR AND POSTERIOR LESIONS AT T4 - Cz AND T3 - Cz LEADS
Fig. AXXI: Mean amplitude (± SD) of AEPS (P1, N1, and P2) of patients with dorsal, anterior and posterior frontal lesions at T4-Cz and T3-Cz leads.

Fig. AXXII: Showing the percentages of patients and normals who produced and who did not produce P1 component of AEPs within each group at T4-Cz and T3-Cz leads.
FIGA-XIV SHOWING PERCENTAGES OF NORMALS AND PATIENTS WHO PRODUCED AND WHO DID NOT PRODUCE P1 COMPONENT WITHIN EACH GROUP AT T4-CZ AND T3-CZ LEADS
FIG - 23

CH1 AVERAGE 8.0000 REF +00001 000.000ms 623.77ms

CH2 AVERAGE 10.750 REF -5.2500 REF

CH1 AVERAGE 8.0000 REF -8.0000 REF 000.000ms 623.77ms

CH2 AVERAGE 16.000 REF -16.000 REF
The results presented below have been categorized into two sections: the section (A) deals with the results related to the amplitude of Bereitschafts potential. In this section, results have been presented according to the comparisons carried out between various groups on the basis of lesion locale. The section (B) on the other hand deals with the results related to the onset of BP. The results have also been presented according to comparisons between groups on the basis of lesion site.

At least three measures were considered and utilized for the comparative analyses. These measures were (1) amplitude (2) Onset (3) the presence or absence of BP. (Details are given in Chapter III). The absence of BP in terms of its morphological attributes was considered and analysed separately.

A. AMPLITUDES:

COMPARISON BETWEEN NCG AND SOLG:

NCG obtained the mean amplitude of -16.8 $(SD = 9.9)$ and -15.3 $(SD=7.9)$ at Cz and Fz leads. No significant difference was observed between the two electrodes sites. $(t=0.77, P>0.05)$ on the other hand, SOLG, obtained the mean amplitude of 12.9 $(SD=5.6)$ and 12.8 $(SD=8.47)$ at Cz & Fz leads, $t$-test was employed to examine the difference between the two groups. No statistically significant difference was observed at Cz.
(t = 1.9, \( P > .05 \)) and \( Pz \) (t =1.2, \( P > .05 \)) (See fig. B-I).

It was noted that on both the electrode sites 12 patients failed to produce this potential (see fig B- XI & B XII). The results indicate that the NCG obtained the higher mean amplitude values on both the electrode sites as compared to the SOLG as is shown in the Fig BI. Topographically, (between \( Cz \) and \( Pz \) leads) no significant difference was observed within the SOLG (t=1.31, \( P > .05 \)) however a slightly increased amplitude was observed at \( Cz \) lead of NCG. These results bring out the adverse effect of cortical lesions on the amplitude of BP.

**COMPARISON BETWEEN ASOLG, PSOLG and NCG:**

The two sub-divisions of the total sample on the basis of the lesion locale yielded following results: The patients with anterior lesions (ASOLG) obtained the mean amplitude of 41.32 (SD\( \pm \)3.91) and -13.05 (SD\( \pm \)8.08) at \( Cz \) and \( Pz \) leads respectively. These values indicate a slightly higher mean amplitude at \( Pz \) lead. But statistically no significant difference could be established (t=0.40, \( P > .05 \)). Thus, no topographical variation could be discerned across \( Cz \) and \( Pz \) electrode sites in ASOLG. On the other hand, the PSOLG obtained
the mean amplitude of 16.11 (SD=3.4) at Cz and Fz leads. Contrary to ASOLG, the mean amplitude was slightly higher mean amplitude at Cz lead but statistically no significant difference could be established across both the leads (t=0.9, p>.05). A significant difference was observed between ASOLG and PSOLG (t=2.25, P<.05) at Cz lead. The same was however not true at Fz lead where, no significant difference emerged between the two groups (t=0.14, P>.05). It was however important to note that generally, PSOLG obtained higher mean amplitudes. When compared with NCG, both the groups on both the leads were found to have lower mean amplitudes as compared to NCG. The independent comparisons between NCG and ASOLG yielded statistically significant difference at Cz lead (t=2.29, P<.05). With respect to Fz lead, no statistically significant difference was observed (t=0.93, P>.05). The comparison between NCG and PSOLG did not reveal statistically significant difference at Cz (t=0.23, P>.05) and Fz (t=0.66, P>.05) leads respectively. The results point out the vital role of the frontal lobe (ASOLG) in the production of BP amplitude as the anterior lesioned group showed larger impairment in terms of reduced amplitudes as compared to the patients with posterior lesions involving temporal and parietal lobes. (See fig B-II).
COMPARISON BETWEEN LFSOLG AND RFSOLG:

In order to examine the effect of unilateral frontal SOL on the frontal and vertex BP, the ASOLG was further bifurcated into left and right sided lesion groups. The RFSOLG obtained the mean amplitude of \(-10.45\) (SD=3.53) at Cz and \(-10.04\) (SD=2.24) at \(Fz\) leads respectively. Evidently, there was no topographical difference observed between the two electrode sites. \((p>0.05)\). On the other hand, the LFSOLG obtained the mean amplitude of \(-13.23\) (SD=4.8) and \(-15.21\) (SD=6.1) at Cz and \(Fz\) leads respectively. With in this group too, no statistically significant difference was observed between the two electrode sites \((p>0.05)\). The comparison between the two groups at Cz\((t=4, p>0.05)\) and \(Fz\) \((t=1.7, p>0.05)\) leads did not reach the level of statistical significance. However, LFSOLG obtained the higher mean amplitudes at both Cz and \(Fz\) leads in comparison to the RFSOLG. These values have been shown in the Fig B-III which possibly suggest the important role of right frontal lobe in the production of BP amplitudes.
The mean values of all the four groups have been shown in the following table:

<table>
<thead>
<tr>
<th></th>
<th>C2</th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AFSOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>-16.8</td>
<td>-12.39</td>
<td>-20.13</td>
<td>-10.74</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>9.9</td>
<td>3.56</td>
<td>5.02</td>
<td>4.54</td>
<td></td>
</tr>
</tbody>
</table>

The independent comparisons between NCG and the rest of the three groups namely DFSOLG, AFSOLG and PFSOLG were carried out separately. A significant difference was found between the NCG and DFSOLG at Cz (Z=3.16, P<.01) leads, but at Fz lead (Z=0, P>.05) this difference was not significant. A comparison between NCG and AFSOLG did not yield a statistically significant difference at Cz (Z=1.38, P>.05) and Fz (Z=1.25, P>.05) leads. On the other hand, a comparison between NCG and PFSOLG showed a statistically significant
difference at Cz (z=2.16, p <.01) and Fz (z=2.12, p <.01) leads. The results generally point out the amplitude decrement in all the three groups as compared to the NCG.

The comparisons were also carried out between these groups independently. The comparison between DFSOLG and AFSOLG did not reveal a statistically significant difference at both Cz (z=1.26, p >.05) and Fz (z=1.53, p >.05) leads. Similarly, a comparison between DFSOLG and PFSOLG also did not yield a statistically significant difference at Cz (z=1.92, p >.05) and Fz (z=0, p >.05) leads. An analogous trend was apparent in a comparative analysis between PFSOLG and AFSOLG at Cz (z=0.95, p >.05) and Fz (z=1.53, p >.05) leads. Though none of the comparisons showed a significant difference, the trend indicated a greater role of anterior and posterior frontal regions in the regulation of BP amplitude as a greater impairment in terms of decreased amplitude is evident in these groups. The mean differences between these groups have been shown in fig B-IV.

**COMPARISON BETWEEN TEMPORAL AND PARIETAL SOLGS:**

The bifurcation of the PSOLG was done to understand the individual contribution of the temporal and parietal structures in the production of BP amplitudes. The parietal SOLG obtained the mean amplitude of -13.62 (SD=3.48) and -13.33 (SD=7.56) at Cz and Fz leads respectively. Evidently,
no significant difference was found between both the electrode sites (t=0.67, p>.05). On the other hand, the temporal SOLG obtained the mean amplitude of -14.22 (SD=12.1) and -7.32 (SD=7.29) at Cz and Fz leads respectively. At Fz lead this group obtained considerably higher values. Though the difference between the two leads was non-significant (t=0.64, p>.05). It may be pointed out that the variance was very high especially in case of temporal SOLG.

A comparative analysis between the PSOLG and TSOLG did not show a significant difference between the two groups at both Cz (z=0.51, p>.05) and Fz leads (z=0.63, p>.05). Thus the results failed to establish a relationship between the impairment of amplitude and the specific posterior cortical region (temporal/parietal).

Also, a comparison between TSOLG and NCG did not demonstrate a significant difference at Cz (z=0.68, p>.05) and Fz leads (z=1.67, p>.05). Similarly, a comparison between PSOLG and NCG also did not show a statistically significant difference at both Cz (z=0.08, p>.05) and Fz (z= 0.5, p>.05) leads. These results indicate a minimum role of the posterior structures in the production of vertex (Cz) and frontal (Fz) amplitudes. The mean difference between these groups have been illustrated in fig. B-V.
B ONSET:

A separate analysis with respect to the onset is presented below. The groups were compared as was done in case of amplitudes.

COMPARISON BETWEEN NCG AND SOLG:

As a general comparison between the SOLG and NCG the mean difference was found to be statistically significant. NCG obtained the mean onset of -2.9 (SD=0.34) and -3.02 (SD=0.36) at Cz and Fz leads respectively. In comparison to it, the SOLG obtained the mean onset of -2.2 (SD=0.38) and -2.11 (SD=0.45) at Cz and Fz respectively. On Cz lead the statistical difference between the two groups reached (t=8.004, p<.001) the level of statistical significance. Likewise, this difference was also significant at Fz lead (t=8.9, p<.001).

It is important to mention that within the NCG, the early BP on set was found at Fz leads. There was a significant difference between the Cz and Fz leads of NCG (p<.05). Similarly within the SOLG a significant difference was found between the two electrode sites (t=3.0, p<.05). This was in the direction of the lower mean onset at Cz lead.
A significant difference between the NCG and SOLG at both the electrode sites points out that the BP onset is adversely affected by the lesion in the cortex irrespective of its site. These differences have been shown in fig B-VI.

**COMPARISON BETWEEN ASOLG, PSOLG AND NCG:**

A comparison between ASOLG and PSOLG did not show a statistically significant difference both at $C_z$ ($t=0.89$, $p>.05$) and $F_z$ ($t=0.41$, $p>.05$) leads. The ASOLG obtained the mean onset of -2.21 (SD=0.52) and -2.19 (SD=0.47) on $C_z$ and $F_z$ leads respectively, as compared to the mean onset of -2.07 (SD=29) close and -2.11 (SD=0.57) obtained by PSOLG on both $C_z$ and $F_z$ respectively. Hence, the localization of the lesions in the anterior and retro-rolandic structures did not affect the onset of BP differentially. It is important to note that within the ASOLG a significant difference was found between the mean onset values obtained at $C_z$ and $F_z$ leads ($t=6.8$, $p<.01$) in the direction of a delayed onset at $F_z$ lead.

In a separate comparison between the ASOLG and NCG, a glaring difference was observed between the two groups which was statistically significant at $C_z$ electrode site ($t=5.42$, $p<.01$). On the other hand, at the $F_z$ lead no significant difference was observed ($t=0.07$, $p>.05$).
The comparison between NCG and PSOLG also highlighted the statistical difference between the two groups at \( C_z \) leads \((t=7.88, p<.01)\), but not at \( F_z \) electrode site \((t=0.25, p>.05)\). Thus on both the above described comparisons, both ASOLG and PSOLG showed a marked delay in the BP onset as compared to NCG. These differences have been shown in the figure B-VII.

**COMPARISON BETWEEN LFSOLG AND RFSOLG:**

In order to evaluate the affect of the lateralized lesion on BP onset, the anterior SOLG was subdivided into two sub-groups namely LFSOLG and RFSOLG. No significant difference emerged with respect to \( C_z \) \((t=0.9, p>.05)\) and \( F_z \) \((t=0.17, p>.05)\) leads. The mean onset of LFSOLG was found to be \(-1.8 (SD=0.43)\) and \(-2.27 (SD=0.40)\) of RFSOLG at \( C_z \) lead. The trend however indicated the greater duration (early onset) of BP in RFSOLG. At \( F_z \) lead, the mean onset of \(2.3 (SD=0.33)\) was observed to be of the LFSOLG and RFSOLG respectively. These differences have been shown in figure B-VIII.

**COMPARISON BETWEEN DFSOLG, AFSOLG AND PFSOLG:**

The ASOLG was further classified into three sub-groups. Their mean and SD’s are shown in the table below.
A perusal of the table points out the highest mean onset value of the NCG. On the statistical analysis, a comparison between NCG and DFSOLG demonstrated a statistically significant difference at $C_z$ ($t=15.5$, $p<.01$) and $F_z$ ($t=5.9$, $p<.01$) leads. A comparison between NCG and AFSOLG did not reach the level of statistical significance at $C_z$ ($z=2.74$, $p>.01$) and at $F_z$ lead this difference was not statistically significant ($z=1.71$, $p>.05$). A comparison between NCG and PFSOLG did not reach the level of significance at $C_z$ ($z=0.63$, $p>.05$) lead, though this difference was significant at $F_z$ lead ($z=2.72$, $p<.01$). Hence the results generally point out the attenuated nature of BP onset in all the three patient groups.

The results of the difference between the DFSOLG and AFSOLG show a significant difference between the two groups.
at $C_z$ ($z=2.67$, $p<.01$) and $F_z$ ($z=2.6$, $p<.01$) leads. Which highlight the significantly reduced BP on set in DFSOLG.

A comparison between DFSOLG and PFSOLG yielded non-significant difference between the two groups at $C_z$ ($z=.30$, $p>.05$) and $F_z$ ($z=1.78$, $p>.05$) leads. On the other hand, a comparison between PFSOLG and AFSOLG at $C_z$ ($z=.39$, $p>.05$) and $F_z$ ($z=.91$, $p>.05$) yielded no significant difference (See fig B-IX). These group comparisons bring out the important role of dorsolateral and anterior frontal regions in the BP onset.

**COMPARISON BETWEEN PARIETAL AND TEMPORAL SOLGS;**

The temporal SOLG obtained the mean onset of 2.17 (SD=0.67) and 1.79 (SD=0.46) at $C_z$ and $F_z$ leads. No significant variation was found between the two electrode sites of this group ($t=0.79$, $p>.05$). Though a slightly higher mean onset was evident at Cz lead. The parietal SOLG obtained the mean onset of 2.00 (SD=0.32) and 1.13 (SD=0.33) at $C_z$ and $F_z$ leads respectively. No topographical difference was observed between the two electrode sites ($t=1.9$, $p>.05$).

A comparative analysis between TSOLG and PSOLG showed a non-significant difference at $C_z$ ($z=.66$, $p>.05$) and $F_z$ ($z=.78$, $p>.05$) leads. The results failed to establish the exclusive role of either of these posterior structures in the BP onset (See fig B-X.)
A comparison between NCG and TSOLG missed the level of statistical significance at \( C_Z \) \( (z=1.72, p>0.05) \) and \( F_z \) \( (z=1.86, p=0.05) \) leads. It was however interesting to observe a statistically significant difference between NCG and PSOLG both \( C_Z \) \( (z=4.0, p<0.01) \) and \( F_z \) \( (z=4.43, p<0.01) \) leads. These results might suggest the greater role of parietal lobe in regulation of BP onset.

A correlation co-efficient was carried out to determine the relationship between the magnitude of BP amplitude and the event \( (r=0.36) \). As is evident no such relationship could be established.

A sizable number of patients failed to produce BP, as seen in fig B-XII at least 31% patients did not produce BP in general. Within the ASOLG, 45% of patients did not produce BP. Similarly within PSOLG, 24% of patients did not produce BP. Similarly in LFSOLG and RFSOLG, 45% and 46% patients failed to produce BP. There was no clear cut indication of a strikingly higher number of patients with absence of BP in one particular group. Also there did not appear to be a relationship between the site of lesion and the absence of BP as is evident from the group-wise perusal (see fig B XII and B XIII).

Neuropsychological test findings based upon the ideometric interpretation revealed that patients with SOL generally performed...
poorly on all the five tests as is shown in fig. c-g. ASOLG performed poorly as compared to PSOLG (See fig. c-g). No association was observed between the neuropsychological deficits and the generation of BP. Also no clear cut differentiation could be established between the performance of those who produced and those who did not produce BP on neuropsychological tests. These differences have been shown in fig. B-XIV.
FIG. 1: MEAN BP AMPLITUDE (± SD) OF NORMALS AND PATIENTS WITH SPACE OCCUPYING LESIONS AT CZ AND Fz LEADS
MEAN AMPLITUDE IN MICROVOLTS

Fig 8: III - Mean AP amplitude (±SD) of patients

FZ  CZ

MEAN AMPLITUDE IN MICROVOLTS

Anterior and Posterior Cortical Lesions at 5z and
6.5z: Mean AP amplitude (±SD) of normal and patients

FZ  CZ

Anterior Solg  Posterior Solg
BIV-MI
WITH DORSAL ANTERIOR AND POSTERIOR
FRONTAL LESIONS AT CZ AND FZ LEADS

BIV-MI
MEAN AMPLITUDE (±SD) OF NORMALS
AND PATIENTS WITH TEMPORAL, PARIETAL LESIONS
AT CZ AND FZ LEADS

MEAN AMPLITUDE IN MICROVOLTS

CZ  FZ

TEMPORAL SOLG  PARIETAL SOLG

NCG
FIG.BIII-MEAN BP ONSET (± SD) OF NORMALS AND PATIENTS WITH SPACE OCCUPYING LESIONS AT Cz AND Fz LEADS.

FIG.BVII-MEAN BP ONSET (± SD) OF NORMALS AND PATIENTS WITH ANTERIOR AND POSTERIOR LESIONS AT Cz AND Fz LEADS.
FIG. BX-MEAN BP ONSET (± SD) OF PATIENTS WITH PARIETAL AND TEMPORAL LESIONS AT Cz AND Fz LEADS

- PARIETAL SOLG
- TEMPORAL SOLG

MEAN ONSET IN MILLISECONDS

Cz  Fz

FIG.BXI-MEAN BP ONSET (± SD) OF PATIENTS WITH THE RIGHT AND LEFT SPACE OCCUPYING LESIONS AT Cz AND Fz LEADS
FIG. 8-XIII. SHOWING THE NUMBER OF PATIENTS WHO PRODUCED AND WHO DID NOT PRODUCE 8P WITHIN EACH GROUP AT C0 LEAD

- Produced
- Not Produced

A: ADGAL
B: P3GAL
C: LSGAL
D: RGAL
E: LTBGAL
F: MP3GAL
G: LPGAL
H: RP3GAL
I: MP3GAL
J: LPPGAL
K: TEMPORAL BOLD
L: TEMPORAL BOLD
M: BILATERAL
FIG. B-XI—SHOWING THE NUMBER OF PATIENTS WHO PRODUCED AND WHO DID NOT PRODUCE BP WITHIN EACH GROUP AT PA LEAD

- A: ASOLO
- B: PBELA
- C: LBELA
- D: LBELA
- E: LFSOLO
- F: RFSOLO
- G: LPSOLO
- H: RPSOLO
- I: RPSOLO
- J: APOSOLO
- K: PBESOLO
- L: PAERIAL SOLO
- N: TEMPORAL SOLO
- M: BILATERAL

- PRODUCED
- NOT PRODUCED
FIG B.XIV IMPAIRMENT OF NUMBER OF PATIENTS ON NEUROPSYCHOLOGICAL TESTS

PATIENTS WHO PRODUCED BP

PATIENTS WHO DID NOT PRODUCE BP

A ATTENTION MENTAL SET
B VISUAL SCANNING
C PERSEVERATION
D DELAYED RESPONSE
E PSYCHO-MOTOR
F IDEATIONAL FLUENCY

NUMBER OF PATIENTS

A B C D E F A B C D E F

NEUROPSYCHOLOGICAL TESTS
RESULTS: CONTINGENT NEGATIVE VARIATION

The results related to this paradigm are presented according to the group comparisons carried out as per the sub-divisions conceived on the basis of lesion locale.

NORMAL CONTROL GROUP: The mean amplitude of this group with respect to the early component at Cz was found to be -11.4 (SD=6.9), and at Fz lead it was -12.64 (SD=4.8). With respect to the late component the mean amplitude at Cz lead was found to be -15.5 (SD=10.8) & at Fz lead it was -14.5 (SD=10.08). Apart from the higher amplitudes at both the leads this group did not show a statistically significant difference with regard to the comparison between vertex and frontal leads (p>.05). Similarly with respect to the early component also no marked variation was seen across both the leads (p>.05). The mean amplitudes of this group with respect to early and late components have been shown in fig. C-1.

It was important to note that CNV was identified in all the subjects with the exception of two.

COMPARISON BETWEEN NCG AND SOLG:

The SOLG obtained the mean amplitude of -8.8 (SD=4.3) at Cz and -8.23 (SD=4.4) at Fz leads with respect to the early component. No statistically significant difference
emerged between the two electrode sites (p>.05) as is shown in fig C-1. With respect to the late component the SOLG obtained the mean amplitude of $-10.46$ (SD=7.2) at Cz and $-9.17$ (SD=5.5) at Fz leads. No marked variation between the two leads was observed. The mean amplitudes were evidently higher in case of late component.

A comparative analysis between the SOLG and NCG did not show a statistically significant difference at the Cz lead of the early component ($t=1.77, \text{p}>.05$). Contrarily, a significant difference was observed between the two groups at Fz leads ($t=3.55, \text{p}<.01$). These findings indicate the impaired nature of the early component of CNV in the patient group. With regard to the late component a statistically significant difference was found at both Cz ($t=2.14, \text{p}<.05$) and Fz ($t=2.78, \text{p}<.01$) leads between the two groups. These differences have been shown in fig C-1.

It was striking to observe that a sizable number of patients failed to produce CNV. 33% of patients did not produce CNV at Cz and Fz leads in SOLG. This further indicated the adverse effect of cortical lesions on the production of CNV. (See fig 0-6)

In summary, the above results revealed (1) significant impairment of early component of CNV at Cz lead in SOLG.
significant impairment of late component of CNV at both Cz and Fz leads of SOLG. (3) Both groups did not show marked variation between the two electrode sites. (4) The number of patients who failed to produce CNV was strikingly higher in SOLG.

**COMPARISON BETWEEN NCQ AND ASOLG:**

The patients with frontal SOLG (ASOLG) obtained the mean amplitude of -9.48 (SD=4.9) and -7.43 (SD=4.63) at Cz and Fz electrode sites with respect to the early component of CNV. A statistically significant difference was observed between the NCQ and SOLG at Fz lead (t =3.38, P < .01), but this difference failed to reach the level of statistical significance at Cz lead ($X^2 = 1.98$ P $>$ .05). Also, the mean amplitude of early component was higher at Cz lead as compared to Fz lead. The impairment of early component of CNV was more pronounced at Fz lead. In general, the ASOLG performed poorly at both the electrode sites.

A similar trend was observed in case of late component of CNV where, no statistically significant difference was observed between the two groups at Cz lead ($X^2 = 0.38$, P $>$ .05). In contrast, at Fz lead, this difference reached the level of statistical significance ($X^2 = 5.4$, P $<$ .05). Thus, a general impairment in terms of reduced mean amplitude
Was obvious in case of the early and late components of CNV and this impairment was more pronounced at Fz lead. It was noted that number of patients at Cz (N = 16) and Fz (N = 12) leads failed to produce CNV components. The mean differences between the two groups with regard to the early and late components have been illustrated in Fig C-2.

COMPARISON BETWEEN NCG AND PSOLG:

With respect to the early component, no significant difference was observed between the two groups at Cz (t=1.8, p > .05). Thus with respect to early component of CNV, PSOLG tends to obtain lower amplitude as compared to NCG, but the difference between the two groups was significant only at Fz lead.

With respect to late component both at Cz (t=0.67, p > .05) and Fz (t=1.25, p > .05) leads, no significant difference emerged between the two groups. (See fig C-2).

Overall, the results illustrate that the posterior SOL possibly does not cause much impairment to the early and late components of CNV, a finding that does not hold for ASOLG
A comparative analysis carried out between these two groups across both leads yielded interesting results. The ASOLG obtained the mean amplitude of $-8.48$ (SD=5.69) at Cz lead of the early component of CNV. Whereas, the PSOLG obtained the mean amplitude of $-9.82$ (SD=5.69) at Cz lead of the early component of CNV. No statistically significant difference was found between the two groups at Cz lead ($X^2= 0.02, p > .05$). A similar trend was observed at Fz lead, where the ASOLG obtained the mean amplitude of $-7.43$ (SD=4.6) and PSOLG $-8.6$ (SD=6.4). This difference was also not significant ($t=0.54, p > .05$). Thus this comparative analysis of early component of CNV at the Cz and Fz leads of both the groups did not demonstrate a statistically significant difference between the two groups, but generally, at both the leads, a larger impairment in terms of relatively attenuated amplitudes of the ASOLG was obvious (See fig. C-2).

The findings with respect to the late component followed a similar trend at the Cz lead, the ASOLG obtained the mean amplitude of $-10.13$ (SD=6.6), and the mean amplitude of PSOLG was $-12.98$ (SD=9.02) thereby showing no statistically significant difference ($X^2 = 3.89, p > .05$). Likewise, at the Fz lead, the ASOLG obtained a mean amplitude of $-8.80$ (SD=2.14) and the PSOLG obtained a mean amplitude of $-10.20$ (SD=7.10). Obviously, there was no statistically significant difference.
between the two groups ($x^2=0.4, p>0.05$). The results are in line with the results obtained with respect to the early component. Even in the absence of statistically significant differences at both the leads, it was evident that the ASOLG performed more poorly than the PSOLG in terms of obtaining lower mean amplitudes. It was important to note that at Cz lead in ASOLG 44.8% of patients did not produce CNV. Likewise in PSOLG, 37.5% of patients at both the leads did not produce CNV. At Fz lead, in ASOLG 41.38% of patients did not produce CNV. Thus, in terms of absence of CNV, there appears to be no clear cut distinction between the two groups.

These findings highlight the following points: (1) Between ASOLG and PSOLG no statistically significant difference was observed at Cz and Fz leads with respect to early and late components of CNV. (2) ASOLG comparatively performed poorly as compared to PSOLG. (3) both anterior and posterior cortical structures seem to have an important role in the generation of CNV (early and late components) (4) a comparison between the two groups with respect to the absence of CNV components showed no striking difference, however, the absence of early and late component was slightly higher in case of ASOLG. (5) within PSOLG, as in case of ASOLG, no marked topographical variation was observed between Cz and Fz leads. The mean difference between the two groups is illustrated in fig.C-2.
The analogous trend was observed at both the leads of late component. The RFSOLG obtained the mean amplitude of \(-11.7\) (SD=8.10) at Cz leads whereas the mean amplitude of LFSOLG was \(-9.29\) (SD=4.40). No statistical significance difference was observed between the two groups \( (x^2= 1.65, p > .05) \). Although the larger impairment was observed in the LFSOLG in terms of relatively mean amplitudes, but at the Fz lead the RFSOLG obtained the mean amplitude of \(-7.7\) (SD = 4.09) and the LFSOLG obtained the mean amplitude of \(-8.13\) (SD = 4.1). Evidently there was no statistically significant difference between the two groups. \( (t=0.20, p > .05) \) (See fig C-3).

As was in the case of the early component, the left frontal cortex seems to have an important role in the production of late component of CNV at Cz lead whereas the right frontal cortex seems to have an important role to play in the mediation of this component at Fz lead.

It is to be noted that 67% of the patients of LFSOLG and 47% of patients of RFSOLG at Cz lead did not produce CNV. Whereas at Fz lead, 5% of LFSOLG and 47% of RFSOLG patients failed to produce CNV. This absence might indicate the severity of the pathological condition following a brain lesion (See fig C-6).
In order to ascertain the role of the right and left frontal structures in the production of CNV, a comparative analysis was carried out. The results at the Cz electrode site of the early component revealed that the RFSOLG obtained the mean amplitude of -11.05 (SD = 5.4) and LFSOLG attained the mean amplitude of -6.9 (SD = 4.02), and the LFSOLG attained the mean amplitude of -12.41 (SD = 6.31). Again, there was no statistically significant between the two groups (t = 1.9, p > .05). However, it was interesting to note that at Cz lead the larger impairment in terms of lower mean amplitude was observed in LFSOLG. Whereas the impairment was more pronounced in RFSOLG at Fz lead. (See fig. C3).

Thus, the results evince a trend whereby the left frontal cortex might have a primary role in the regulation or generation of CNV early component, (Cz) whereas, the right frontal cortex seems to have an important role in the regulation of this potential at Fz lead.
At least three sub-divisions within the frontal cortex were conceived, and their comparative results with respect to early and late components have been shown in the table below:

**EARLY COMPONENT**

<table>
<thead>
<tr>
<th></th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AFSOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean</strong></td>
<td>-11.4</td>
<td>-8.89</td>
<td>-8.29</td>
<td>-8.33</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>6</td>
<td>9</td>
<td>2.58</td>
<td>2.39</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th></th>
<th>DFSOLG</th>
<th>AFSOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean</strong></td>
<td>-12.64</td>
<td>-7.6</td>
<td>-9.5</td>
</tr>
</tbody>
</table>

---

The mean values with respect to the early component shown in the table above clearly indicate a striking difference between the mean amplitude obtained by NCG and the rest of the three groups. However, there is no indication of a statistically significant difference between the 4 sub-sub-groups with frontal lobe lesions: The comparison between AFSOLG & DFSOLG did not show a significant difference (Z= 0.23, p > .05).
Also, the comparison between AFSOLG and PFSOLG 
($z = 0.44$, $P > .05$) & DFSOLG & PFSOLG ($z = 0.23$, $P > .05$) 
did not show a statistically significant difference at Cz lead 
similarly at Fz lead, no significant difference was observed 
between DFSOLG & AFSOLG ($z = .48$, $P > .05$), & AFSOLG 
& PFSOLG ($z=0.45$, $P > .05$) and PFSOLG and DFSOLG ($z=0$, 
$P > .05$). Hence at both Cz and Fz leads with respect to 
the early component none of the independent group comparisons 
were found to be significant. Therefore the role of one 
independent frontal structure, in the production of the early 
component could not be determined.

With respect to the late component the mean values 
obtained by these groups are shown in the table below:

<table>
<thead>
<tr>
<th></th>
<th>NCG</th>
<th>DFSOLG</th>
<th>AFSOLG</th>
<th>PFSOLG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cz</td>
<td>-15.54</td>
<td>-7.07</td>
<td>-1.2</td>
<td>-5.00</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>10.14</td>
<td>3.46</td>
<td>6.06</td>
<td>2.5</td>
</tr>
<tr>
<td>Fz</td>
<td>-14.5</td>
<td>-9.6</td>
<td>-9.44</td>
<td>-4.09</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>10.08</td>
<td>6.5</td>
<td>3.9</td>
<td>1.7</td>
</tr>
</tbody>
</table>
As was observed in case of the early components, all the three groups scored considerably lower amplitudes as compared to NCG. With respect to the late component the statistical analysis did not show a significant difference between AFSOLG and AFSOLG (Z=1.84, P > .05) PFSOLG and AFSOLG (Z=0, P > .05) and DFSOLG and PFSOLG, (Z=0, P > .05). With respect to the Fz lead the comparison between DFSOLG and AFSOLG (Z=0.23, P > .05), AFSOLG and PFSOLG (Z = .74, P > .05) and DFSOLG and PFSOLG (Z = .90, P > .05) also did not demonstrate a significant difference (See fig C-4).

Thus the above results do not clearly establish the exclusive role of a particular frontal region in the production of the early and late components of CNV.

COMPARISON BETWEEN TEMPORAL AND PARIETAL SOLG

A comparison between temporal and parietal SOLG was carried out for the purpose of determining the specific roles of temporal and parietal lobes in the production of early and late components of CNV.

The temporal SOLG obtained the mean amplitude of -10.41 (SD=8.73) and -8.24 (SD=4.04) at Cz and Fz leads
of early and late components. The parietal SOLG obtained the mean amplitude of $-8.26$ (SD=3.62) and $-11.00$ (SD =7.15) at Cz and Fz leads with respect to the early components. The results indicate a discrepancy with regard to Cz and Fz leads of both the groups. The temporal SOLG obtained slightly higher amplitude and the statistical analysis did not show a significant difference at Cz lead ($Z=0.027$, $p > 0.05$), at the Fz lead this group attained the higher mean amplitude though the difference between the two electrode sites was not significant ($Z=0.05$).

With respect to the late components, the temporal SOLG obtained the mean amplitude of $-12.5$ (SD=9.5) and $-12.9$ (SD=7.8) at Cz and Fz leads respectively. No topographical variation was evident between the two electrode sites. The parietal SOLG on the other hand obtained the mean amplitude $-15.5$ (SD =10.2)and $-10.00$ (SD =5.9) at Cz and Fz leads respectively (See fig C-5).

A comparison between temporal and parietal SOLGs across Cz lead did not show a significant difference ($Z=0$ $p > 0.05$). Similarly at Fz lead also, it missed the statistically significant difference ($Z=1.91$, $p > 0.05$) Thus the results failed to establish a clear evidence for
the exclusive role of temporal or parietal lobes in the production of early late components of CNV.

It may also be pointed out; at least 27.27% of patients of parietal SOLG and 20% of patients of temporal SOLG failed to produce CNV at Cz lead. Likewise 45.45% patients of parietal SOLG and 20% of patients of temporal SOLG did not produce CNV at Fz leads. No clear cut difference with regard to the absence of CNV emerged between the two groups.

NEUROPSYCHOLOGICAL TEST FINDINGS:

An ideometric interpretation of these tests shows that a number of patients performed poorly on all the tests as is shown in fig C-9. The test of attention/ and mental set evoked a higher degree of impairment, as compared to ASOLG, pSOLG exhibited less impairment on all these tests as has been shown in fig C-8. It was also observed that patients who did not produce CNV showed greater impairment than those who produced CNV as has been illustrated in fig C-7.
FIG. C 1 - MEAN CNV AMPLITUDE (± SD) OF NORMALS AND PATIENTS WITH SOL OF EARLY AND LATE COMPONENTS AT CZ AND FZ LEADS

EARLY COMPONENT

LATE COMPONENT

P < 0.05

P < 0.01

NCG

SOLG
FIG. C2 MEAN CNV AMPLITUDE (± 90) OF NORMALS AND PATIENTS WITH ANTERIOR AND POSTERIOR LESIONS OF EARLY AND LATE COMPONENTS AT Cz AND Fz LEADS

EARLY COMPONENT

LATE COMPONENT

FIG. C3 MEAN CNV AMPLITUDE (± 90) OF NORMALS AND THE RIGHT AND LEFT FRONTAL SOLGOS OF EARLY AND LATE COMPONENTS AT Cz AND Fz LEADS (CNV)
**Fig. 4**  Mean CNV amplitude (± SD) of early and late components of patients with posterior, dorsal, anterior frontal lesions at Cz and Fz leads.

**Fig. 5**  Mean amplitude (± SD) of early and late components of patients with temporal and parietal lesions at Cz and Fz leads (CNV).
Fig. C-6 showing the percentages of patients who produced and who did not produce CNV.
FIG. C-7 - IMPAIRMENT OF NUMBER OF PATIENTS ON NEUROPSYCHOLOGICAL TESTS

☐ PATIENTS WHO PRODUCED CNV ☐ PATIENTS WHO DID NOT PRODUCE CNV

A. ATTENTION MENTAL SET
B. VISUAL SCANNING
C. PERSEVERATION
D. DELAYED RESPONSE
E. PSYCHO-MOTOR
F. IDEATIONAL FLUENCY

NUMBER OF PATIENTS

8 12 18 18 22 1

NEUROPSYCHOLOGICAL TESTS
**Fig. C-8 - Impairment of Number of Patients on Neuropsychological Tests**

<table>
<thead>
<tr>
<th>ASOLG (Frontal SOLG)</th>
<th>PSOLG (Parietal &amp; Temporal SOLG)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A. Attention Mental Set</td>
</tr>
<tr>
<td></td>
<td>B. Visual Scanning</td>
</tr>
<tr>
<td></td>
<td>C. Perseveration</td>
</tr>
<tr>
<td></td>
<td>D. Delayed Response</td>
</tr>
<tr>
<td></td>
<td>E. Psycho-Motor</td>
</tr>
<tr>
<td></td>
<td>F. Ideational Fluency</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>NEUropsychological Tests</th>
<th>NUMBER OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>22</td>
</tr>
<tr>
<td>B</td>
<td>22</td>
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<td>C</td>
<td>22</td>
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<tr>
<td>F</td>
<td>10</td>
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<table>
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</thead>
<tbody>
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</tr>
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<td>C</td>
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<td>D</td>
<td>2</td>
</tr>
<tr>
<td>E</td>
<td>2</td>
</tr>
<tr>
<td>F</td>
<td>2</td>
</tr>
</tbody>
</table>
IMPAIRMENT OF NUMBER OF PATIENTS ON NEUROPSYCHOLOGICAL TESTS

ASIOLG (FRONTAL SOLG)
N = 16

PSIOLG (PARIETAL/TEMPORAL SOLG)
N = 5

A ATTENTION MENTAL SET
B VISUAL SCANNING
C PERSEVERATION
D DELAYED RESPONSE
E PSYCHO-MOTOR
F IDEATIONAL FLUENCY
FIG.C.10. IMPAIRMENT OF NUMBER OF PATIENTS WHO DID NOT PRODUCE C MV AND BP, ON NEUROPSYCHOLOGICAL TESTS

A SOLG (FRONTAL SOLG )
N = 8

P SOLG (PARIETAL - TEMPORAL SOLG )
N = 2

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
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</thead>
<tbody>
<tr>
<td>N</td>
<td>20</td>
<td>18</td>
<td>16</td>
<td>14</td>
<td>12</td>
<td>10</td>
</tr>
</tbody>
</table>

A ATTENTION MENTAL SET
B VISUAL SCANNING
C PERSEVERATION
D DELAYED RESPONSE
E PSYCHO-MOTOR
F IDEATIONAL FLUENCY

NEUROPSYCHOLOGICAL TESTS
Figure II: Impairment of Number of Patients on Neuropsychological Tests

A1 Attention Mental Set  
B Visual Scanning  
C Perseveration  
D Delayed Response  
E Psychomotor  
F Ideational Fluency

<table>
<thead>
<tr>
<th>TESTS</th>
<th>A1</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
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<tr>
<td>Number of Patients</td>
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<td>22</td>
<td>8</td>
<td>20</td>
<td>18</td>
<td>24</td>
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</table>

Neuropsychological Tests
FIG. 29-32: TYPICAL CNV RECORDINGS OF NORMALS AND PATIENTS