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CHAPTER 7

Electrophysiological Representation of the Upper and Lower Half Field

7.1 Introduction

The Visual Evoked Response to upper half field stimulation has been studied by several groups who have reached conflicting conclusions as to the latency, polarity and location of the response (Michael and Halliday 1971, Lehmann and Skrandies 1979). A polarity inversion of the response has been suggested with the maximal response occuring in the same location with both upper and lower field stimulation (Michael and Halliday 1971). They concluded that a polarity reversal to upper octant stimulation was due to the fact that the generators lie on the under surface of the occipital lobe, the dipolar source orientations are therefore inverted with respect to one another. Using pattern onset stimulation Jeffreys and Axford (1979) supported the concept that the upper response was a polarity reversal and suggested that the pattern reversal response was a composite containing two distinct overlapping components, these having the same polarity on lower field stimulation where they overlap temporally. This has been disputed by Lehmann and Skrandies (1979) who concluded that there was a latency shift in the maximum response after upper half field stimulation with no apparent polarity reversal. The location of the maximum positivity was over the posterior region of the scalp, consistent with the upper field projection to the visual cortex. A significant increase in the latency of the upper half field response has been verified (Flanagan and Harding 1986). Employing the technique of source localisation a stationary source was demonstrated irrespective of the stimulating field with only a shift in latency, this finding lends support to those of Lehmann and Skrandies.

A functional superiority of the upper hemiretina over the lower retina has been suggested from contrast sensitivity measurements (Skrandies 1985), visual acuity (Millodot and Lamont 1974) and electrophysiological recordings (Lehmann and Skrandies 1979, Skrandies, Richter and Lehmann 1984). This is supported by reports on the density of photoreceptors, demonstrating an increased density in the upper retina (Osterberg 1935). Studies on the electrooculogram light peak amplitude have also shown that a reduced amplitude is elicited from the lower retina at 10° eccentricity, in addition the lower retina also showed a lower sensitivity to light increments (Skrandies and Baier 1986), thus suggesting that light stimuli

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presented to the upper retina are followed by increased activity in interactions between the photoreceptors and retinal pigment epithelium. Contrary to these reports recent studies on contrast sensitivity have shown no asymmetry at eccentricities of 8° and 40° in the superior and inferior visual fields, visual acuity was also found to decline at a similar rate in both fields (Anderson, Mullen and Hess 1991).

7.2 The Topographic Distribution of the Upper and Lower Half Field Response

7.2.1 Method

Group mean waveforms and maps were constructed from the data from 12 subjects with an age range 22-55 years, all with 6/6 acuity wearing corrections if necessary. Twenty one silver-silver chloride electrodes were placed over the occipital area, see fig. 5.2. Before the electrodes were positioned the scalp surface was gently abraded with Omniprep™. The electrodes were then attached with blenderm™ tape, or glue if required and the electrode impedence was maintained below 5KΩ. The reference electrode was positioned at Fz.

Responses were recorded on a Biologic Brain Atlas III mapping system, recording protocol was the sane as that used in chapter 4. The amplifiers were set to a gain of 30,000, the filters used were high pass 1Hz and low pass 30Hz (3dB down point and 12dB/octave roll off). The time window was set at 512ms. Fifty responses were recorded for each stimulus presentation, and each stimulus was presented at least twice. Once the response had been recorded it was stored on disc for subsequent analysis. Monocular right eye stimulation was used for all the studies unless otherwise stated.

The check size used was fixed at 27' for all the stimuli, the field size used was 0-10°. The potential distribution is presented as maps at the position of the major peaks in the response.

7.2.2 Results

	N75	P80	P100	N105	P120	N145
Upper HF	9 subjects	3 subjects	12 subjects	----------	10 subjects	12 subjects
Lower HF	11 subjects	3 subjects	12 subjects	----------	4 subjects	12 subjects

Table 7.1 The Number of Subjects Producing the Different Components of the Pattern Reversal Response to Upper and Lower Half Field Stimulation.

	N75	P80	P100	N105	P120	N145
Upper HF	A	P	P		A	P
Lower HF	P	---------- -	CE		P	CE

Table 7.2 The Location of the Maximum Potential From Group Mean Maps for the Components Recorded After Upper and Lower Half field Stimulation.

Latencies of Major Components (ms)

	Upper	Lower
N75	76.44 (4.56)	73.45(7.43)
P100	101.8(8.92)	103.2(6.18)
P120	127.6(8.42)	119.5(4.43)
N145	154(11.05)	141 (8.84)

Amplitudes of Major Components (μV)

Upper	Lower
-2.69(2.69)	-1.66(1.73)
4.46(1.48)	8.56(3.09)
3.48(1.09)	4.23(2.07)
-2.62(1.12)	-5.64(3.48)

Table 7.3 The mean and standard deviation, in brackets, of the latencies and amplitudes of the major components in the response following upper and lower half field stimulation.

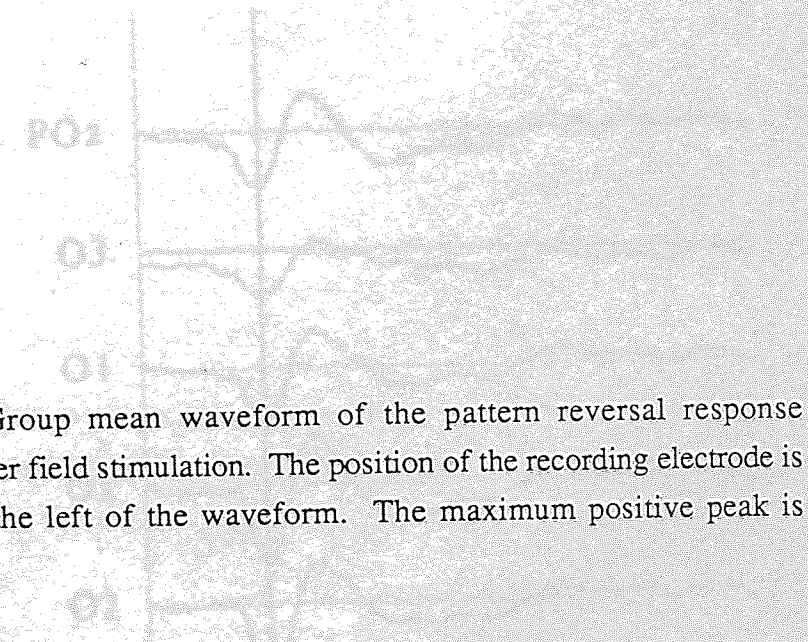


Figure 7.1. Group mean waveform of the pattern reversal response following lower field stimulation. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.

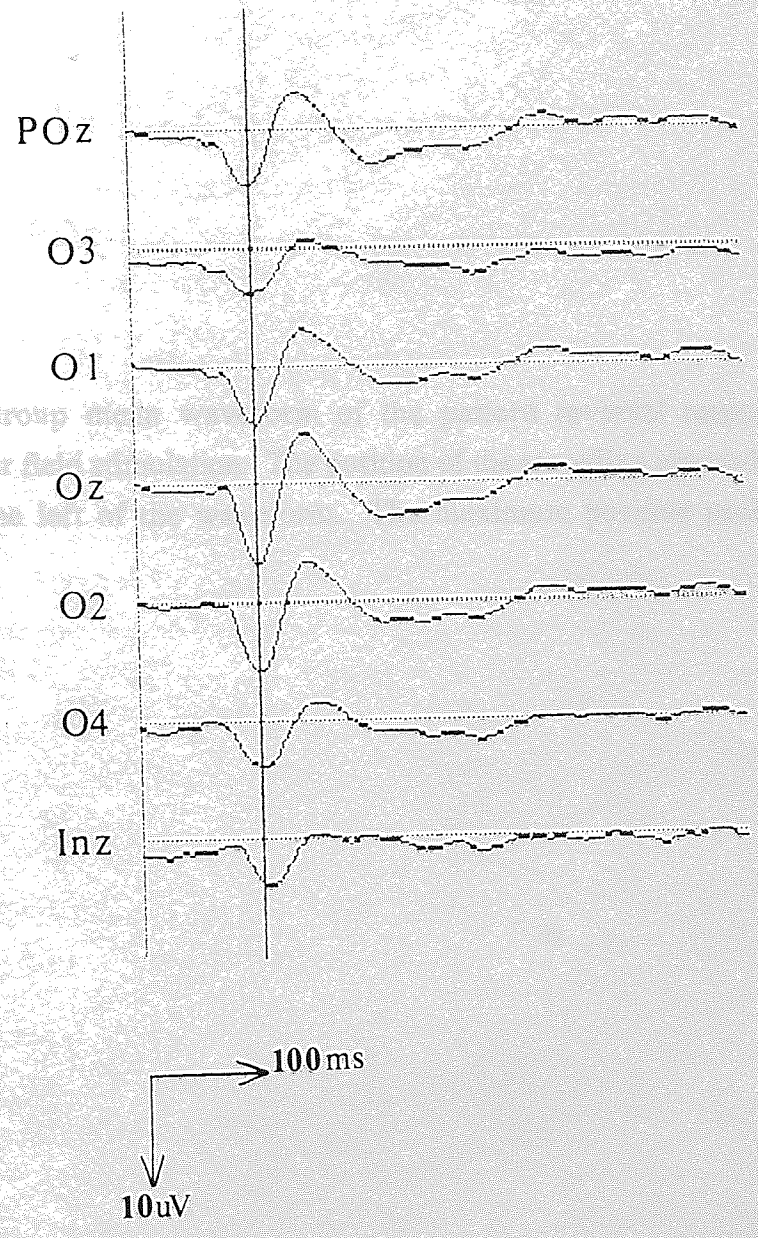
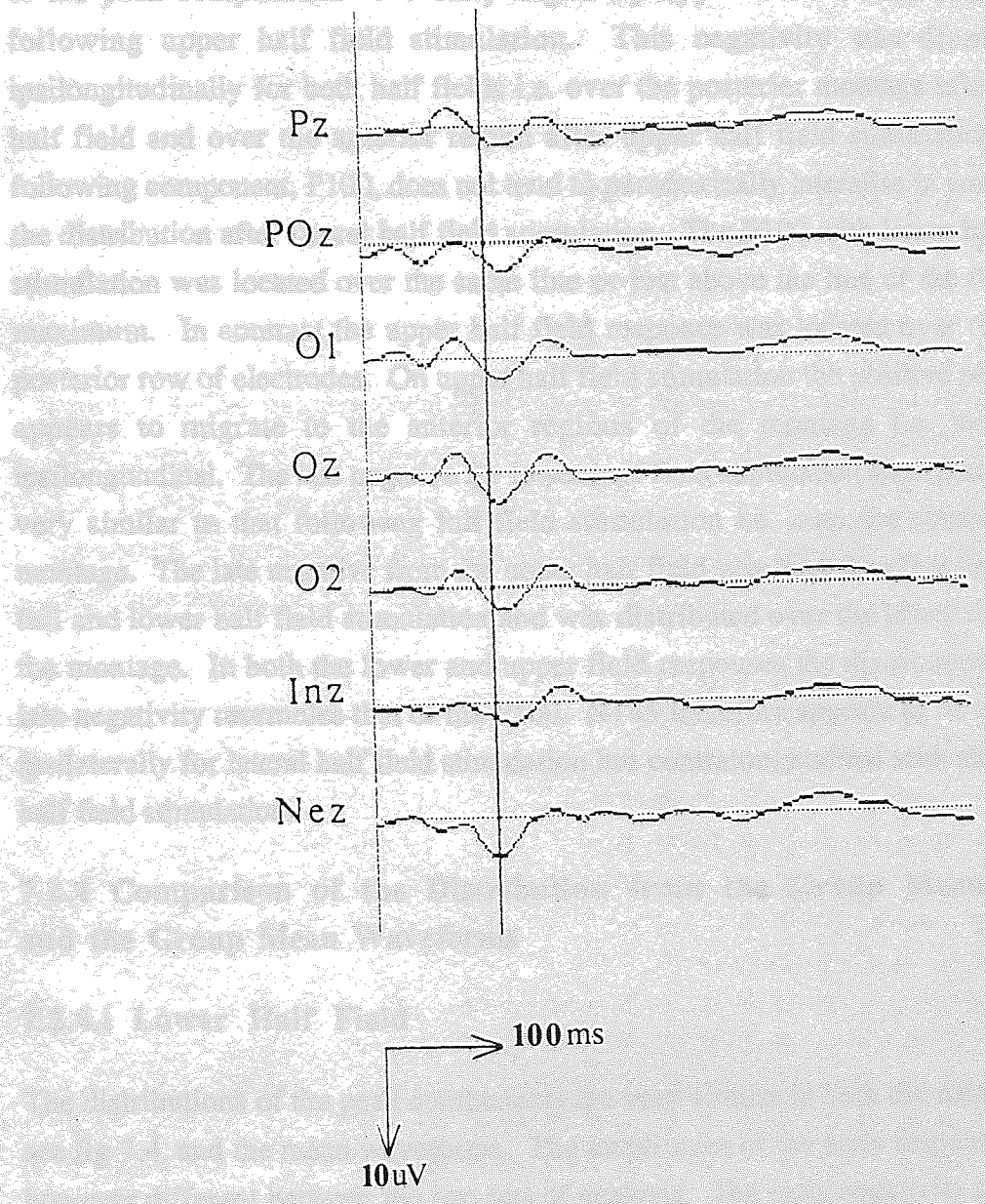

POz
O3
O1
Oz
O2
O4
Inz
100ms
10uV

Figure 7.2. Group mean waveform of the pattern reversal response following upper field stimulation. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.


Pz
POz
O1
Oz
O2
Inz
Nez
100 ms
10uV

7.2.3 Upper and Lower Half Fields

7.2.3i Topographic Distribution from Group Mean Waveform

Figures 7.1 and 7.2 illustrate the group mean waveforms of the upper and lower half field responses, figure 7.3 shows the topographical distribution at the latency of the peak components. The early negativity appears to be more prominent following upper half field stimulation. This negativity was distributed ipsilongitudinally for both half fields i.e. over the posterior montage after lower half field and over the anterior region after upper half field stimulation. The following component, P100, does not tend to paradoxically lateralise in contrast to the distribution after lateral half field stimulation. The P100 with lower half field stimulation was located over the same line or just above the line of the full field maximum. In contrast the upper half field response was located over the most posterior row of electrodes. On upper half field stimulation the positive peak then appears to migrate to the anterior regions of the montage i.e. becomes ipsilongitudinal. The late negative for lower half field stimulation has a distribution very similar to that following full field stimulation i.e. over the centre of the montage. The late negative from the upper half field was later than that from both full and lower half field stimulation and was distributed over the lower region of the montage. In both the lower and upper field responses the distribution of this late negativity resembles that of the P100. N145 therefore appears to be maximal ipsilaterally for lateral half field stimulation but contralongitudinal with altitudinal half field stimulation.

7.2.4 Comparison of the Distribution from the Group Mean Maps and the Group Mean Waveforms

7.2.4.i Lower Half Field

The distributions of the peak components are very similar in both the mean maps, see fig 7.4. and the mean waveforms. The amplitudes of the early responses were however different between the two sets of analysis. The early negativity appeared to be larger with the group mean maps, in contrast the early positivity was largest with the group mean waveforms. No late positive was observed in the group mean waveforms, this may be due to the fact that this peak was only recorded from four subjects. In the group mean map this peak was maximally distributed over the posterior region of the montage.

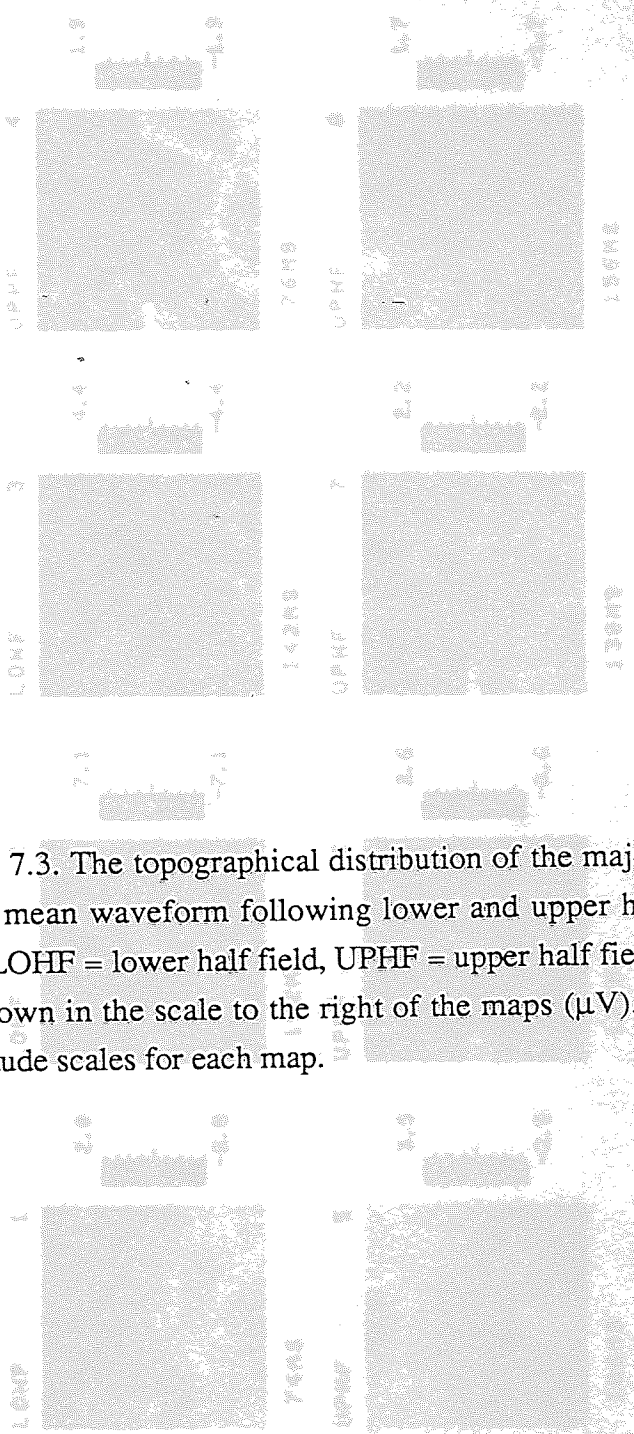
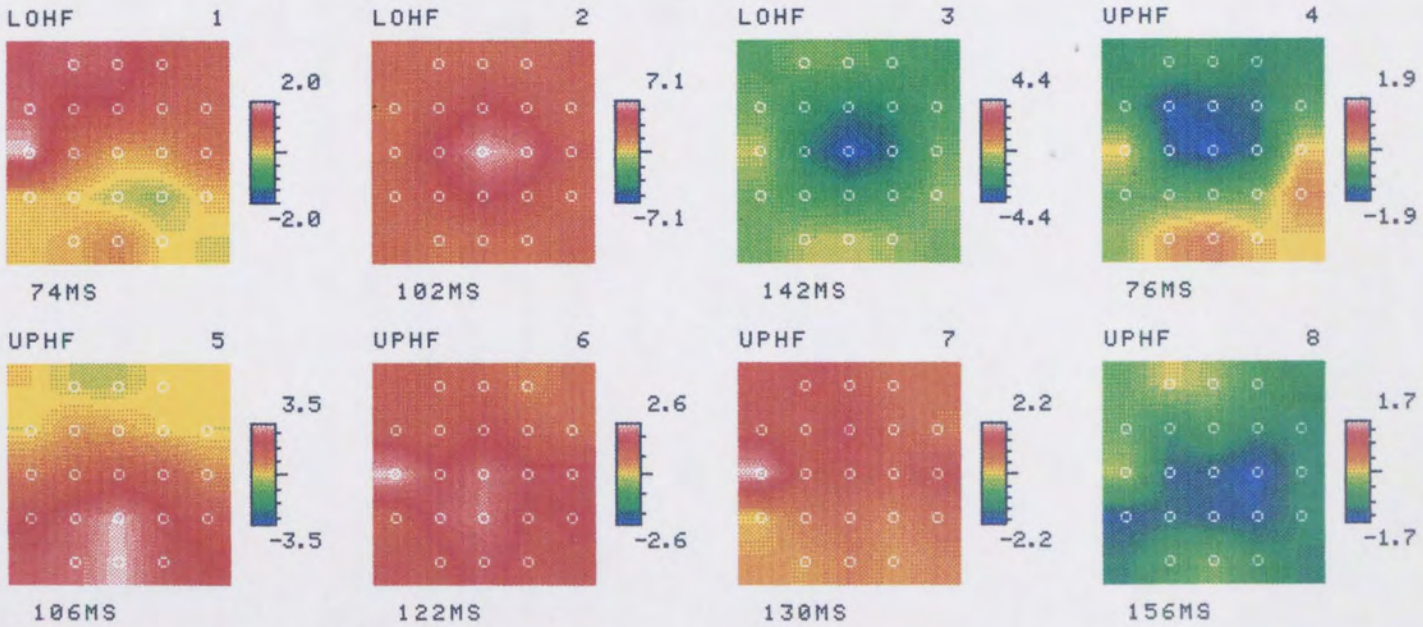


Figure 7.3. The topographical distribution of the major components in the group mean waveform following lower and upper half field stimulation. Key; LOHF = lower half field, UPHF = upper half field. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.


LOHF 1
2.0
-2.0
74MS
LOHF 2
7.1
-7.1
102MS
LOHF 3
4.4
-4.4
142MS
UPHF 4
1.9
-1.9
76MS
UPHF 5
3.5
-3.5
106MS
UPHF 6
2.6
-2.6
122MS
UPHF 7
2.2
-2.2
130MS
UPHF 8
1.7
-1.7
156MS

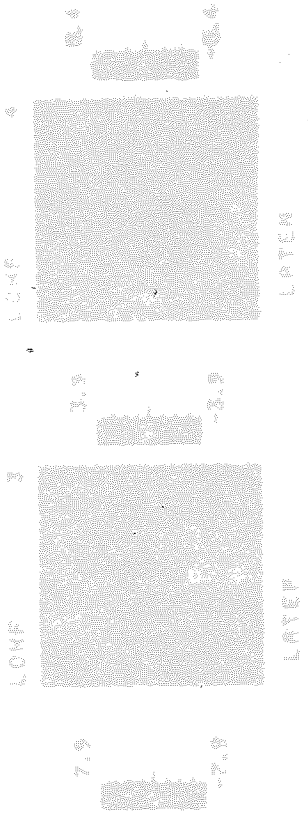
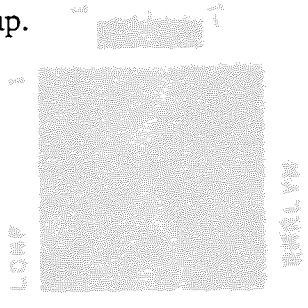
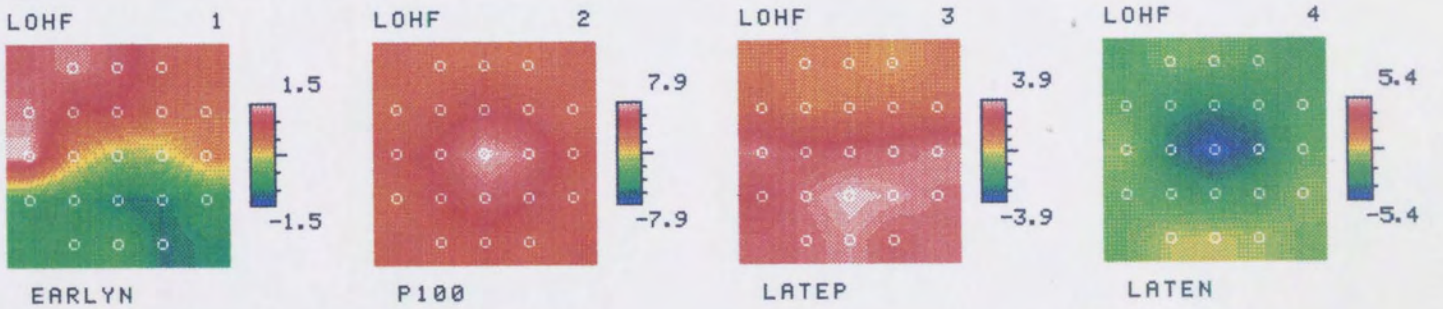


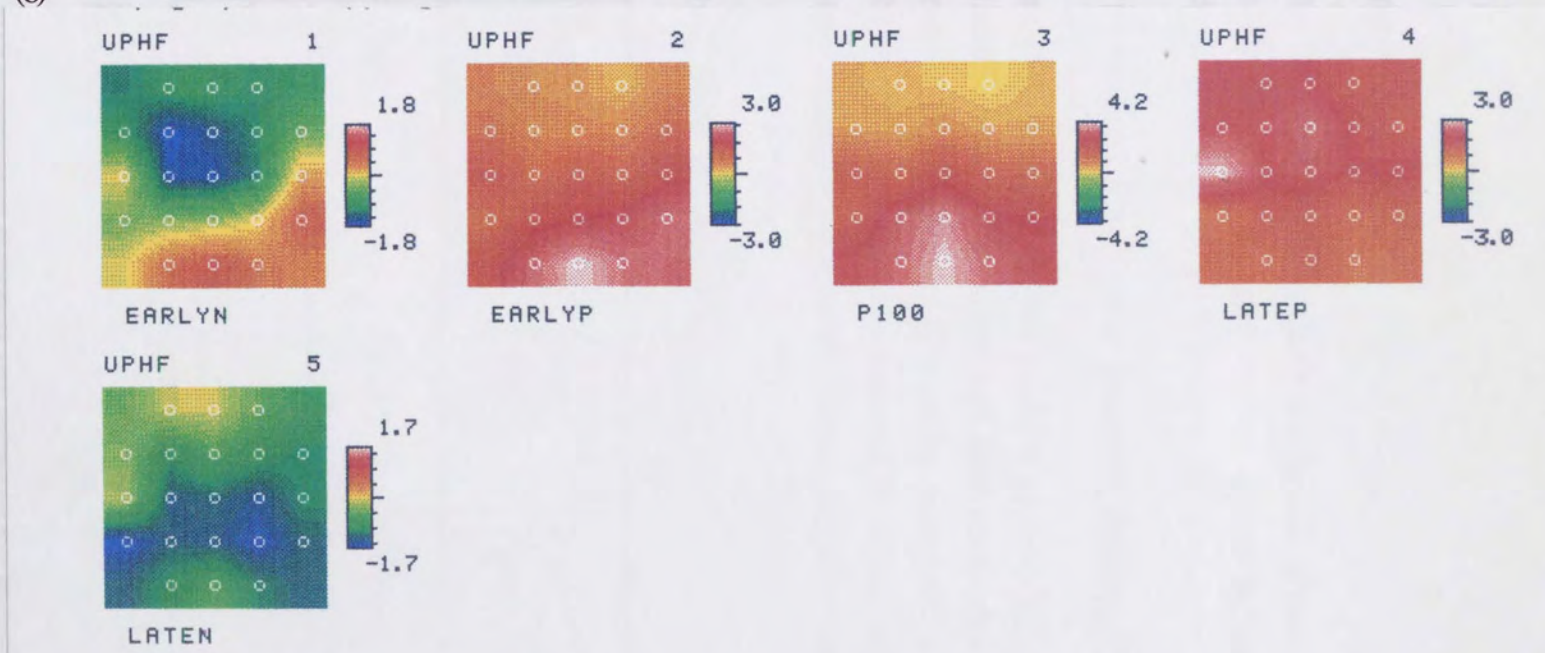
Figure 7.4 a and b. The topographical distribution of the major components in the group mean map following lower (a) and upper half field (b) stimulation. Key; LOHF = lower half field, UPHF = upper half field, EARLYN = early negativity, EARLYP = early positivity, LATEP = late positivity and LATEN = late negativity. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.



(a)


LOHF 1
1.5
-1.5
EARLYN
LOHF 2
7.9
-7.9
P100
LOHF 3
3.9
-3.9
LATEP
LOHF 4
5.4
-5.4
LATEN

(b)


UPHF 1
1.8
-1.8
EARLYN
UPHF 2
3.0
-3.0
EARLYP
UPHF 3
4.2
-4.2
P100
UPHF 4
3.0
-3.0
LATEP
UPHF 5
1.7
-1.7
LATEN

7.2.4.ii Upper Half Field

Similarly after upper field stimulation the distribution of the peak components in the group mean maps, see fig.7.4. and the group mean waveforms were comparable. In contrast the amplitude of the early positive was greater in the group mean map.

7.2.5 Morphological Variations Between Subjects

Forty percent of the subjects did not produce an early negativity with lower half field stimulation. However on upper half field stimulation only twenty percent failed to produce this response. The major positivity was recorded from all subjects. The later positive peak was recorded in 40% of the subjects to lower and in 90% to upper half field stimulation. The late negative was again recorded in all subjects.

7.2.6 Global Field Power Analysis.

Stimulus Location	Field Power Latency	Field Power Amplitude	Field Power Latency	Field Power Amplitude
Upper Half Field	**98ms**	**1.17**	142ms	0.56
Lower Half Field	**104ms**	**1.18**	134ms	1.16

Table 7.4 The latencies and strengths of the peaks following global field power analyisis of the group mean wavefroms following upper and lower half field stimulation.

Lower half field stimulation produces two peaks in the global field plot with similar strengths, these represent a central positive followed by a central negative with approximately the same distribution. The maximal field power following upper field stimulation is produced by a posterior positivity and an anterior negativity. The latency of the maximal field power was earlier for the upper field response than the maximal positivity in the group mean waveform, this resulted in the anterior negativity being slightly more prominent producing a more dipolar distribution. The lower field respone was at a similar latency.

7.2.7 Comparison of the Peak Components Following Lower and Upper Half Field Stimulation

7.2.7i N75

No significant difference in latency or amplitude was demonstrated after statistical analysis, using the Student t test, although this peak did appear to be more prominent following upper field stimulation.

7.2.7ii P100

Michael and Halliday have previously proposed that the major peak components from the two altitudinal half fields are of opposite polarities and of equal amplitudes, this was not confirmed in this study. A major positive peak was recorded after both lower and upper half field stimulation, the amplitude of the upper half field response was found to be significantly less than the lower half field ($p<0.001$). Subsequent investigations by Michael and Halliday (1971) concluded that the components responsible for the reversal in polarity originated in surface dipole sheets in the extrastriate region of the cortex. The polarity reversal was a result of electrodes above the inion facing opposite sides of the cortex representing the upper and lower fields. Dipole orientation has also been shown to rotate through 135° when comparing upper and lower field stimulation after pattern onset stimulation (Butler et al 1985).

The P100 following upper half field stimulation was found to be positioned more posteriorly when compared with the lower half field response, as has previously been demonstrated (Skrandies, Richter and Lehmann 1984), however in contrast to previous reports of a longer latency response from upper half field stimulation (Skrandies, Richter and Lehmann, Lehmann, Meles and Mir 1977, Lehmann and Skrandies 1979) no significant difference in the latency between the two half fields was demonstrated. It had been concluded that the differences of latency were related to differences in processing of the information by the upper half field.

No significant amplitude difference was demonstrated between the amplitudes of the P100 following lower and full field stimulation, the distribution of the response was also similar for the two stimuli. As a consequence the response after full field stimulation has been attributed to activation of the lower half field. Visual field loss in the upper half field may fail to be demonstrated in the VEP if the response is recorded from electrodes positioned laterally over the Oz line.

7.2.7iii Late Positivity.

On lower half field stimulation a late positive was observed in four subjects, the group mean map showed a posterior distribution. In contrast on upper half field stimulation a late positivity was recorded in eight subjects and the group mean map showed an anterior distribution. This later positivity was therefore ipsilongitudinal for the altitudinal fields, in contrast lateral half field stimulation produced a contralateral late positivity.

7.2.7iv N145.

N145 was found to be significantly greater following lower field stimulation, $p<0.05$ Student t test, there was however no significant effect on latency. This effect is therefore similar to that shown by the P100 peak. In addition the topographical distribution of this component is similar to that of the P100.

7.2.8 Similarities Between the Altitudinal and Lateral Half Fields.

The lateralisation of the early components was the same for all fields i.e. the early negativity was ipsilongitudinal/ipsilateral and the early positivity was maximal contralongitudinally/contralaterally. With succeeding components the laterality was dependant on whether the stimulus was positioned in the altitudinal or lateral half fields. The P100 was ipsilateral for the lateral half fields and contralongitudinal with the altitudinal fields, the converse was true for the later positivity. The late negativity appeared to have the same distribution as P100, for all fields. It appears that the responses from the four half fields behave with the same trends for the early components but for the later components the response is then similar between related half fields.

7.2.9 Summary:

No significant difference in latency was demonstrated between the upper and lower half field P100 response however, a significant reduction in amplitude on upper half field stimulation was shown. Stimulation of the upper and lower half field did reveal, in a number of subjects two positive peak responses, the earlier peak was located contralongitudinally with the later peak being maximally distributed ipsilongitudinally. The occurence of a later positive peak to upper half field stimulation has previously been reported (Edwards 1987) however, no attempt to ascertain the origin of this response has been made. The latency of this

later peak was too delayed to be a P100 response however the ipsilongitudinal distribution would correspond with that following lateral half field stimulation. A significant reduction in the amplitude of N145 was shown following upper field stimulation with no effect on latency, this was similar to that found with the P100 peak. In addition the topographical distributions of these two components were similar.

7.3 Latencies of the Pattern Electro-retinogram to Upper and Lower Half Field Stimulation

7.3.1 Introduction

The delay in the pattern reversal VEP upper half field response recorded by previous investigators has been attributed to inferior processing in the lower hemiretina (Lehmann and Skrandies 1979). Previous studies have suggested that activity in the interactions between the photoreceptors and retinal pigment epithelium is greatest following upper retinal stimulation.

The electroretinogram (ERG) reflects activity in the retina after visual stimulation, the morphology of the response depends on the stimulus used, as with the VEP the most common stimuli in use are flash (uniform light) and steady state and transient pattern reversal. The flash and pattern ERG (PERG) are thought to arise from independent generators, studies following optic nerve sectioning of the cat have shown that the flash ERG still remains while the only PERG response remaining is to low spatial frequency stimuli (Tobimatsu et al 1989). The PERG is therefore thought to comprise of two responses; a local luminance and a pattern specific response, large check stimulation produces a local luminance response. The transient pattern reversal PERG consists of a positivity occuring around 50ms (P50) followed by a negativity at around 95ms (N95), see fig.7.5. A negativity preceeding the P50 may also be recorded in some patients (N35) (Holder 1987).

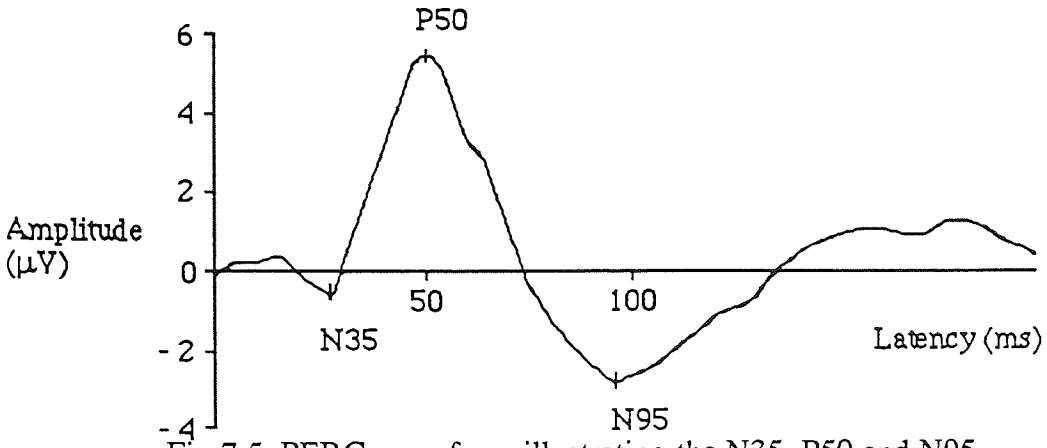


Fig.7.5. PERG waveform illustrating the N35, P50 and N95 components

The transient PERG is thought to be more sensitive in detecting retinal and optic nerve lesions (Kaufman and Celesia 1985), following optic nerve section in the cat

the steady state response was abolished whilst the transient response was only reduced in amplitude (Maffei and Florentini 1990). Wide debate however, exists over the site of generation of the PERG. A study of human patient groups (Holder 1987) has lead to the proposal that the N95 reflects optic nerve functioning and as a result is generated in the proximal retina, presumably the ganglion cells. Macular or general retinal dysfunction resulted in a reduction of the P50 component, this lead to the conclusion that the P50 was generated in distal retina layers. In some cases the PERG was completely absent with retinal dysfunction but never with optic nerve disease. Preservation of the PERG with optic atrophy may indicate that the PERG must have a preganglionic origin or that the cell degeneration may not be complete (Kaufman and Celesia 1985). In addition the N95 component shows spatial tuning thus further indicating ganglion cell generation, the P50 component amplitude increases with check size suggesting a more luminance related response (Holder 1990). Contrary to Holders findings the PERG was found to be reduced to just an N95 component with low spatial frequency stimuli and no response was observed to high spatial frequency stimuli after resectioning of the optic nerve in the cat. This would suggest that the P50 component is more susceptible to optic nerve sectioning and therefore more dependent on the ganglion cells for generation (Tobimatsu et al 1989). The PERG has been shown to be absent after traumatic section of the human optic nerve further suggesting that the whole response is a result of ganglion cell activity (Dawson et al 1982). However, as we are not yet certain of the widespread consequences of optic nerve dysfunction on the balance of the intricate retinal structure it is not possible to precisely pinpoint the retinal sources of the PERG (Apkarian and Spekreijse 1990). The PERG may reflect passive and not direct ganglion cell activity (Harrison et al 1987, Maffei and Florentini 1990) it is obvious however, that the integrity of the ganglion cell is essential for the production of the PERG.

The PERG was first developed to alleviate problems of stray light in the flash response (Riggs et al 1964), it was therefore thought more practical to investigate the upper and lower retinal areas with the PERG as compared to the flash ERG.

7.3.2 Method

Four consenting subjects were used, age range of 25-29, with no ophthalmological deficits and visual acuities of 6/6 or better with optical correction if required. The PERG was recorded from DTL fibre electrodes placed along the lower limbus from outer to inner canthi. The reference electrode was placed at the outer canthi and the earth was placed at Fpz. The response was recorded from both eyes

simultaneously, 256 responses were averaged with a reversal rate of 2Hz. The stimulus was a 27' black and white checkerboard with a circular full field size of 20°, responses were recorded from full, upper and lower half field stimulation. A TV monitor was used, to eliminate the effect of the frame reversal rate the same area was blanked off for both upper and lower half field stimulation, the monitor was inverted for the opposite field.

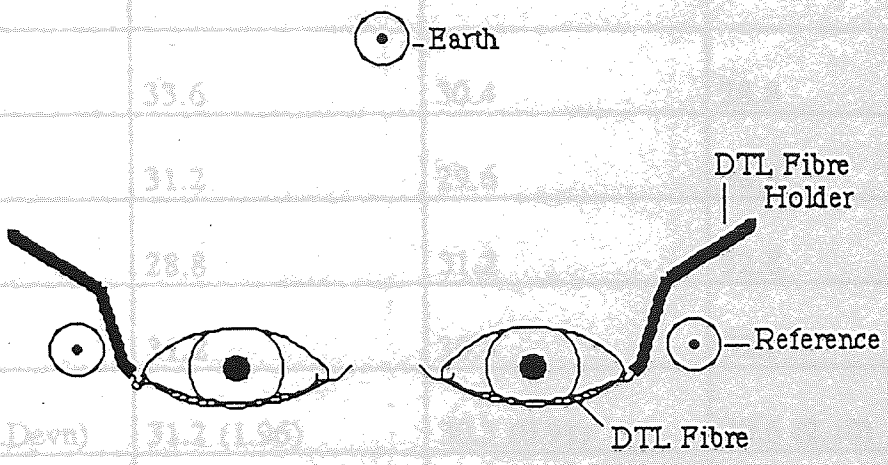


Fig.7.6 Electrode placement for recording the PERG.

7.3.3 Results

The tables show the latencies and peak to peak amplitudes of the N35, P50 and N95 components to full, upper and lower half field stimulation.

Component/Subject	Full Field (ms)	Lo Half Field(ms)	Up Half Field(ms)
N35			
RD	33.6	30.4	28.8
EW	31.2	29.6	28.8
CN	28.8	31.2	32.8
VT	31.2	30.4	32.0
Mean(Std .Devn)	31.2 (1.96)	30.3 (0.91)	30.6 (2.10)
P50			
RD	56.8	55.2	52.0
EW	56.8	56.0	56.0
CN	52.8	52.8	50.4
VT	54.4	52.8	53.6
Mean (Std .Devn)	55.2 (1.96)	54.2 (1.65)	53.0 (2.39)
N95			
RD	89.6	88.8	89.6
EW	98.4	94.4	96.8
CN	96.0	97.6	96.0
VT	96.0	92.0	94.4
Mean (Std .Devn)	95 (3.71)	93.2 (3.72)	94.2 (3.22)

Table 7.5 Latencies of the Major Components Recorded from the Right Eye.

Component/Subject	Full Field (μV)	Lo Half Field(μV)	Up Half Field(μV)
N35-P50			
RD	4.64	2.36	1.84
EW	4.96	3.28	3.40
CN	4.68	2.80	2.40
VT	4.26	2.24	2.16
Mean (Std .Devn)	4.64 (0.29)	2.67 (0.47)	2.45 (0.67)
P50-N95			
RD	-8.52	-4.32	-4.40
EW	-7.64	-3.96	-3.56
CN	-5.36	-3.44	-2.64
VT	-5.22	-2.32	-2.60
Mean (Std .Devn)	-6.69 (1.65)	-3.51 (0.87)	-3.30 (0.86)

Table 7.6 Peak to Peak Amplitudes of the Major Components in the Responses from the Right Eye.

Two factor analysis of variance in split plots was performed on the data to investigate the effect of both field size and stimulated eye. No effect of left versus right eye was demonstrated, results are therefore only shown from the right eye. A significant amplitude effect was found for the both P50 and N95 components, the full field response was significantly greater than both the upper and lower half field responses ($F=90.78$ (P50), 57.86 (N95) 2,12 $p<0.001$). No significant difference was demonstrated between the response amplitudes after upper and lower half field stimulation for either the P50 or N95 components. In addition no effect of field was demonstrated on the latencies of the N35 and N95, a significant effect was however demonstrated on the P50, the upper half field response was shown to be earlier than the full field response ($F=8.8$, 1,6 $p<0.05$). No significant difference

was demonstrated between the latencies of the P50 to lower and upper half field stimulation.

7.3.4 Discussion

No significant effect of field was demonstrated on either the latency or amplitude of the N95 component of the PERG, this may suggest that any latency difference previously shown in the VEP was not the result of ganglion cell activity. The only significant latency effect found was on the upper half field P50 component, this was significantly earlier and not later than the full field response, this latency shift only just reached significance in the $p<0.05$ range. A significant difference between subjects was found for the latencies of both the P50 and N95 components ($F=19.84$ (P50), 64.99 (N95), 3,12 $p<0.01$)

It may be expected that the full field amplitude would be greater than the half field responses as the area of field stimulated has increased, the PERG amplitude has previously been shown to increase with increasing field size (Sokol and Bloom 1976). No significant amplitude difference for either the P50 or N95 component was observed between upper and lower half field stimulation. This would suggest that the processes generating the PERG are being activated to a similar extent following upper and lower half field stimulation. The amplitude difference between the upper and lower half field VEP response is therefore most probably the result of the ventral position of the upper half field projections on the striate cortex.

7.4 The Effect of Central and Peripheral Stimulation on the Topographical Distribution of the Upper and Lower Half Field Response.

7.4.1 Introduction

In contrast to the ipsilateral distribution of the major positivity following lateral half field stimulation, altitudinal field stimulation appears to produce a central or contralongitudinal distribution. Following the major positive peak a second positive peak was evident, this being more prominent on upper half field stimulation. The distribution of this peak was ipsilongitudinal to the stimulating half field. A late positive peak is recorded with lateral half field stimulation however this is maximal contralateral to the field.

These lateral half field ipsilateral and contralateral components have been shown to be preferentially stimulated with central and peripheral stimulation. Progressive occlusion of the central area of the lateral half field has been shown to increasingly attenuate the amplitude of the ipsilateral P100. Reduction of the field size has been shown to have relatively little effect on the amplitude of the P100, in contrast the contralateral components were attenuated (Blumhardt et al 1978). The P100 did however become more contralateral with a reduction in field size (Harding et al 1980). Other workers have reported no effect of check or field size on the ipsilateral and contralateral components, the major positive still being maximal over the ipsilateral hemisphere with a reduction in both and the contralateral negative response still present with a field size of 0-3° (Onofroj et al 1991). Occlusion of the central or peripheral parts of the half field could therefore lead to facilitation or reduction in amplitude of the components they were, however, never completely extinguished.

Central and peripheral altitudinal field stimulation was investigated to assess the effect on the two positive peaks recorded after upper half field stimulation and to assess whether they behaved in a similar pattern to any of the components in the lateral half field responses. The response was also recorded using an average reference to determine if the use of a frontal reference was producing any effect on the topographical distribution.

7.4.2 The Effect of Using an Average Reference on the Distribution of the Upper Half Field Response

7.4.2.i Introduction

A negativity has previously been shown to be maximally recorded over the vertex and Fz with a pattern reversal stimulus (Shih et al 1988). This negativity may amplify the P100 response if both are synchronous, if not then recording between the occiput and Fz may produce a broad poorly defined peak, a 'W' waveform or a shift in peak latency (Spitz 1986).

An average reference takes into account the activity on all channels and produces a mean value to which all channels are referred. When using an average reference recording electrodes should be spaced equally over the two hemispheres and over the whole scalp (Bertrand 1985). The electrodes were therefore positioned on a montage based on the 10-20 system.

7.4.2.ii Method

The electrodes were positioned at In1, Inz, In2, O3, O1, Oz, O2, O4, T5, P3, Pz, P4, T6, T3, C3, Cz, C4, T4, F3, Fz, F4. One male subject (age 29) was used for this study, giving a clear anterior and posterior distribution of the response to upper field stimulation.

7.4.2.iii Results

The upper half field response has two distinct maxima over the posterior and anterior region of the montage.

The distributions to upper half field stimulation are shown in fig.7.7. The anterior and posterior distribution were still evident on the maps. The global field plot of this subject with an upper half field stimulus shows two distinct peaks at the latencies of the two areas of maximum positivity. The amplitude of the global field power being 1.49 at 96ms and 1.48 at 130ms.

7.4.2.iv Discussion

The two distinct positive components in the upper half field response were recorded with both an average and common reference, this would suggest that the common reference was not affecting the response morphology. The two major

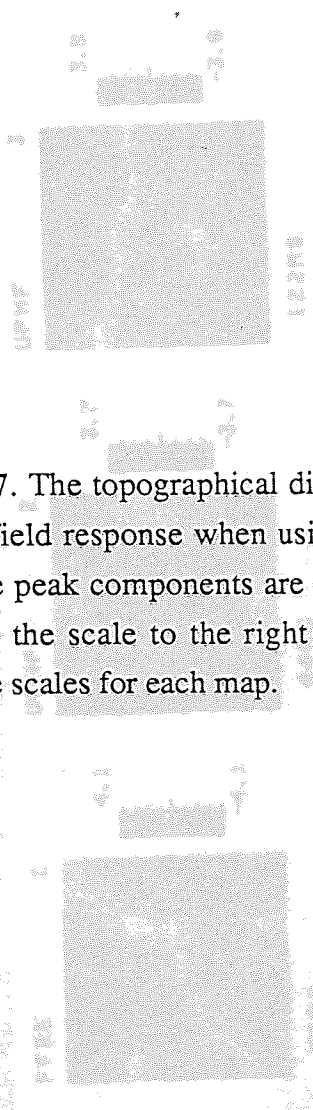


Figure 7.7. The topographical distribution of the full (FF) and upper half (UPHF) field response when using an average reference. The latency at which the peak components are mapped is shown. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

FFRE
1
4.1
-4.1
98MS
UPHF
2
3.7
-3.7
94MS
UPHF
3
3.8
-3.8
122MS

positivities recorded may therefore be classed as real responses to the upper half field stimulus.

7.4.3 Investigation of the VER After Central and Peripheral Stimulation of the Upper and Lower Field.

7.4.3.i Method.

The electrode placement was the same as that used in the first experiment; twenty electrodes attached to the occipital cortex, see fig 5.1.. The field stimuli were central 0-1°, 0-2° radius and peripheral 1-10° and 2-10° radius. The stimuli were projected independently to both the upper and lower half fields. Ten consenting subjects were used (age range 23-33) with no ophthalmological or neurological deficits and visual acuities of 6/6 or better with correction if necessary. Monocular right eye stimulation was used as previously. All stimulus parameters were the same as those used in the previous study i.e. check size of 27', reversal rate of 1Hz, luminance 1050 cd/m^2 and contrast of 80%

7.4.3.ii Results

Group mean waveforms, see figures 7.8 - 7.11, of the different stimulus presentations were produced from the individual responses of the ten subjects, the topographical distribution of the successive peaks were then mapped, see figures 7.12 - 7.15.

	Lo.HF	Lo.P 1-10°	Lo.P 2-10°	Lo.C 2°	Lo.C 1°
N75	-2.77(1.23)	-1.61(1.14)	-1.48(0.90)	-0.53(0.30)	-1.02(0.50)
P100	6.75(1.55)	5.95(1.74)	4.86(0.78)	4.39(1.12)	2.73(0.72)
LATEP	3.13(2.12)	2.68(0)	3.46(0.60)	2.69(0.91)	2.55(0.39)
N145	-5.26(2.51)	-4.8(2.86)	-4.77(2.34)	-3.5(2.17)	-3.05(1.68)

Table 7.7 The Mean Amplitudes and Standard Deviation, in brackets, of the Major Peaks after Lower Half Field, Peripheral and Central Stimulation.

	Up HF	Up P 1-10°	Up.P 2-10°	Up.C 2°	Up.C 1°
N75	-3.05(3.05)	-1.57(1.54)	-2.24(1.71)	-1.64(0.68)	-1.08(0.84)
P100	3.62(1.19)	5.09(1.57)	4.07(0.98)	3.80(1.55)	3.74(1.28)
LATEP	3.91(0.95)	2.83(0)	4.09(1.22)	3.74(1.26)	2.94(0)
N145	-2.95(1.47)	-2.24(1.25)	-2.66(1.50)	-2.86(0.16)	-2.49(0.62)

Table 7.8 The Mean Amplitudes and Standard Deviation, in brackets, of the Major Peaks after Upper Half Field, Peripheral and Central Stimulation.

	Lo.HF	Lo.P1-10°	Lo.P2-10°	Lo C 2°	Lo.C 1°
N75	74.57(3.78)	75.25(5.21)	72.89(6.17)	77(3.46)	73.5(9.15)
P100	97.8(3.46)	97.4(2.67)	98(5.89)	98.6(8.27)	99.6(12.36)
LATEP	125.3(14.47)	126(0)	127(18.38)	125(12.73)	124(5.66)
N145	140.8(9.72)	141(14.52)	146.4(12.89)	138.4(10.52)	147(14.07)
	Up.HF	Up.P1-10°	Up.P2-10°	Up C 2°	Up.C 1°
N75	76(1.51)	74.85(1.95)	72.29(3.55)	80.28(5.22)	77.33(8.07)
P100	100.6(7.06)	101.4(8.74)	98.6(8.79)	111(10.35)	112.8(10.76)
LATEP	123.2(8.19)	122(0)	121(7.07)	117.33(4.62)	110(0)
N145	156.4(17.81)	147.25(13.4)	150.8(13.26)	166(5.29)	157.33(7.97)

Table 7.9 The Mean Latencies and Standard Deviation, in brackets, of the Major Peaks after Lower and Upper Half Field, Peripheral and Central Stimulation.

	N75	P100	P120	N145
Lower Field	6 subjects	10 subjects	3 subjects	10 subjects
Lo Per 1-10	7 subjects	10 subjects	----------	9 subjects
Lo Per 2-10	8 subjects	10 subjects	----------	10 subjects
Lo Cen 2	6 subjects	10 subjects	3 subjects	8 subjects
Lo Cen 1	5 subjects	9 subjects	2 subjects	7 subjects
Upper Field	8 subjects	10 subjects	5 subjects	10 subjects
Up Per 1-10	8 subjects	9 subjects	1 subject	6 subjects
Up Per 2-10	7 subjects	9 subjects	2 subjects	6 subjects
Up Cen 2	5 subjects	9 subjects	1 subject	4 subjects
Up Cen 1	4 subjects	9 subjects		6 subjects

Table 7.10 The Number of Subjects Producing the Major Components of The Central and Peripheral Upper and Lower Half Field Response

7.4.3.iii The Effect of Central Stimulation and Occlusion on the Latencies and Amplitudes of the Major Peaks.

The following statistical analyses of the data were all performed using the Student t test.

7.4.3.iiia Upper Field

N75: The amplitude of the early negativity was not significantly affected when either the field was reduced or the central field occluded. The latency was however significantly reduced following upper peripheral 2-10° stimulation ($p<0.05$).

P100: An effect on both the amplitude and latency of this component was shown. The amplitude was significantly reduced following central 1° occlusion ($p<0.05$), this significant reduction was however not evident with 2° occlusion. In addition the amplitude was not significantly affected by a reduction in stimulating field size,

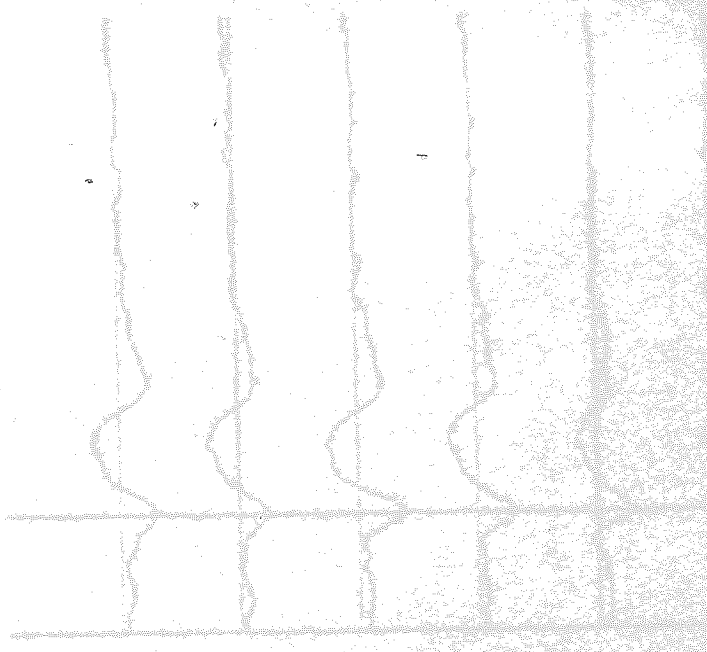
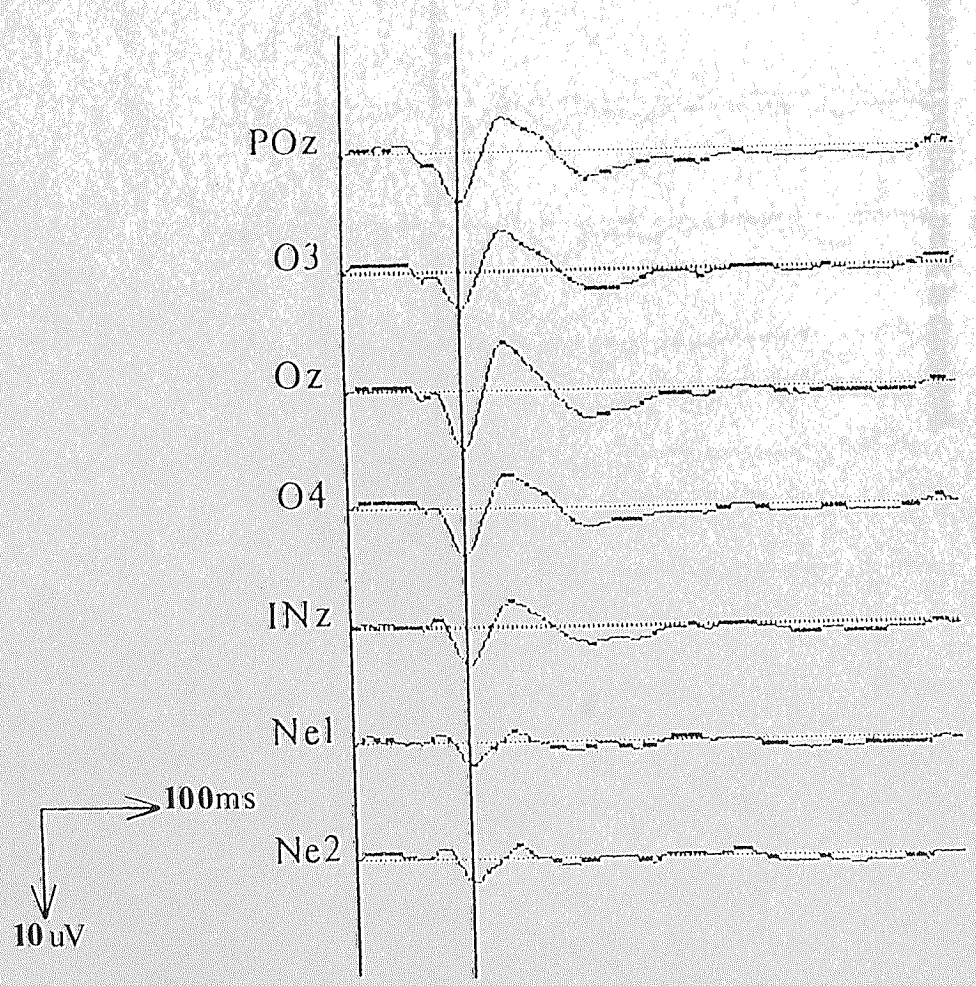
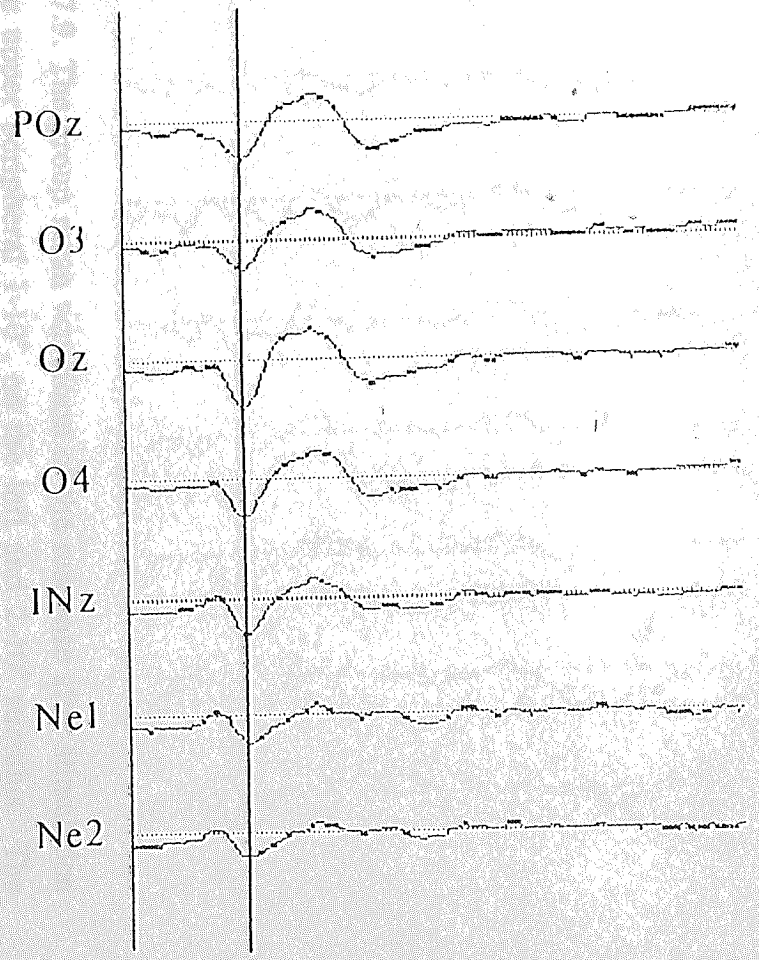


Figure 7.8. The group mean waveform of the response after stimulation with the lower peripheral 1-10°, on the left and 2-10° stimulus. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.


POz
O3
Oz
O4
INz
Ne1
Ne2
100ms
10uV


POz
O3
Oz
O4
INz
Ne1
Ne2

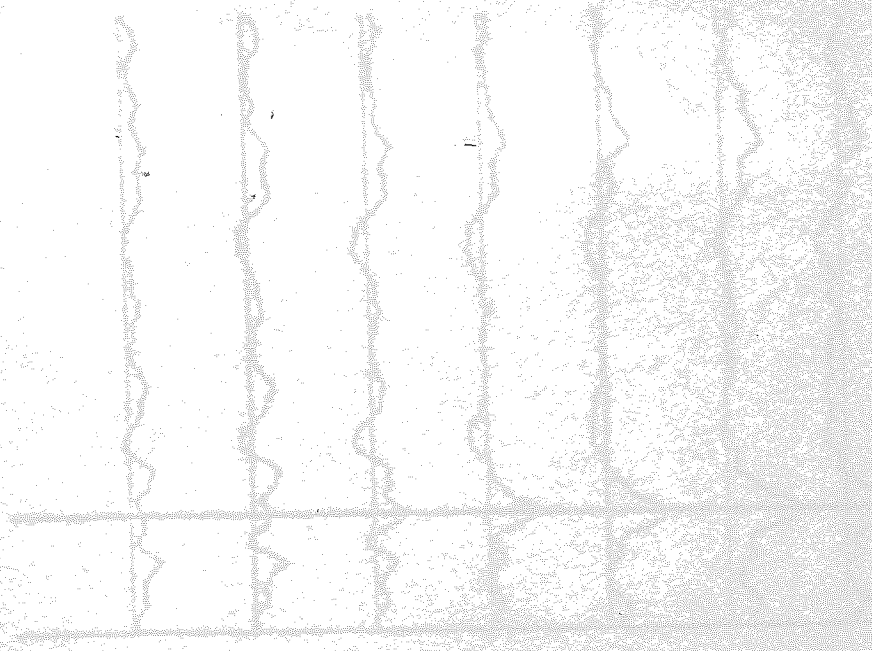
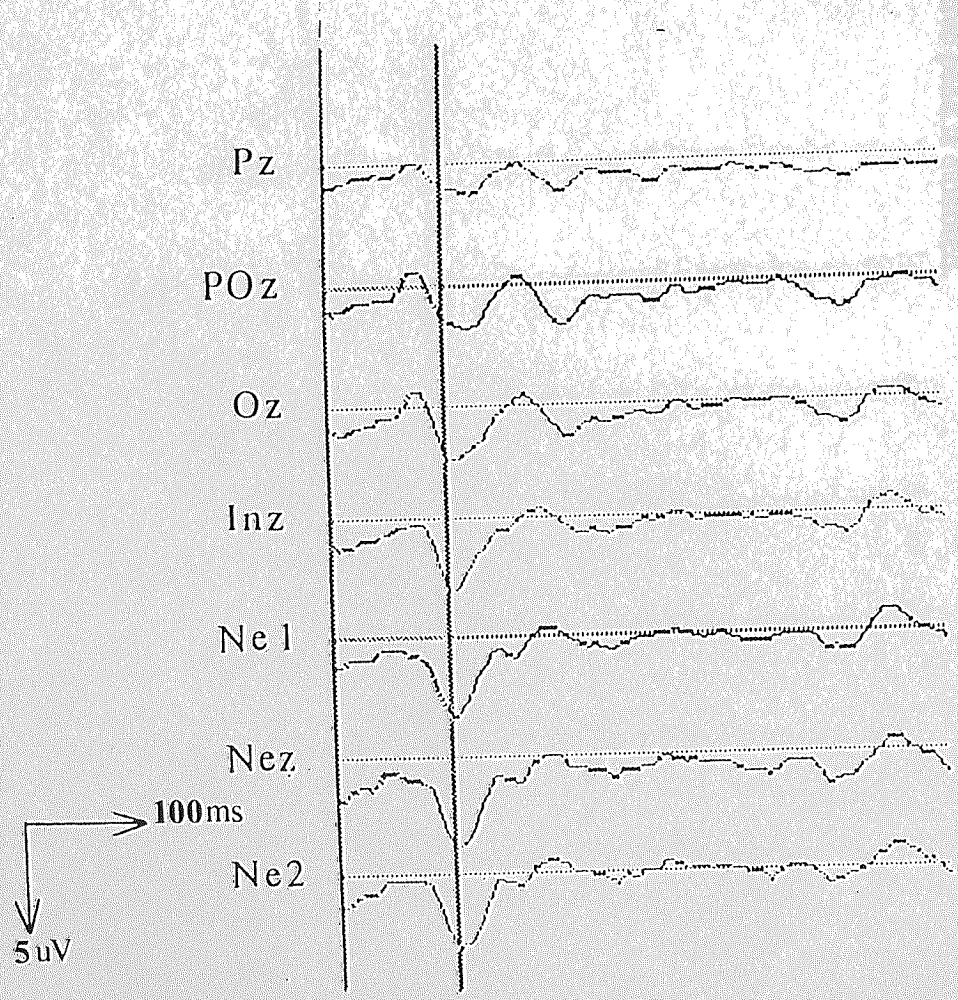
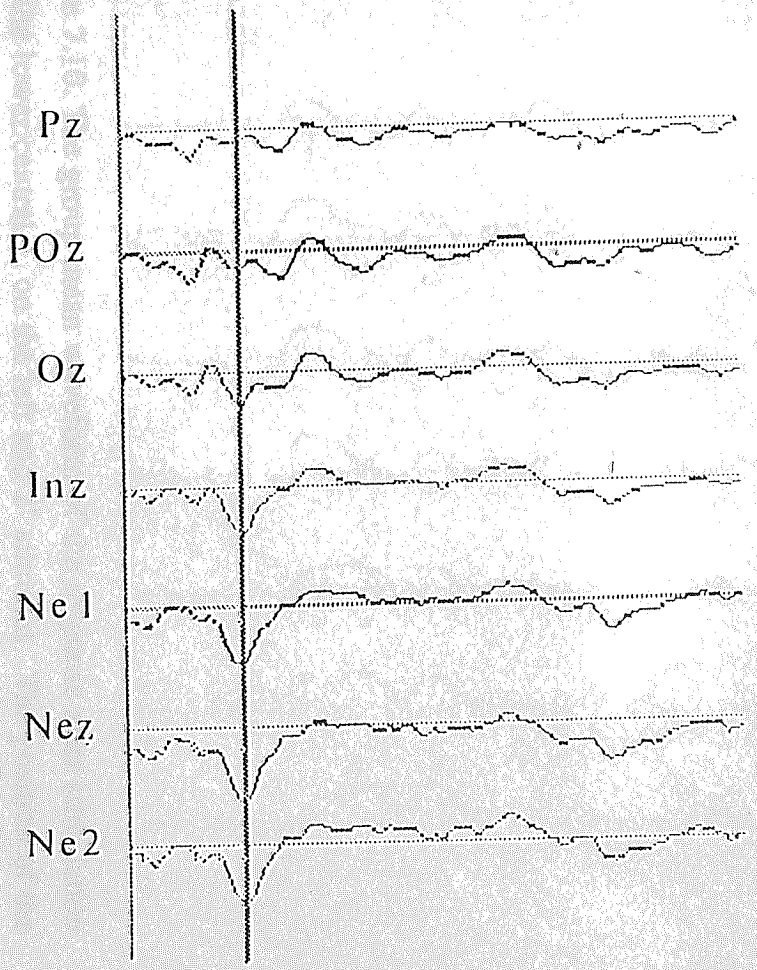


Figure 7.9. The group mean waveform of the response after stimulation with the upper peripheral 1-10°, on the left and 2-10° stimulus. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.


Pz
POz
Oz
Inz
Ne1
Nez
Ne2
100ms
5uV


Pz
POz
Oz
Inz
Ne1
Nez
Ne2

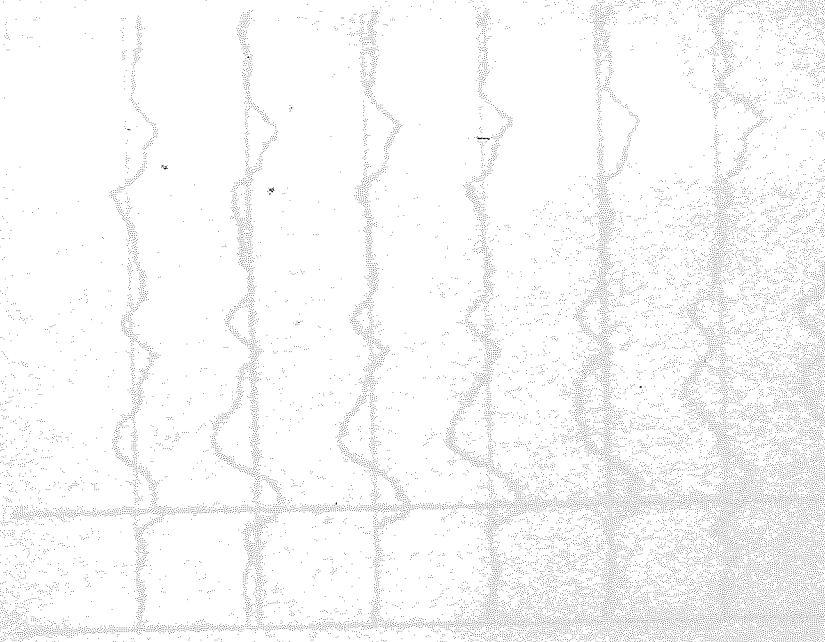


Figure 7.10. The group mean waveform of the response after stimulation with the lower central 1, on the left and 2° stimulus. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.

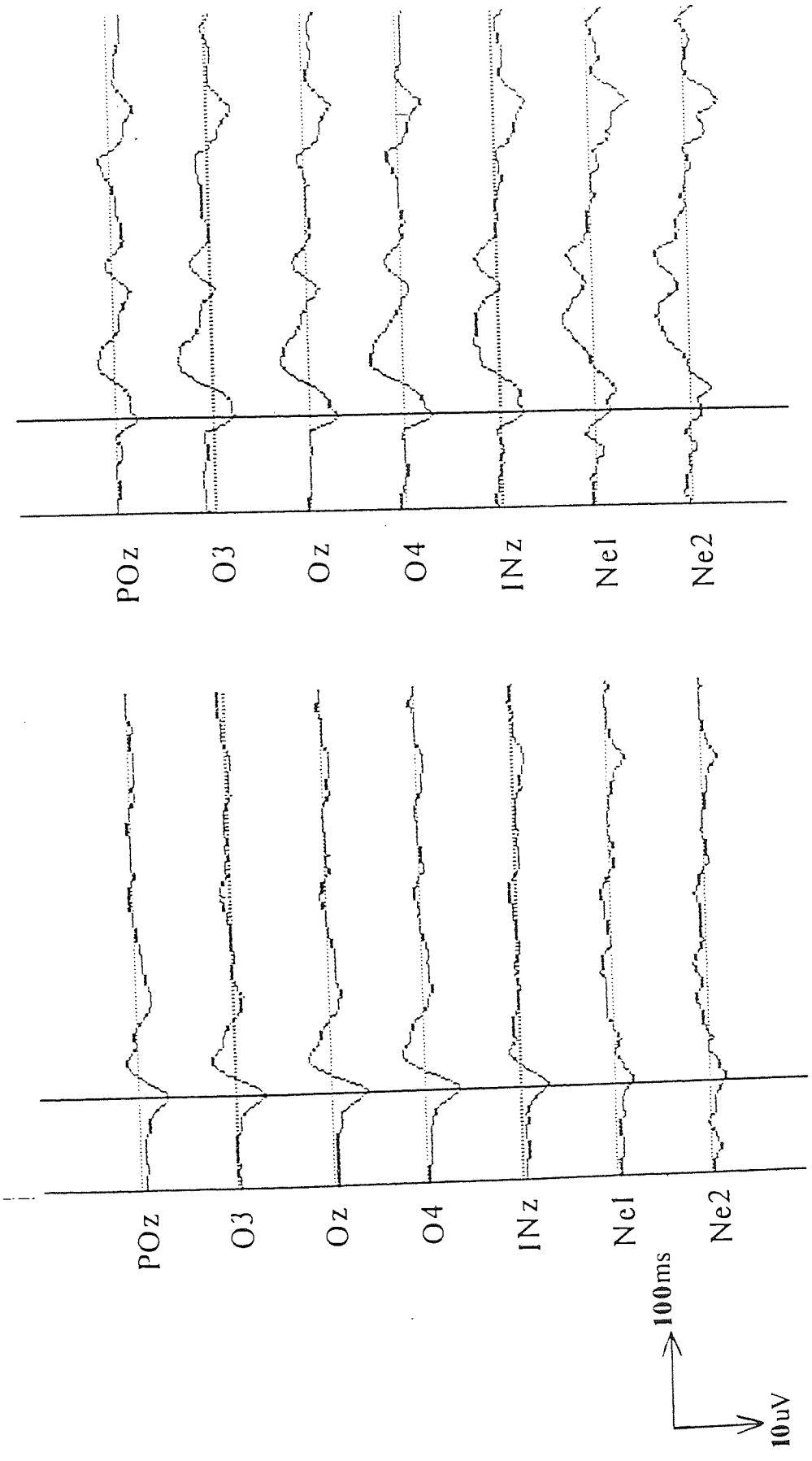
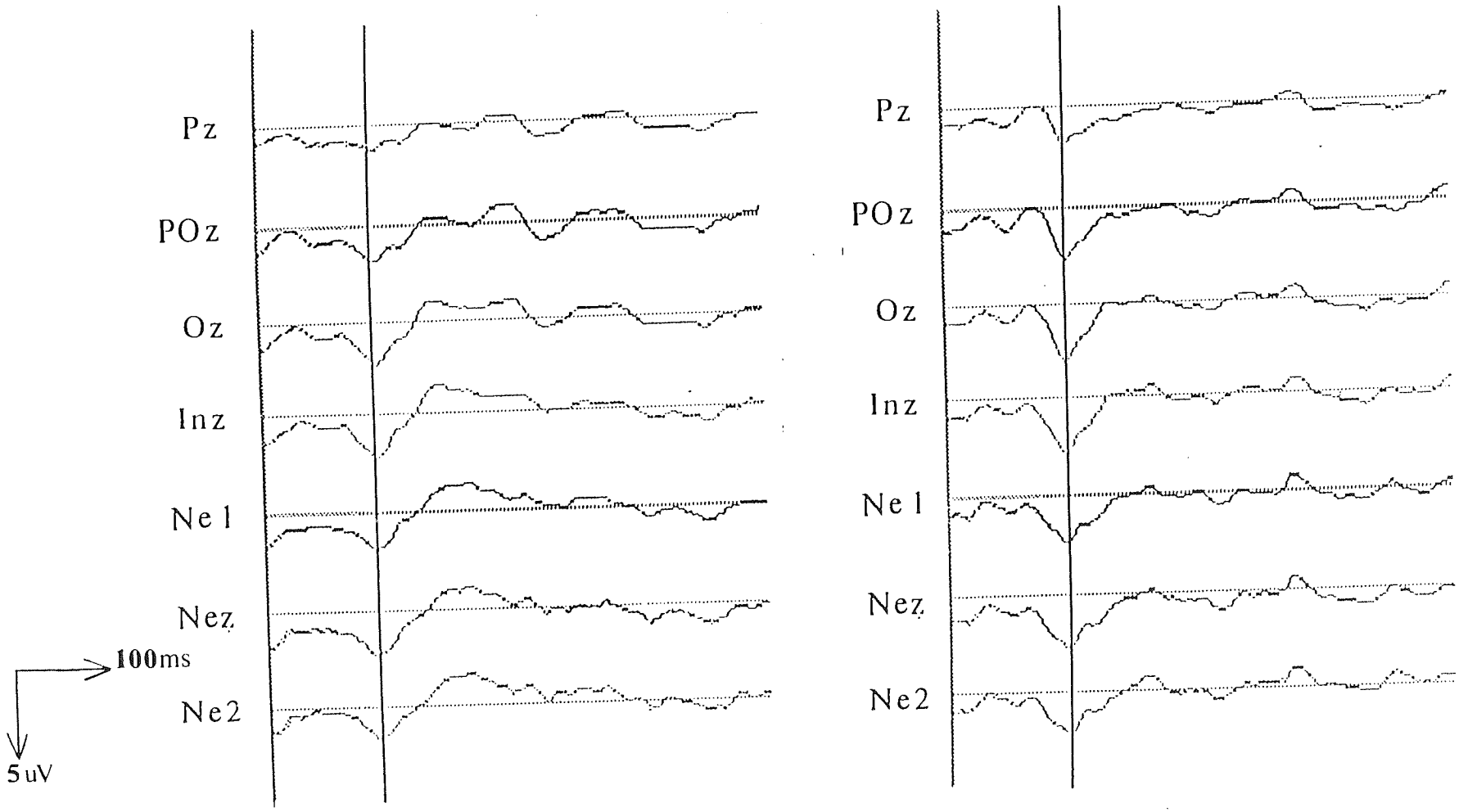

POz
O3
Oz
O4
INz
Ne1
Ne2
POz
O3
Oz
O4
INz
Ne1
Ne2
100ms
10uV

Figure 7.11. The group mean waveform of the response after stimulation with the upper central 1°, on the left and 2° stimulus. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.


Pz
POz
Oz
Inz
Ne1
Nez
Ne2
100ms
5uV
Pz
POz
Oz
Inz
Ne1
Nez
Ne2

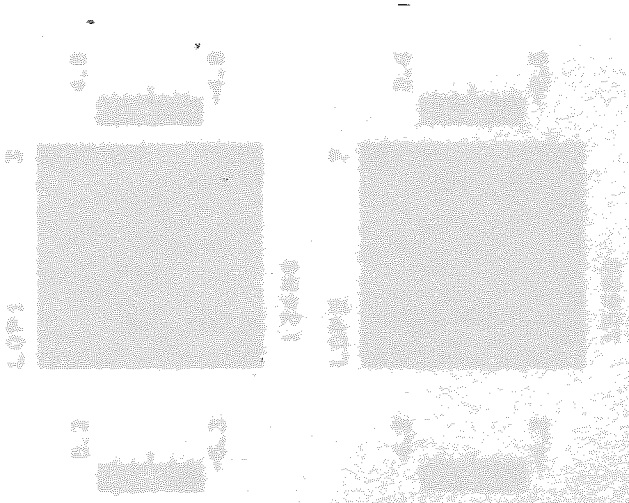
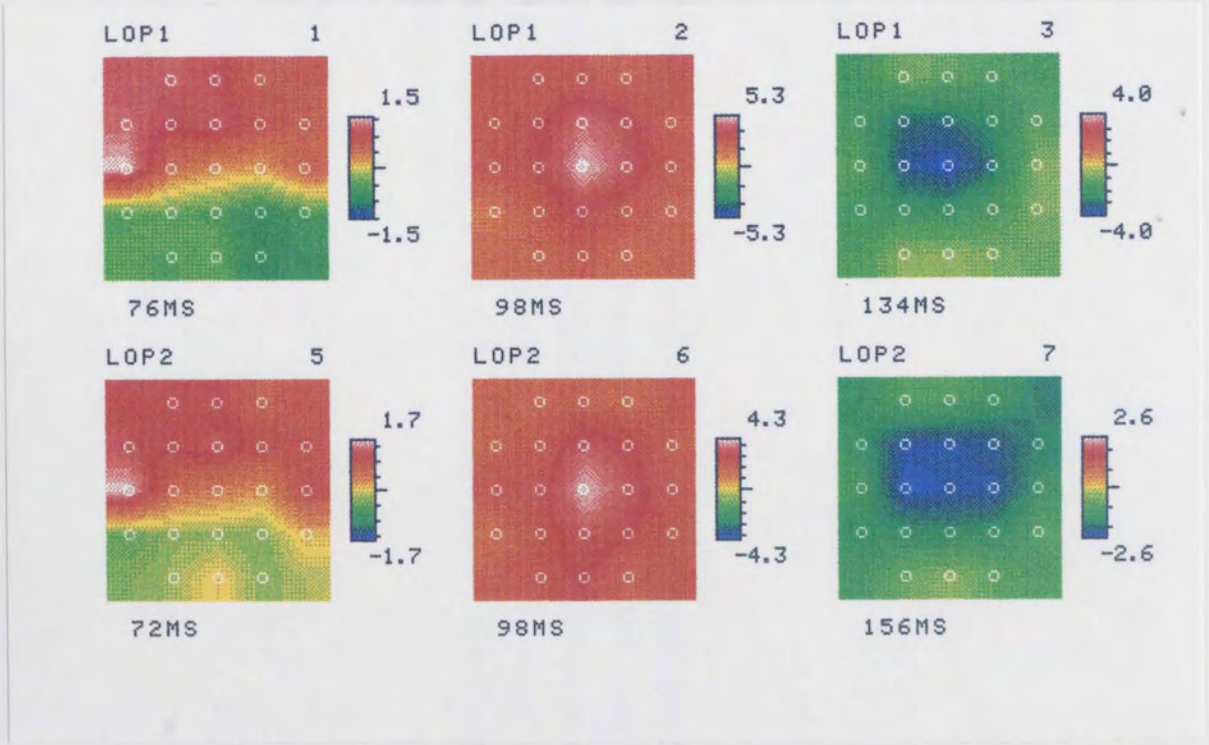


Figure 7.12. The topographical distribution of the major components in the group mean waveform following lower peripheral 1-10° and 2-10° stimulation. Key; LOHF = lower half field, LOP1 = lower peripheral 1-10° and LOP2 = lower peripheral 2-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.


LOP1 1
1.5
-1.5
76MS
LOP1 2
5.3
-5.3
98MS
LOP1 3
4.0
-4.0
134MS
LOP2 5
1.7
-1.7
72MS
LOP2 6
4.3
-4.3
98MS
LOP2 7
2.6
-2.6
156MS

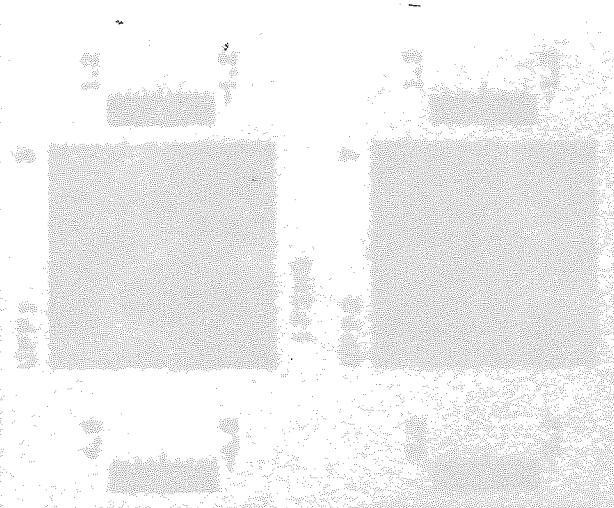
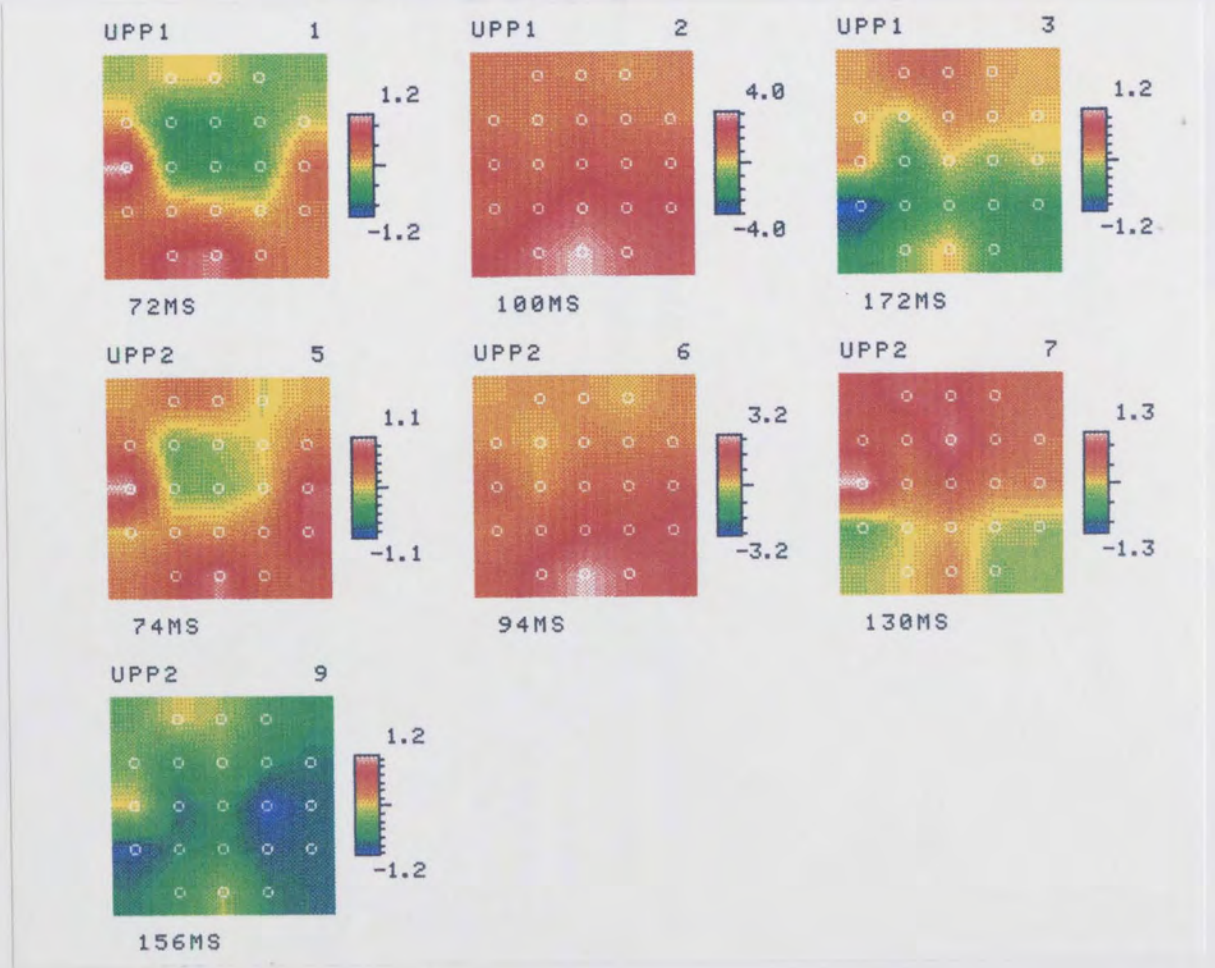


Figure 7.13. The topographical distribution of the major components in the group mean waveform following upper peripheral 1-10° and 2-10° stimulation. Key; UPHF = upper half field, UPP1 = upper peripheral 1-10° and UPP2 = upper peripheral 2-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.


UPP1 1
1.2
-1.2
72MS
UPP1 2
4.0
-4.0
100MS
UPP1 3
1.2
-1.2
172MS
UPP2 5
1.1
-1.1
74MS
UPP2 6
3.2
-3.2
94MS
UPP2 7
1.3
-1.3
130MS
UPP2 9
1.2
-1.2
156MS

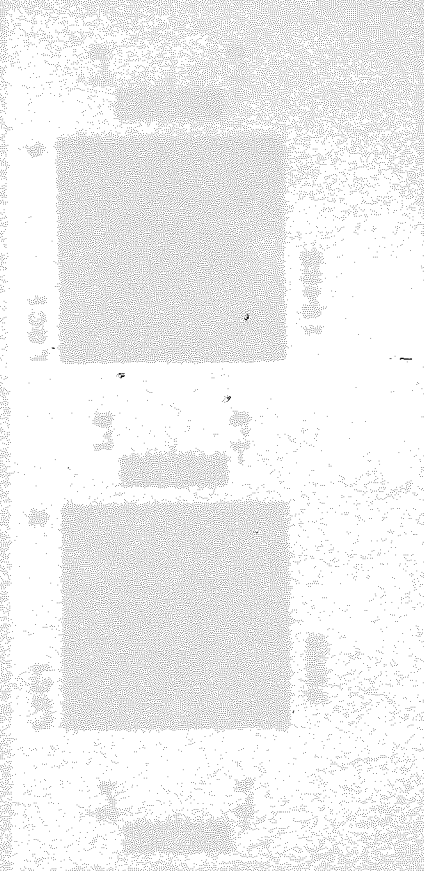


Figure 7.14. The topographical distribution of the major components in the group mean waveform following lower central 1° and 2° stimulation. Key; LOHF = lower half field, LOC2 = lower central 2° and LOC1 = lower central 1°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

LOC2
1
4.1
-4.1
96MS
LOC2
2
2.4
-2.4
132MS
LOC1
3
1.8
-1.8
96MS
LOC1
4
1.8
-1.8
154MS

Figure 7.15. The topographical distribution of the major components in the group mean waveform following upper central 1° and 2° stimulation. Key; UPHF = upper half field, UPC2 = upper central 2° and UPC1 = upper central 1°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

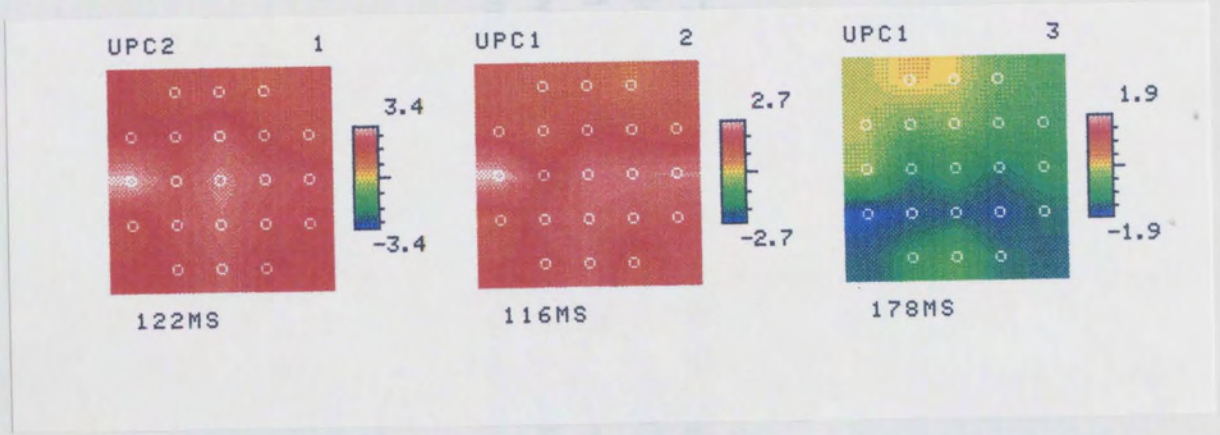

UPC2
1
3.4
-3.4
122MS
UPC1
2
2.7
-2.7
116MS
UPC1
3
1.9
-1.9
178MS

Figure 7.16. The topographical distribution from the group mean maps of the major early and late negativity and P100 following lower peripheral 1-10° and 2-10° stimulation. Key; LP1 = lower peripheral 1-10° and LP2 = lower peripheral 2-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

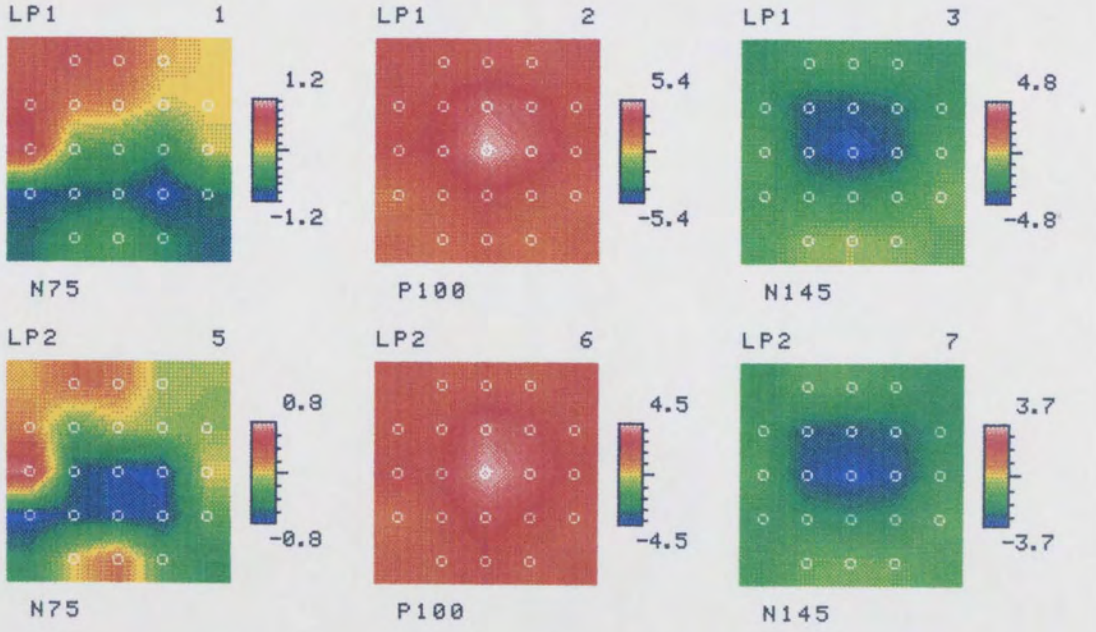

LP1
1
1.2
-1.2
N75
LP1
2
5.4
-5.4
P100
LP1
3
4.8
-4.8
N145
LP2
5
0.8
-0.8
N75
LP2
6
4.5
-4.5
P100
LP2
7
3.7
-3.7
N145

Figure 7.17. The topographical distribution from the group mean maps of the major early and late negativity and P100 following upper peripheral 1-10° and 2-10° stimulation. Key; UP1 = upper peripheral 1-10° and UP2 = upper peripheral 2-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

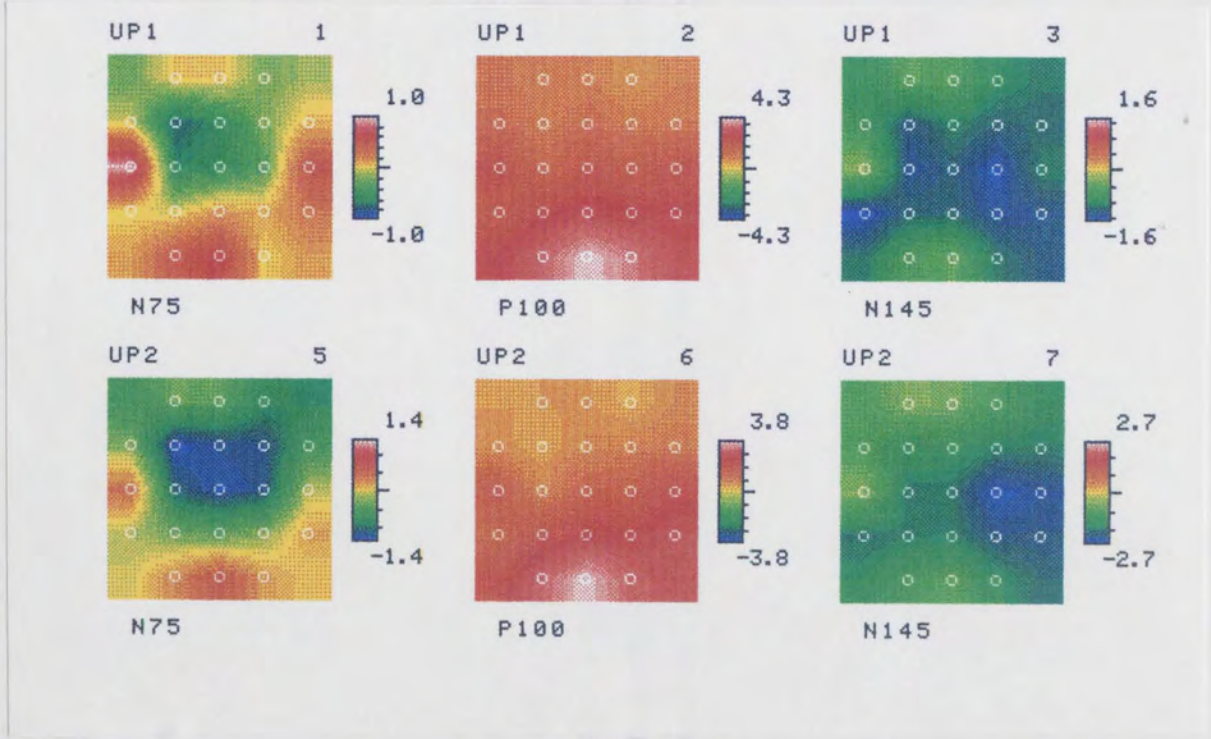

UP1
1
1.0
-1.0
N75
UP1
2
4.3
-4.3
P100
UP1
3
1.6
-1.6
N145
UP2
5
1.4
-1.4
N75
UP2
6
3.8
-3.8
P100
UP2
7
2.7
-2.7
N145

Figure 7.18. The topographical distribution from the group mean maps of the major early and late negativity and P100 following lower central 1° and 2° stimulation. Key; LC1 = lower central 1° and LC2 = lower central 2°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

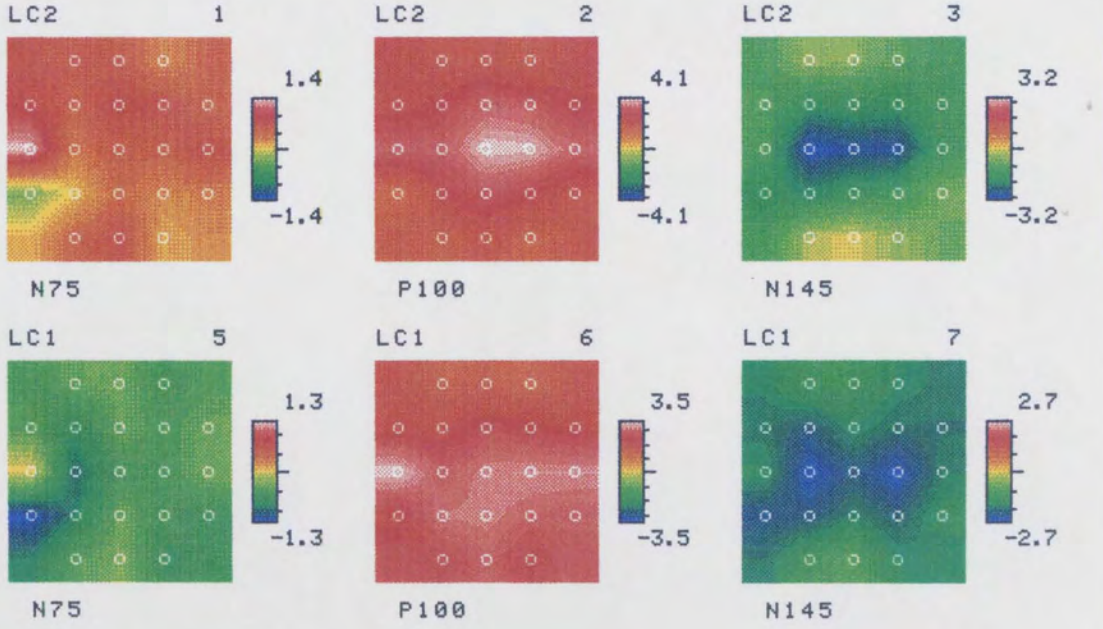
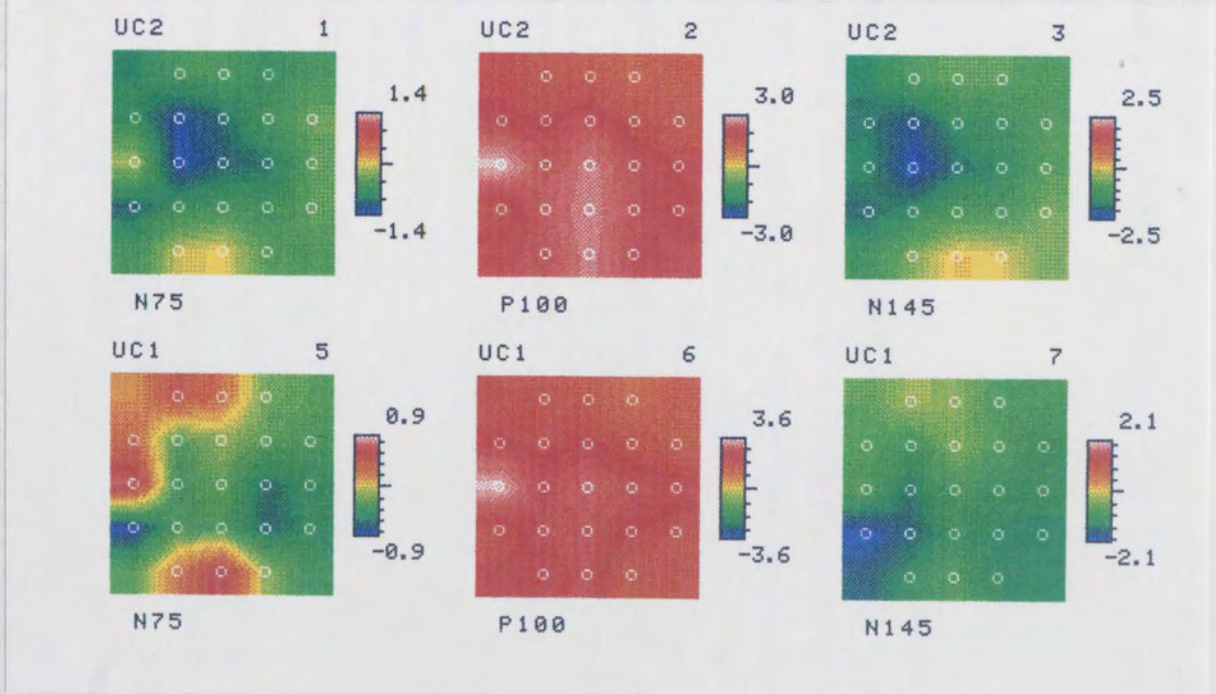

LC2
1
1.4
-1.4
N75
LC2
2
4.1
-4.1
P100
LC2
3
3.2
-3.2
N145
LC1
5
1.3
-1.3
N75
LC1
6
3.5
-3.5
P100
LC1
7
2.7
-2.7
N145

Figure 7.19. The topographical distribution from the group mean maps of the major early and late negativity and P100 following upper central 1° and 2° stimulation. Key; UC1 = upper central 1° and UC2 = upper central 2°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.


UC2
1
1.4
-1.4
N75
UC2
2
3.0
-3.0
P100
UC2
3
2.5
-2.5
N145
UC1
5
0.9
-0.9
N75
UC1
6
3.6
-3.6
P100
UC1
7
2.1
-2.1
N145

although a significant delay in the P100 was found with central 1° stimulation ($p<0.02$).

N145: There was no significant effect on the amplitude of this peak, the latency was however was significantly delayed following upper central 2° stimulation ($p<0.05$).

7.4.3.iiib Lower Field

N75: In contrast to the upper field responses the amplitude of N75 was significantly reduced following central 1° ($p<0.02$), 2° ($p<0.05$) and 2° occlusion ($p<0.05$). In addition the latency was significantly delayed with central 1° stimulation ($p<0.02$) although only three subjects demonstrated this peak with both stimuli.

P100: The amplitude was significantly reduced with all stimuli except central 1° occlusion (2° occlusion $p<0.01$, 2° field $p<0.001$, 1° field $p<0.01$).There was however no significant effect on latency.

N145: There was no significant effect on the latency of this peak and the amplitude was only significantly reduced following lower 2° stimulation ($p<0.02$).

7.4.3.iiic Upper versus Lower Field

There was no significant difference between either the latency or amplitude of the N75 peak in the upper and lower responses after peripheral and central stimulation. In contrast P100 was significantly later following both upper 2° and 1° stimulation (2° $p<0.02$, 1° $p<0.05$). Although P100 was significantly larger following lower half field stimulation there was no evidence of any significant amplitude difference between the upper and lower responses following peripheral and central stimulation. N145 was significantly greater with lower field and lower peripheral stimulation (half field $p<0.05$, 1° occlusion $p<0.05$, 2° occlusion $p<0.05$), there was no significant effect with the central fields. In addition N145 was significantly later following upper half ($p<0.01$) and upper central 2° ($p<0.01$) stimulation when compared with the corresponding lower field response.

7.4.3.iv Topographic Distributions fron the Group Mean Waveforms;

7.4.3.iva Upper Field Stimulation

The upper half field response showed an early negative maximal over the anterior region of the montage i.e. ipsilongitudinal to the stimulating field. This was followed by an ipsilongitudinal positivity maximal approximately 22ms later, occuring at the P100 latency. A low amplitude negativity then followed which was maximal ipsilongitudinally with a simultaneous positivity occuring on the edge of the montage. Stimulation of the upper 1-10° periphery resulted in a reduction in amplitude of the early negativity and the appearance of a simultaneous positivity over the posterior region, maximal over O3 and Nez. The amplitude of this positivity exceeded the negativity. This was followed by a positivity over the posterior montage at 100ms. A negativity was then recorded being maximal over the left of the montage. Further central occlusion produced a similar progression of peak components. The amplitude of the early negativity was again reduced but the positivity remained unchanged. The major positivity remained maximal over Nez at 94ms, a later low amplitude positivity was also recorded over the anterior region of the montage with a similar distribution as that recorded after full upper half field stimulation. A negativity was then recorded over the posterior lateral region.

No early negativity was evident on upper central 2° and 1° stimulation, the late negativity was also absent after upper central 2° stimulation. The only peak evident from the group waveform for upper central 2° was therefore a positivity maximal over O3 and occuring with a latency of 122ms. On upper central 1° stimulation the positivity was maximal over O3 at 116ms, this was followed by a negativity with the same distribution as the late negativity from upper half field stimulation.

7.4.3.ivb Lower Field Stimulation

On lower half field stimulation the early negativity was maximal over the posterior edge of the montage i.e. ipsilongitudinal to the stimulating field, a positivity also occured at the same latency, maximal over O3. This complex was followed by another positivity maximal over the centre of the montage occuring at a latency of 98ms. This was succeeded by a negativity with a similar central topographical distribution.

Central 1° occlusion appeared to reduce the amplitude of the early negative component with no effect on the distribution, simultaneously the amplitude of the positivity appeared to increase. These components were followed by a positivity with a similar distribution to lower half field stimulation, and then a negativity which was maximal over the centre of the montage. On lower peripheral 2-10° stimulation the amplitude of the early negativity was again reduced and the amplitude of the positivity again increased, the distribution remained similar to that of lower half field stimulation. The major positivity was central and was followed by a negativity over the centre of the montage with a slightly more diffuse distribution when compared with the lower half field.

On lower central 1° and 2° stimulation no early negativity was evident. With lower central 2° stimulation the major positivity remained maximal over Oz however the distribution was more diffuse compared with the lower half field. The following negativity had a similar distribution to the lower field response and appeared at approximately the same latency. The major positivity was more diffuse with lower central 1° stimulation but remained maximal over the Oz line. The negativity appeared to be split into two foci, being maximal over O1 and O2.

7.4.3.v Comparison of the Distributions From the Group Mean Maps and Maps from the Group Mean Waveforms.

Group mean maps were constructed from the major early and late negative and P100 components present in most subjects, see figures 7.16 - 7.19. The distribution of the major positivity was very similar for all stimulus presentations when the mean maps and group waveforms are compared. Differences in the distributions were however found with the major negative components.

Similar distributions in both analyses are observed when the central 1° was occluded in the upper and lower fields. In contrast the distribution of the N75 component was more central in the mean map when the central 2° was occluded in the lower field. Both N75 and N145 appear to be unaffected by the mode of analysis after upper peripheral 2-10° stimulation. An N75 component was shown in the mean maps for upper and lower central stimulation, this was however not present in the group mean waveforms. This component was larger with lower central 1° when compared with lower central 2° stimulation, in contrast the N75 amplitude decreased with a reduction in field for the upper field. An N145 component was also evident in the mean map after upper central 2° stimulation, this

was not present in the mean waveform. The N145 distribution appears to be similar in the mean and waveform maps on lower central stimulation. This component was slightly more lateralised over the left in the mean map after upper central 1° stimulation.

7.4.3.vi Morphological Variations Between Subjects

7.4.3.via P100

The positive response after lower central 1° stimulation in two subjects appeared to be split into two positive peaks by a negative deflection. In one subject these were approximately equal in magnitude with the earlier maxima over O3 and O4 and the second maximum over O4, the later peak was maximal over O3 for the second subject.

In a third subject there were two distinct peaks with lower central 2° stimulation with no negative deflection. The initial peak was maximal centrally over Oz whereas the second peak was maximal over O4. On lower central 1° stimulation only one peak was recorded this being maximal over O4. These peaks are vaguely (one subject) and definitely (two subjects) present in the lower half field and lower peripheral response, they appear however, to be enhanced with central stimulation. In two subjects the response from lower central 1° stimulation was earlier than the lower half field response (the response was of low amplitude in one subject) and was maximal over O4 in one subject and In1 in the other.

The lower peripheral distribution was very similar to lower half field stimulation in 8 subjects for lower 1-10° stimulation and in 6 subjects for lower 2-10° stimulation. In the remaining, the response was more anterior for 2 subjects on lower 1-10° stimulation and for 3 subjects on lower 2-10° stimulation.

The distribution from lower central 2° stimulation was similar to the lower half field distribution in 3 subjects, more lateralised in 6 and more posterior in 1. The distribution was however more lateralised in all subjects with lower central 1° stimulation.

7.4.3.vii Comparative distribution of the upper and lower central P100 distribution for all subjects

The distributions from lower and upper central 1° are similar in four subjects, being maximal over the lateral region of the montage, O3 and O4. In one subject the later

peak of the upper central 1° was similar to the early peak of the lower central 1° stimulus whereas in another the later peak of both upper and lower central stimulation produced similar distributions.

7.4.3.viii Comparison of the upper and lower half field responses

The N75 component was more pronounced with lower half field stimulation in four subjects. In two subjects however, the early negativity was larger with upper half field stimulation. The early peaks were not present with either upper or lower stimulation in two subjects. The later negative peak has a larger amplitude with lower half field stimulation in six of the subjects, whereas in three of the subjects the amplitudes are approximately the same with both stimuli.

7.4.4 Discussion

7.4.4.i Early Negativity (N75)

The early negativity was absent in the group waveform after central upper and lower 1° and 2° stimulation, this is probably a result of the small numbers of subjects producing the response and the topographical variation between the subjects. The amplitude also appears to reduce when stimulating the peripheral field in the mean waveforms, the amplitude however increased in the upper mean map with an increase in the area of occlusion. It may therefore be suggested that the early negativity can not be exclusively attributed to either central or peripheral stimulation.

7.4.4.ii Major Positivity (P100)

There appeared to be four different types of response to upper half field stimulation, these could be classified as follows;

T1; there is an NPN type response on all channels at the similar latencies, with the positivity being maximal over the posterior region of the montage, 3 subjects (MD,DS,CD) and one subject the positive is maximal over the anterior region of the montage.

T2; there is an NP response on all channels however the latencies vary, the posterior response occurs before the anterior. This is demonstrated in three subjects.(TS,MB,AS).

T3;NPN type response on all channels with the posterior response being earlier than the anterior. Two subjects show this type of response (RD,NP)

T4; NPN response over the posterior region of the montage and a negative over the anterior region (DM).

One previous study has investigated the central and peripheral distribution from quadrant stimuli straddling the vertical meridian (Michael and Halliday 1971).

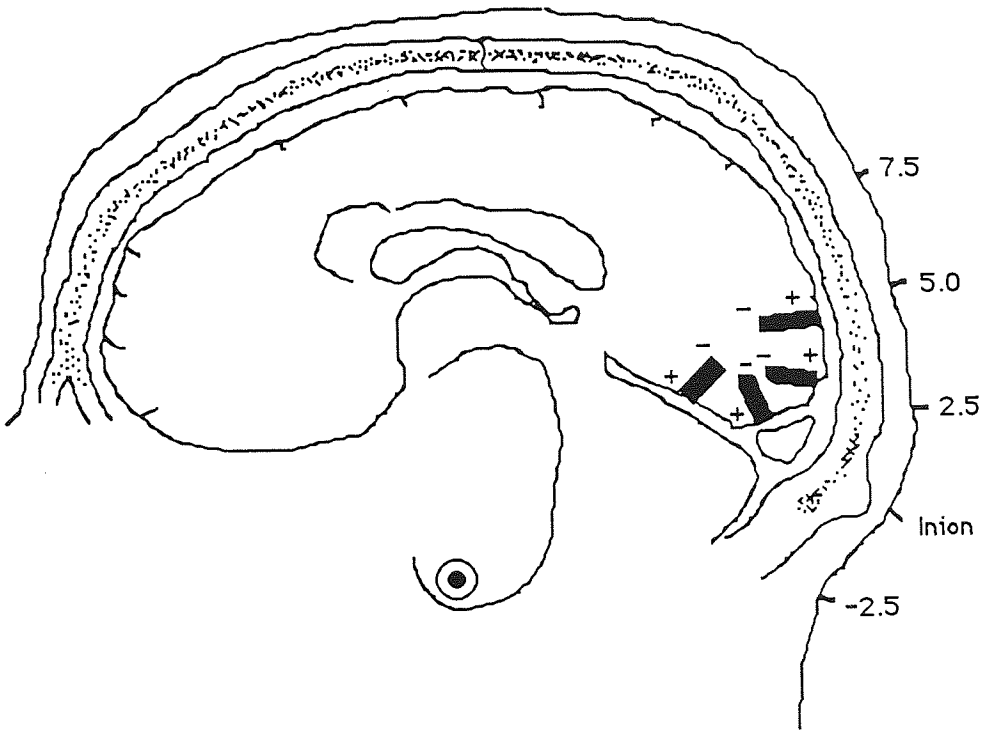


Fig 7.20. The position of the dipoles activated from upper and lower central and peripheral stimulation. The upper dipoles represent lower field stimulation, upper most peripheral, lower dipoles represent upper field stimulation, left hand dipole peripheral. (After Michael and Halliday 1971.)

Both upper and lower quadrants gave maxima located about 5-7.5cm above the inion with opposite polarity, the lower field evoking a positive response. On upper stimulation the central field gave a maximum positive below the inion, the peripheral field gave a maximum negative above the inion. Lower central and peripheral stimulation both produced a positive response, maximal above the inion. Dipole source models were constructed from the response distributions, illustrated in fig 7.20.

If the response is interpreted in terms of contralongitudinal and ipsilongitudinal components for the upper half field, peripheral stimulation appears to preferentially produce the contralongitudinal response however both responses were recorded in one subject. On central stimulation the ipsilongitudinal response appears to be maximally recorded. This is similar to the response from lateral half fields however the ipsilongitudinal response in this case is the P100.

The amplitude of the major positivity appears to reduce with central occlusion of the lower half field, this corresponds to the effect on the P100 after lateral half field stimulation. On central occlusion of the upper half field the amplitude of the posterior positive peak initially increased and then reduced after 2° occlusion.

7.4.4.iii Late Positivity (P120)

In one subject the later peak recorded on upper half field stimulation was also recorded in upper peripheral 1-10° and 2-10° stimulation however, on upper central 1° stimulation only the later peak was recorded, two peaks being recorded with upper central 2° stimulation.

In three of the subjects in which a late positive peak was evident on lower field stimulation, two positive peaks were present after the lower central stimulation. The later peak appeared to be exaggerated by central stimulation when compared with the earlier peak in all three. Although in one of the subjects this later peak was present on peripheral stimulation this was of a lower amplitude compared with the major positive peak.

This was also apparent on upper field stimulation, in one subject both the positive peaks were present with peripheral stimulation however on central stimulation the response was dominated by the later positivity.

After central stimulation the distribution of the response did not appear to exactly correspond to that of the later positives recorded after altitudinal field stimulation. Both distributions from upper and lower central stimulation were quite similar suggesting that maybe the sources were close. The distributions of the upper and lower later positivities were more anterior and posterior, respectively than the central response. This may suggest that the generators of these responses are more divergent.

7.4.4.iv Late Negativity (N145)

The late negativity appears to be more prominent on lower half field stimulation. On lower field stimulation the negativity remains maximal over the centre of the montage with all stimuli, except on lower central 1° stimulation the distribution becomes more lateral having two foci of activity over O1 and O2. This component also appears to remain quite consistent with upper field stimulation, being maximal over the posterior lateral regions of the montage. It is thought that this component arises from activation in the extrastriate areas, the component does however resemble that of the P100, both in topographical distribution and amplitude.

7.5 Summary

The early ipsilongitudinal negativity apppears to be largest with upper half field stimulation, in contrast the late negativity was of the greatest amplitude after lower half field stimulation. A late anterior positive was recorded in ten subjects after upper half field stimulation and was evident in the group average waveform, a late positivity was also recorded from four subjects on lower half field stimulation this was however not transposed onto the group average wave.

No significant difference was demonstrated between the latency of the major positivities following upper and lower half field stimulation, the amplitude of the upper half field response was however significantly reduced, this contradicts previous reports (Lehmann and Skrandies 1979 and Halliday et al 1977). Further investigation of the upper and lower half field response was performed using the PERG. No significant amplitude difference was demonstrated between the upper and lower field responses. A significant latency effect was however shown in the P50 component after upper half field stimulation, this component was significantly earlier than the full field response.

On central and peripheral stimulation of the upper half field the positivity over the posterior of the montage appears to be reduced on central stimulation, the major positivity being located over the midline of the montage. On peripheral stimulation the posterior positivity remained, in addition the anterior positivity was evident on the group average waveform after upper 2-10° stimulation. It cannot be stated conclusively that the posterior and anterior positivities are uniquely the response from either peripheral or central stimulation. The central upper field response does however, appear to be more prominent over the anterior region of the montage, this

may be a result of the source moving towards the occipital pole where the foveal projections are represented. The distribution of the later positive peaks and the central responses did not precisely correspond, this possibly suggests that the sources are not analogous.

A decrease in the stimulating field size reduces the amount of cortex active and concurrently the amount of opposing sources. This may assist in discriminating the components resulting from different cortical areas. Further investigation into component generation was therefore proposed by reducing the stimulating field size.

CHAPTER 8

The Topographic Distribution of the Pattern Reversal VEP to Quadrantic and Octant Stimulation

8.1 Introduction

The previous chapter concluded that the two positive peaks recorded after upper field stimulation may be related to central and peripheral processing. In addition to the differences in anatomical location of the central and peripheral projections there are also differences with respect to the location of the stimulating field. If the response is presumed to originate in the striate cortex then a large proportion of the cortex activated by a full field stimulus may act in opposition. As a consequence further reduction in the size of the stimulating field may lead to an increase in the localisational accuracy of the area of activation producing the response due to the reduced amount of opposing cortex stimulated. Few reports on the distribution of the response after octant stimulation with a pattern reversal stimulus are found in the literature (Halliday et al 1969 and Halliday et al 1977).

8.2 Quadrant Stimulation

8.2.1 Method

Twelve subjects were used (age range 22-55) six male, six female. All subjects had 6/6 visual acuity, with correction if necessary and no ophthalmological or neurological deficits. Twenty one silver-silver chloride electrodes were placed over the occipital area. Before the electrodes were positioned the scalp surface was gently abraded with Omniprep™. The electrodes were then attached with blenderm™ tape, or glue if required and the electrode impedence was maintained below 5KΩ. The reference electrode was positioned at Fz. All stimulation was monocular, right eye only. The stimulus set up was the same as that used previously in chapters 5 and 7. Fifty responses were recorded for each stimulus presentation on a Biologic Brain Atlas III mapping system. The check size used was fixed at 27' for all the stimuli, the quadrant radius was 10°. The potential distribution is presented as maps at the position of the major peaks in the response.

8.2.2 Results

Group mean waveforms and group mean maps were constructed from the data. Figures 8.1 and 8.2 show the group mean waveforms of the lower quadrant responses, the topographical distribution of the major components are illustrated in fig.8.3. The upper quadrant group mean waveforms are shown in figures 8.4 and 8.5, the topographical distribution of the major components are shown in fig.8.6. Group mean maps of the components were also constructed, these are shown in figures 8.7-8.10. Tables 8.1 and 8.2 illustrate the number of subjects producing the components of the response and the location of the maxima of these components on the maps.

	N75	P80	P100	N105	P120	N145
RUPQ	11 subjects	7 subjects	12 subjects	1 subject	8 subjects	10 subjects
LUPQ	8 subjects	5 subjects	12 subjects	1 subject	8 subjects	11 subjects
RLOQ	10 subjects	7 subjects	12 subjects	----------	5 subjects	12 subjects
LLOQ	7 subjects	5 subjects	12 subjects	1 subject	6 subjects	9 subjects

Table 8.1 The Number of Subjects Producing the Different Components of the Response

	N75	P80	P100	N105	P120	N145
RUPQ	I, A	C	I, P	C	C	I, P
LUPQ	CE, A	C	I, P	C	C	I
RLOQ		C, CE	I, CE		C, P	CE
LLOQ	I, P	C, CE	I, CE	C	CE, P	CE

Table 8.2 The Location of the Maximum Potential From the Group Mean Maps. Key A = anterior, CE = central, C = contralateral, I = ipsilateral and P = posterior.

Field	R Up Quad	L Up Quad	R Lo Quad	L Lo Quad
N75	75.2 (6.68)	75.56 (6.31)	72.89 (5.01)	75.0 (4.86)
P80	88.25 (8.51)	88.8 (6.72)	84.4 (3.85)	82.5 (5.93)
P100	104 (6.49)	102.5 (6.78)	99.6 (6.63)	101.5 (6.67)
N105	108(0)	100(0)		100(0)
P120	124 25(9.03)	125.71 (10.73)	115.2 (6.26)	119.43 (9.71)
N145	153.56(14.20)	144.18 (12.41)	142.36(11.55)	141.64 (11.38)

Table 8.3. The Group Mean Peak Latencies (ms) and Standard Deviation, in Brackets, of the Major Components Following Quadrant Stimulation.

Field	R Up Quad	L Up Quad	R Lo Quad	L Lo Quad
N75	-1.78 (1.74)	-1.8 (1.75)	-1.49 (1.78)	-2.30 (2.08)
P80	3.36 (0.94)	4.06 (1.8)	4.19 (1.33)	3.92 (2.39)
P100	4.64 (2.02)	4.67 (1.57)	5.88 (1.59)	6.15 (1.49)
N105	-5.59(0)	-0.27(0)		-0.01(0)
P120	5.43 (1.58)	1.10 (1.20)	3.36 (1.22)	3.34 (0.87)
N145	-2.37 (1.62)	-1.45 (1.16)	-3.63 (3.0)	-3.73 (1.61)

Table 8.4. The Group Mean Peak Amplitudes (μV) and Standard Deviation, in Brackets, of the Major Components Following Quadrant Stimulation.

8.2.2.i Quadrantic Stimulation; Topographic Distribution from Group Mean Waveform

An early negativity was not shown after lower quadrant stimulation. In contrast a low amplitude early negativity was recorded from the upper quadrants, localised over the anterior ipsilateral region of the montage. The negativity was therefore distributed ipsilaterally in both aspects of the stimulus location. A positivity was

Figure 8.1. Group mean waveform of the pattern reversal response following right lower quadrant stimulation. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.

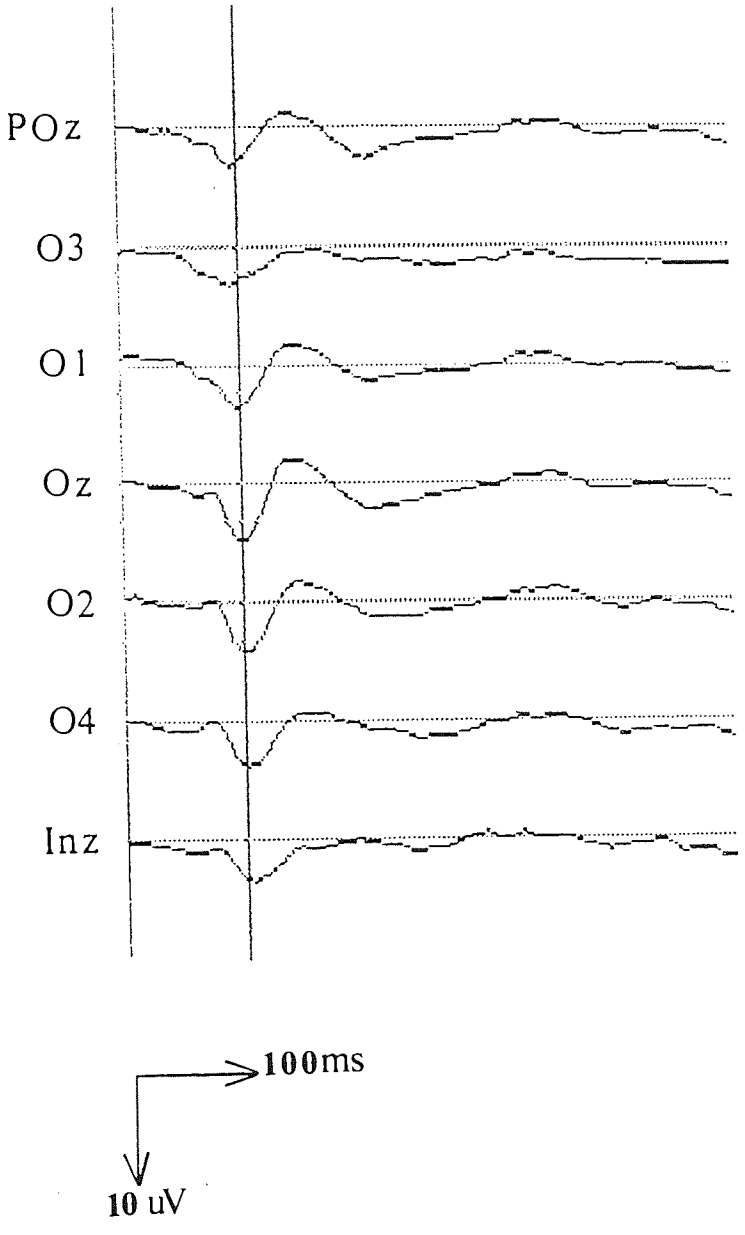

POz
O3
O1
Oz
O2
O4
Inz
100ms
10 uV

Figure 8.2. Group mean waveform of the pattern reversal response following left lower quadrant stimulation. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.

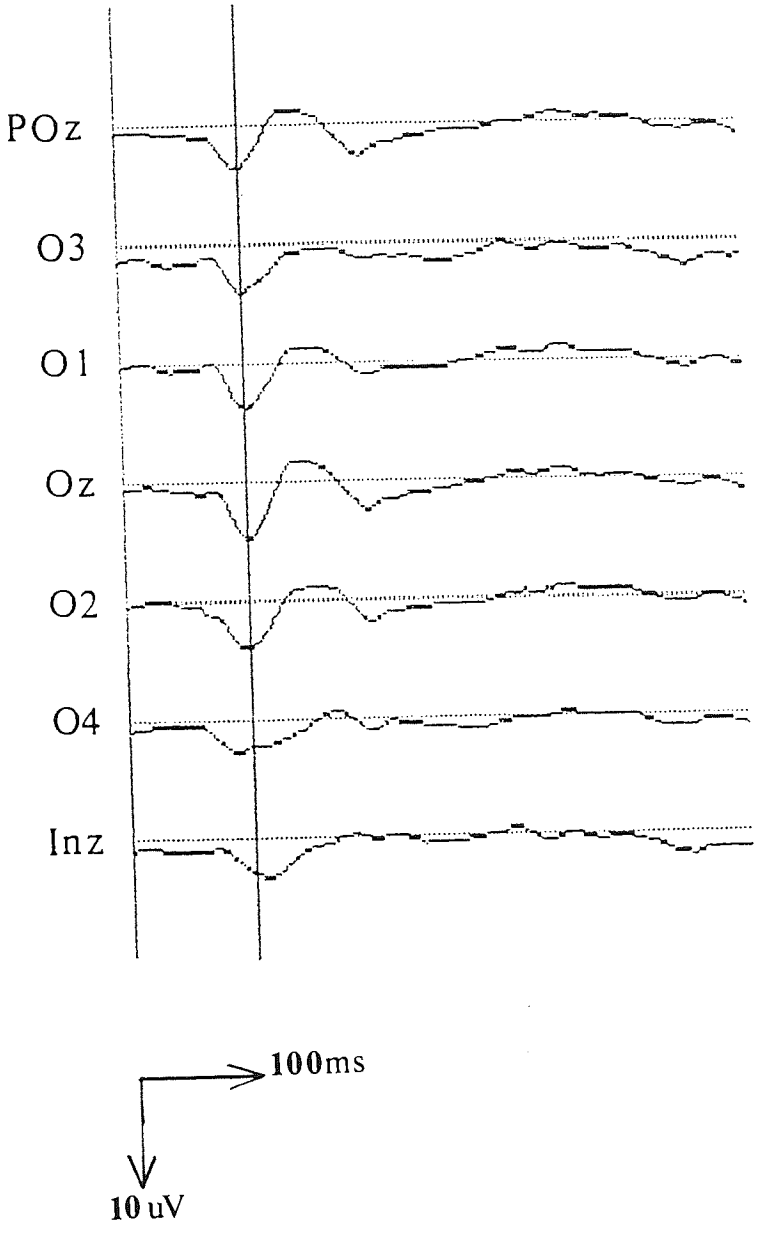
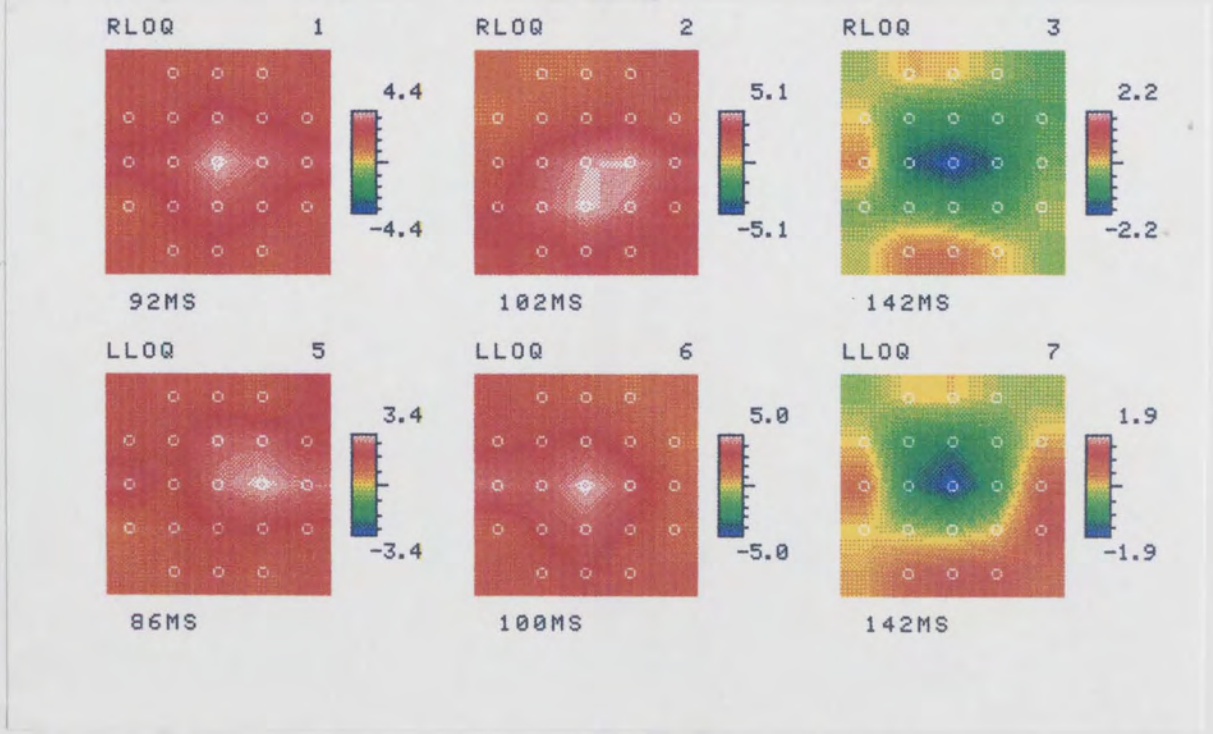

POz
O3
O1
Oz
O2
O4
Inz
100ms
10 uV

Figure 8.3. The topographical distribution of the major components in the group mean waveform following right and left lower quadrant stimulation. Key; RLOQ = right lower quadrant, LLOQ = left lower quadrant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.


RLOQ 1
4.4
-4.4
92MS
RLOQ 2
5.1
-5.1
102MS
RLOQ 3
2.2
-2.2
142MS
LLOQ 5
3.4
-3.4
86MS
LLOQ 6
5.0
-5.0
100MS
LLOQ 7
1.9
-1.9
142MS

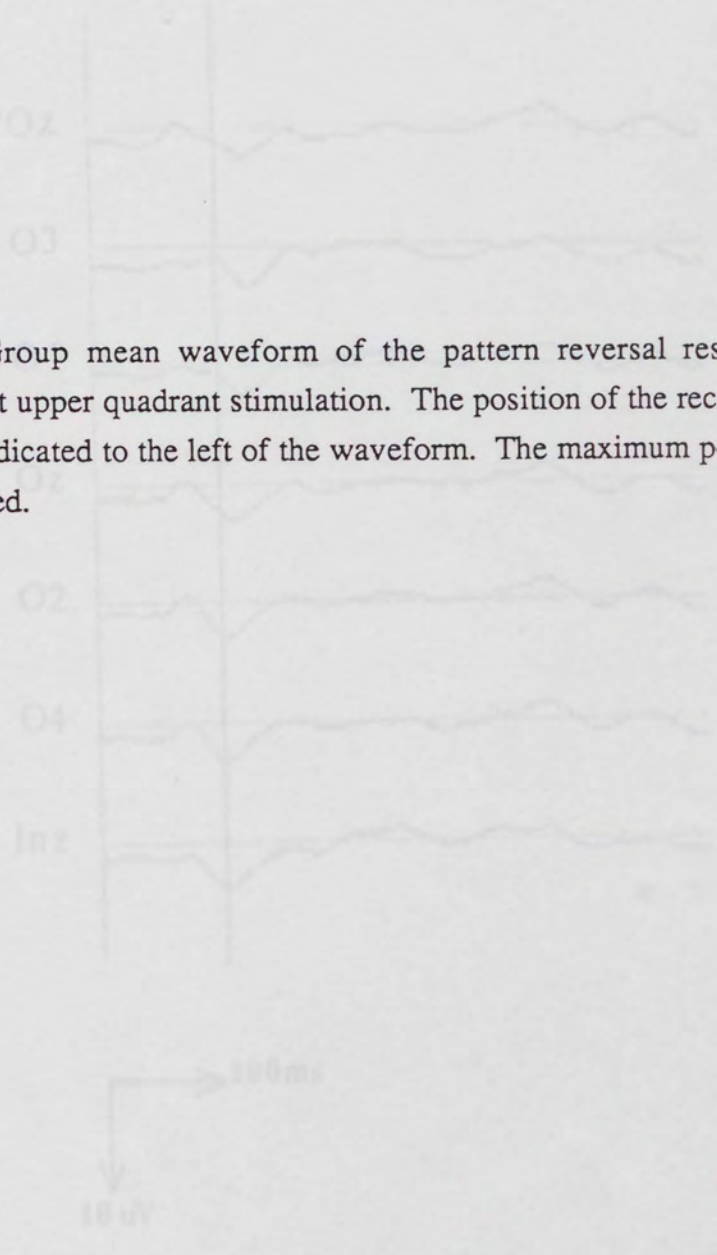
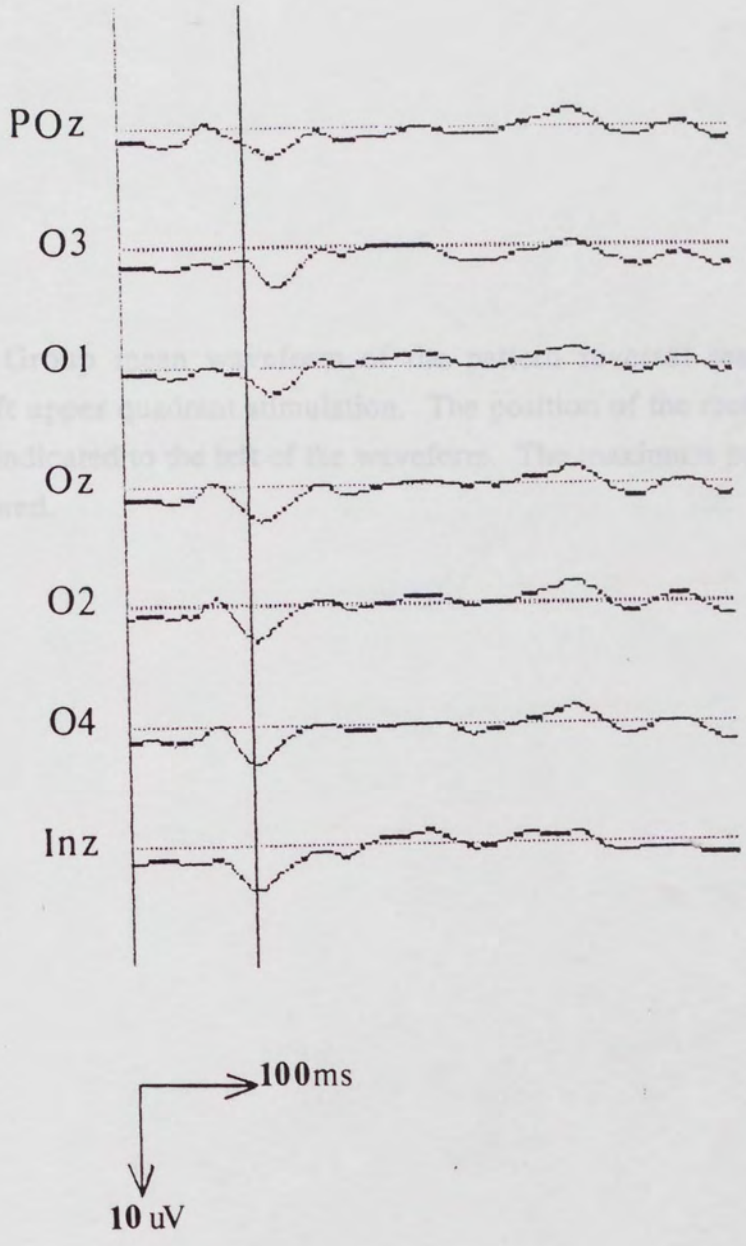


Figure 8.4. Group mean waveform of the pattern reversal response following right upper quadrant stimulation. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.


POz
O3
O1
Oz
O2
O4
Inz
100ms
10 uV

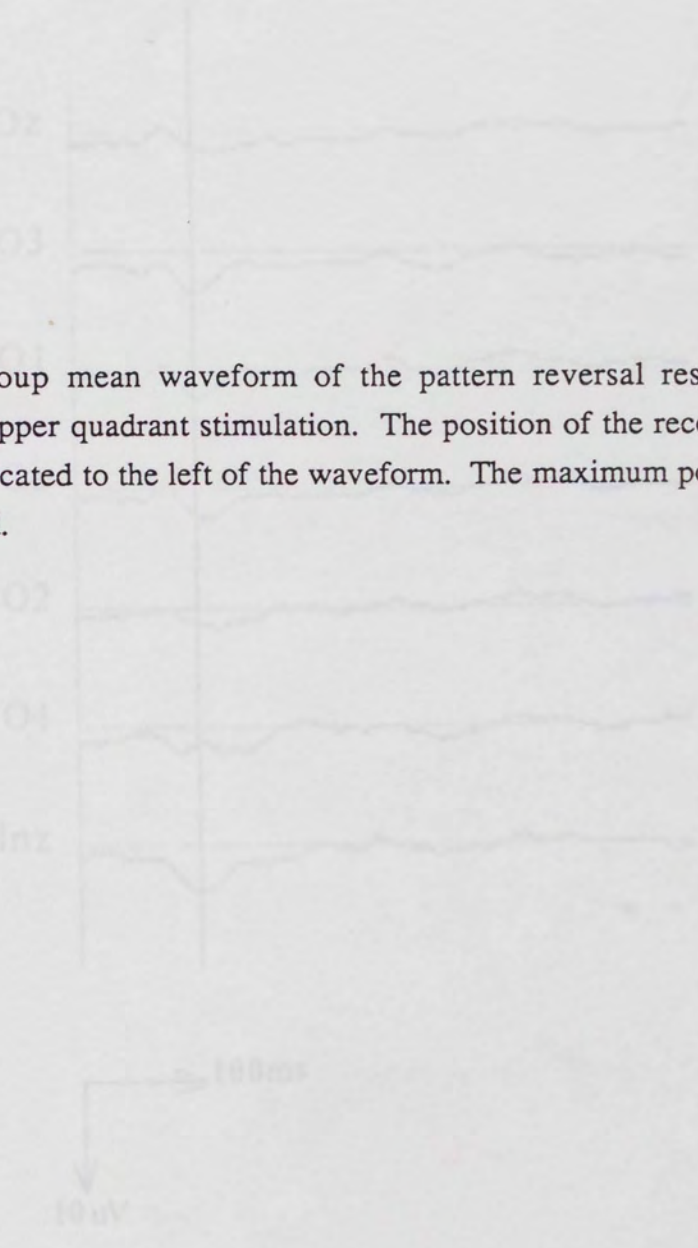


Figure 8.5. Group mean waveform of the pattern reversal response following left upper quadrant stimulation. The position of the recording electrode is indicated to the left of the waveform. The maximum positive peak is cursored.

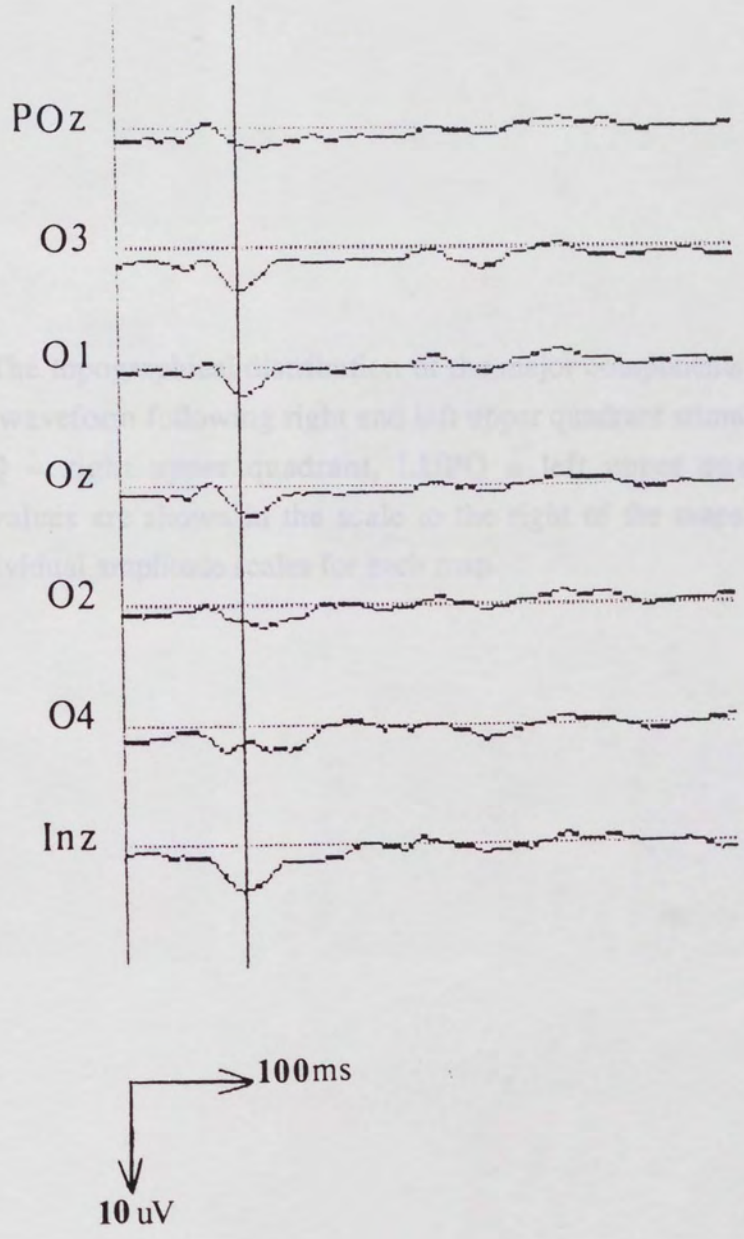

POz
O3
O1
Oz
O2
O4
Inz
100ms
10 uV

Figure 8.6. The topographical distribution of the major components in the group mean waveform following right and left upper quadrant stimulation. Key; RUPQ = right upper quadrant, LUPQ = left upper quadrant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scales for each map.

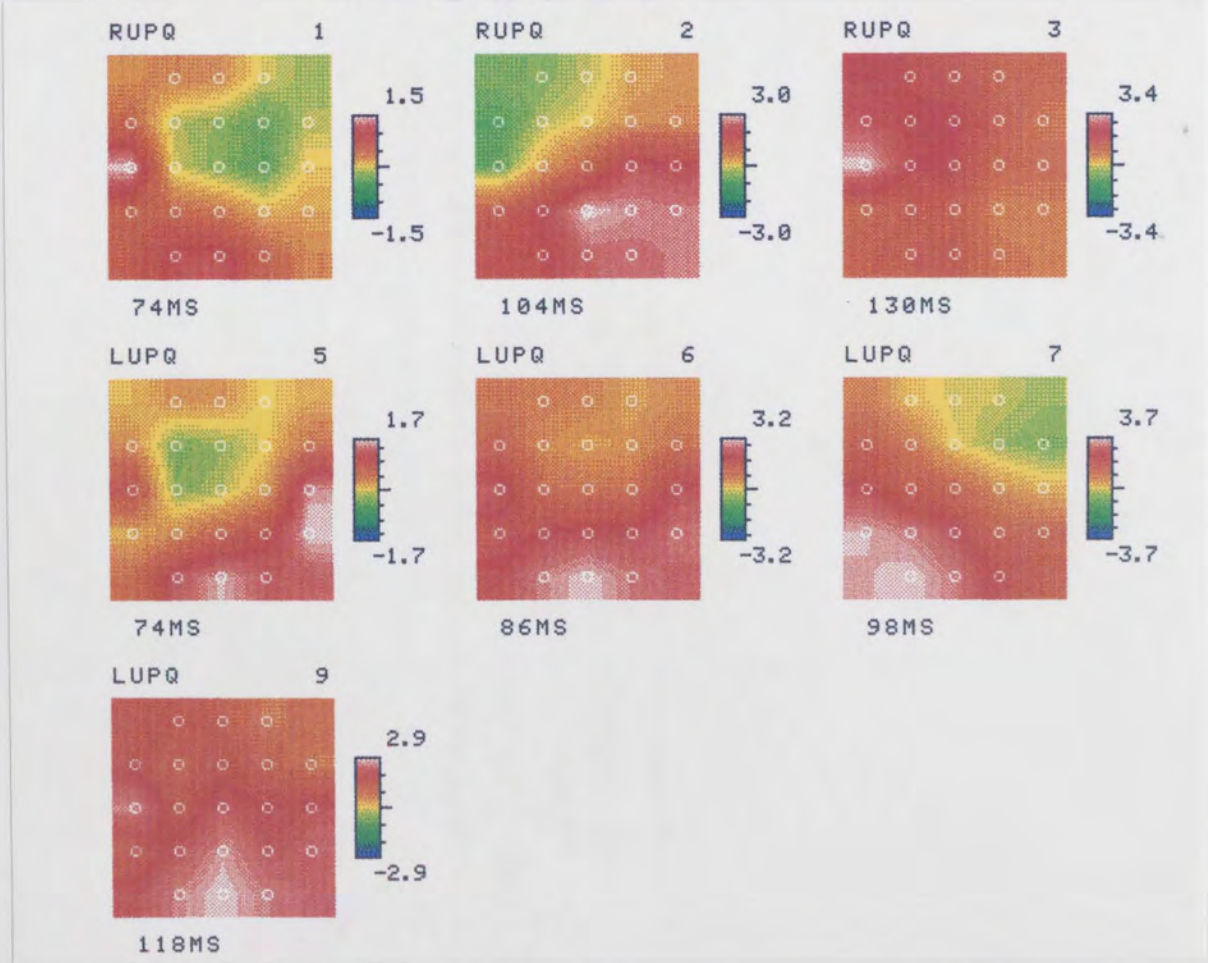

RUPQ 1
1.5
-1.5
74MS
RUPQ 2
3.0
-3.0
104MS
RUPQ 3
3.4
-3.4
130MS
LUPQ 5
1.7
-1.7
74MS
LUPQ 6
3.2
-3.2
86MS
LUPQ 7
3.7
-3.7
98MS
LUPQ 9
2.9
-2.9
118MS

Figure 8.7. The topographical distributions of the major components from the group mean maps following right lower quadrant stimulation. Key RLOQ = right lower quadrant, EARLYN = =early negativity, EARLYP = early positivity, LATEP = late positivity and LATEN = late negativity. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.

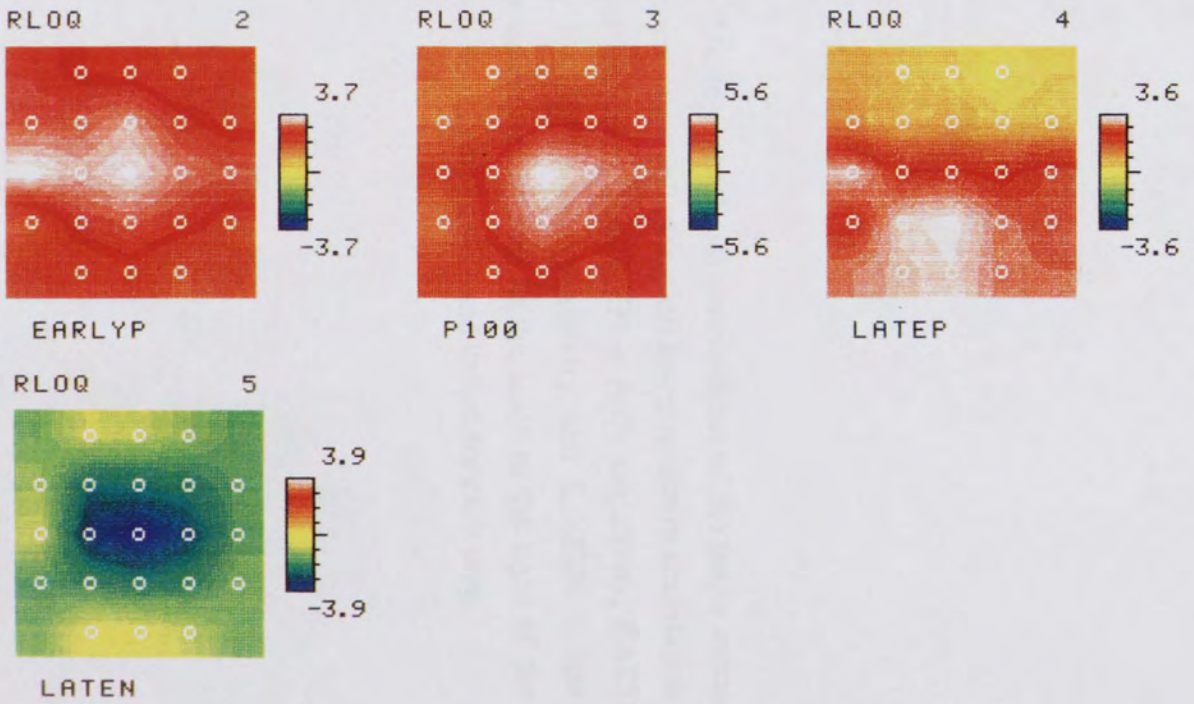

RLOQ 2
3.7
-3.7
EARLYP
RLOQ 3
5.6
-5.6
P100
RLOQ 4
3.6
-3.6
LATEP
RLOQ 5
3.9
-3.9
LATEN

Figure 8.8. The topographical distributions of the major components from the group mean maps following left lower quadrant stimulation. Key LLOQ = left lower quadrant, EARLYN = early negativity, EARLYP = early positivity, LATEP = late positivity and LATEN = late negativity. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.

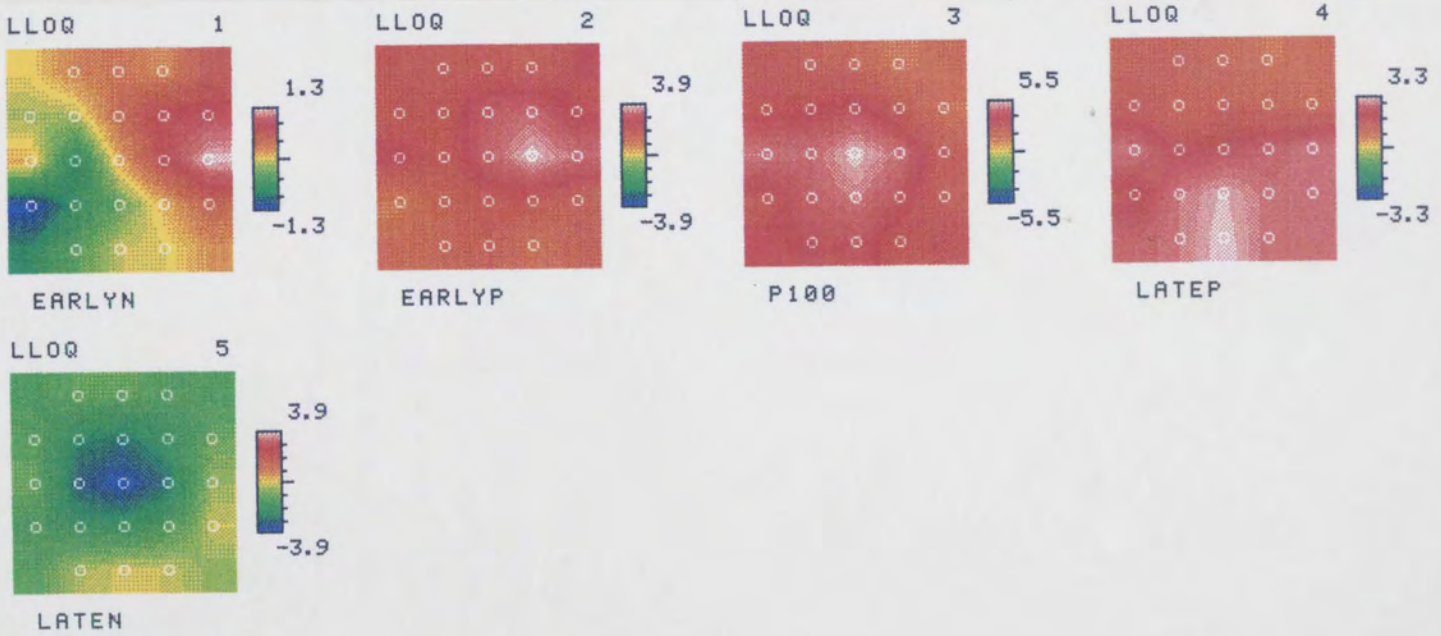

LLOQ 1
1.3
-1.3
EARLYN
LLOQ 2
3.9
-3.9
EARLYP
LLOQ 3
5.5
-5.5
P100
LLOQ 4
3.3
-3.3
LATEP
LLOQ 5
3.9
-3.9
LATEN

Figure 8.9. The topographical distributions of the major components from the group mean maps following right upper quadrant stimulation. Key RUPQ = right upper quadrant, EARLYN = early negativity, EARLYP = early positivity, LATEP = late positivity and LATEN = late negativity. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.

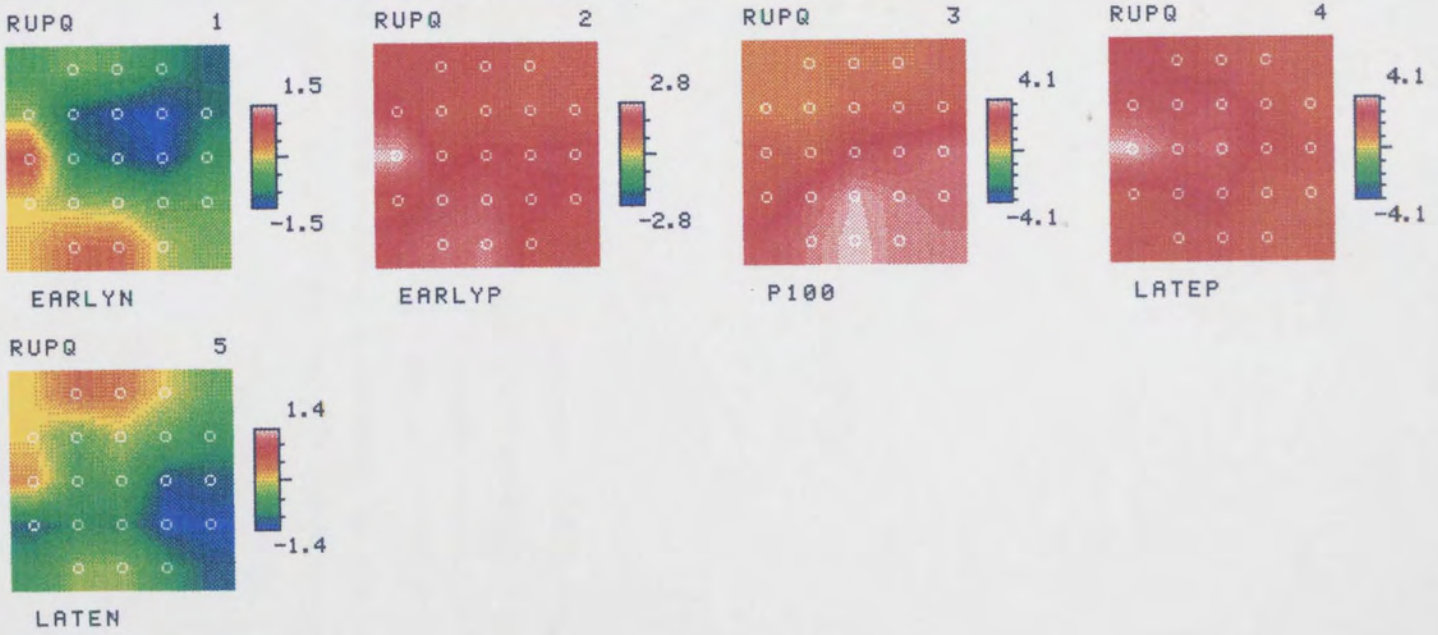
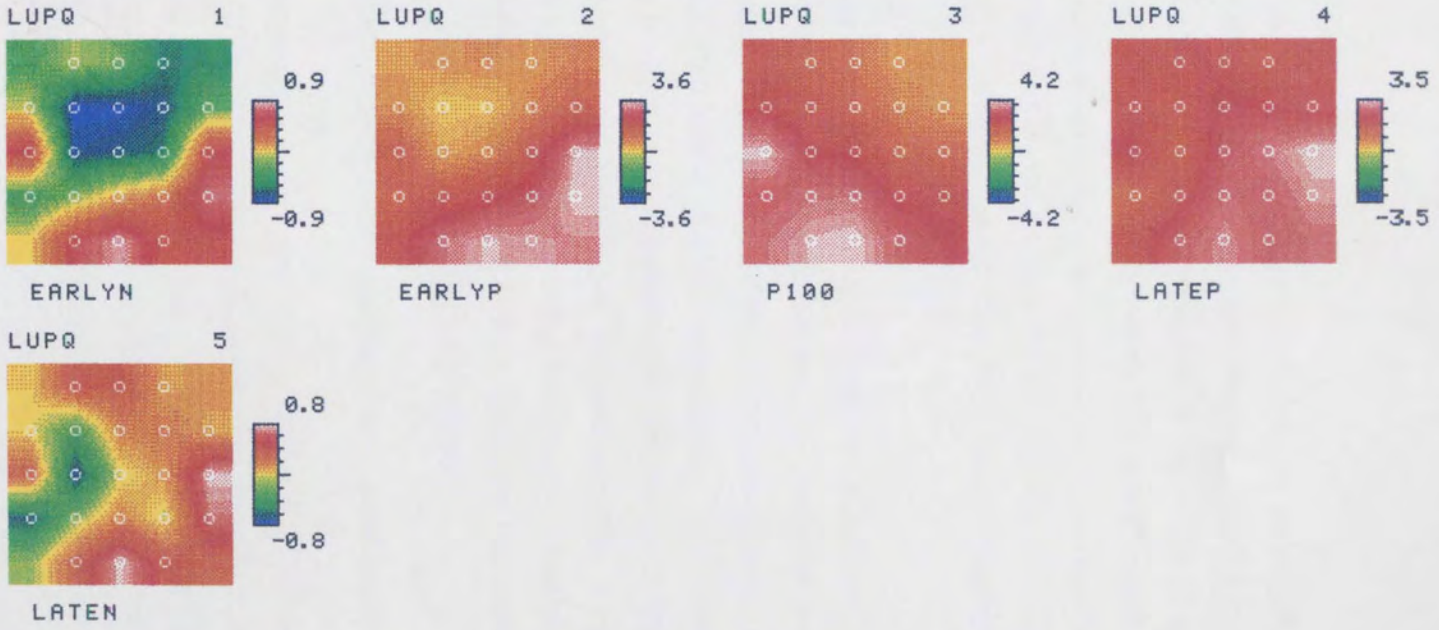

RUPQ 1
1.5
-1.5
EARLYN
RUPQ 2
2.8
-2.8
EARLYP
RUPQ 3
4.1
-4.1
P100
RUPQ 4
4.1
-4.1
LATEP
RUPQ 5
1.4
-1.4
LATEN

Figure 8.10. The topographical distributions of the major components from the group mean maps following left upper quadrant stimulation. Key LUPQ = left upper quadrant, EARLYN = early negativity, EARLYP = early positivity, LATEP = late positivity and LATEN = late negativity. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.


LUPQ 1
0.9
-0.9
EARLYN
LUPQ 2
3.6
-3.6
EARLYP
LUPQ 3
4.2
-4.2
P100
LUPQ 4
3.5
-3.5
LATEP
LUPQ 5
0.8
-0.8
LATEN

then recorded from the left upper quadrant occuring at a similar latency as the maximal early contralateral positivity after lateral half field stimulation. This was distributed over the posterior central region of the montage i.e contralateral to the stimulating field. The next component recorded from both upper quadrants was the 'P100', this was maximal over the lower ipsilateral region, ipsilateral to the lateral aspect of the quadrant but contralateral to the altitudinal part. A later positivity was then observed with a posterior distribution from the left quadrant and an anterior distribution from the right quadrant.

An early positivity was recorded with both lower quadrants, this was centrally distributed after right quadrant stimulation and maximal over the right central area after left stimulation. This peak occured at the latency of the early contralateral positivity recorded from lateral half field stimulation. The following positivity was maximal over the midline and ipsilateral region, the laterality was therefore similar to that after upper quadrant stimulation. A positivity over the posterior region of the montage was also recorded this being of a greater amplitude with left lower quadrant stimulation. The negativity recorded from both lower quadrants was maximal over Oz, as with lower half field stimulation.

8.2.2.ii Morphological Variations Between Subjects

Contralateral components were found to be more pronounced with upper quadrant stimulation with 70% of the subjects demonstrating definite components compared with 20% for the right lower quadrant and 30% for the left lower quadrant. In 30% of the subjects no contralateral components were observed from either lower left or right quadrant stimulation. The ipsilateral components were of a very low amplitude in one subject after upper left quadrant stimulation.

Group mean maps of the upper right quadrant produced no early negativity instead only a reduction in positivity was evident over the ipsilateral hemisphere. This indicates a disparity across the subjects in the location and distribution of the maximal response.

8.2.3 Global Field Power Analysis

Stimulus Location	Field Power Latency	Field Power Amplitude	Field Power Latency	Field Power Amplitude	Field Power Latency	Field Power Amplitude
Left Upper Quadrant			**98ms**	**1.08**	138ms	0.51
Right Upper Quadrant			**102ms**	**1.15**	136ms	0.69
Left Lower Quadrant	76ms	0.56	102ms	0.88	**132ms**	**0.96**
Right Lower Quadrant	74ms	0.69	**106ms**	**1.03**	132ms	0.82

Table 8.5. The Latency and Power of the Peaks Following Global Field Power Analysis.

The lower quadrants show a low amplitude positive over the contralateral hemisphere at the latency of the first peak and an ipsilateral positive for the second peak, the distributions are similar to full lateral half field stimulation with the exception that there is no negativity present on the map of the first peak after quadrant stimulation. The third peak in the power spectrum is a negativity distributed over the anterior region of the montage, this being slightly more central for right lower quadrant stimulation.

The upper quadrants show no initial peak in the global field power as with upper half field stimulation. The second peak represents a positive over the posterior area of the montage with an ipsilateral distribution. The later peak distribution shows a positive over the contralateral hemisphere.

8.2.4 Comparison of the Group Mean Maps and the Group Mean Waveforms

8.2.4.i Group Mean Maps

8.2.4.ia Early Negativity (N75)

N75 was maximal ipsilateral with respect to the lateral half field and ipsilongitudinal with respect to the altitudinal field, on lower quadrant stimulation the amplitude of the negativity was reduced and not present in the group mean map of right lower quadrant. On lower left quadrant stimulation however, the response was present and remained maximal over the ipsilaterally as following upper quadrant stimulation.

8.2.4.ib Major Positivity (P100)

The amplitudes and latencies of the responses from the quadrants was compared with each other and with the half field responses using a two factor analysis of variance in randomised blocks. The amplitude of the left lower quadrant response was found to be significantly greater ($F = 10.14$ $p<0.001$) than the response from the upper left and right quadrants, the difference between the right lower quadrant and the upper quadrants just failed to reach significance. No significant difference in the amplitudes was demonstrated between the lower quadrants and the lateral half fields, in contrast the lateral half fields were found to be significantly larger than the upper quadrant responses ($F = 10.14$ $p<0.001$). The summed quadrant response has previously been shown to be greater than the full half field (Howe and Mitchell 1980). Kakisu (1985) has also shown a reduction in amplitude of the upper quadrants' response to a steady state stimulus. In addition Kakisu demonstrated a delay in the response from upper quadrants not confirmed in this report (Kakisu 1985, 1988).

8.2.4.ic Late Positivity (P120)

In contrast to the early positivity the late positivity was more localised with upper quadrant stimulation when compared with lower quadrant stimulation. The left upper quadrant response was maximal over O4, the right being maximal over O3.

8.2.4.id Late Negativity (N145)

The distribution of the N145 component has previously been shown to be maximal ipsilaterally for lateral half field stimulation but contralongitudinal with altitudinal half field stimulation. This was reflected in the distribution after upper quadrant stimulation, for both left and right upper quadrant stimulation the response is maximal ipsilateral in the lateral field aspect and contralongitudinal in the altitudinal aspect. On lower quadrant stimulation the response was maximal over Oz, with a slight left bias for the lower left quadrant.

8.2.4.ii Contralateral Components

An increase in the prominence of the contralateral components has previously been demonstrated after upper quadrant stimulation. In contrast the responses from lower quadrant stimulation emphasized the ipsilateral components resulting in a spread of positivity across the midline greater than that observed with the full half field response (Blumhardt and Halliday 1979). The widespread ipsilateral positivity and small contralateral complex were consistent with the situation of the central lower field representation on the convexity of the upper surface of the occipital lobe. Emphasis of the contralateral components from the upper quadrant was attributed to the extension of the cortical representation of this area onto the inferior surface of the hemisphere. The lower axis of the upper field generator area was thought to be tilted obliquely so it was directed towards the electrodes on the contralateral scalp and away from those on the ipsilateral cortex. In comparison with this study contralateral components were recorded with both upper and lower quadrants in approximately equivalent numbers of subjects, however they were more pronounced following upper quadrant stimulation. The ipsilateral components especially the P100 and N145 appear to be of larger amplitude with lower quadrant stimulation, while the late positivity appears to be greater with upper quadrant stimulation.

Studies on the magnetic evoked field, which preferentially detects tangential fields have shown greater field strengths from lower quadrant stimulation to pattern reversal stimulation (Seki et al 1991), this would suggest that the lower quadrant dipole was more tangential. This would not fit with the fact that contralateral components are more definite after upper field stimulation, in contrast this would suggest that the source of the upper field response was more tangentially orientated.

8.3 Progressive occlusion of the central area of the left upper quadrant

8.3.1 Introduction

The later positivity observed after upper half field stimulation, see chapter six, was also recorded on quadrant stimulation it being more distinct and of a larger amplitude following upper quadrant stimulation. As a result of the central and peripheral study suggesting that maybe the two components had different sources, a further reduction in the stimulating field size was employed to reduce the amount of opposing sources, thus aiding in interpretation of the results.

8.3.2 Method

Three consenting subjects (V.V, G.B and E.W), two female and one male participated in this study, age range 23-29, with no ophthalmological or neurological deficits and visual acuities of 6/6 or better with correction if necessary. Twenty electrodes were placed according to the montage described in chapter 5, covering the occipital scalp. The stimulus consisted of a reversing black and white checkerboard presented in the left upper quadrant, radius 10°, this was progressively occluded by a central quadrantic mask increasing in 1° radius steps.from 1°. The check size used was 27', reversal rate of 1 Hz, luminance of 1050cd/m^2 and contrast of 80%.

8.3.3. Results

The individual waveforms for each stimulus and subject are shown in the appendices, figs A.1 - A.8.

8.3.3.i Subject V.V.

Left upper quadrant stimulation produced the following components; an early contralateral positivity, a posterior positivity and a later contralateral positivity, see fig.8.11. On occlusion of the central 1° both the early contralateral positivity and the posterior postivity remained but the later contralateral positivity was not present. As the size of occlusion increased the early positivity remained maximal over O4 and was still present with central 5° radius occlusion. The major positivity around 100msec was maximal over the posterior region of the montage, with increasing occlusion this response tended to become more diffuse. With 3°

occlusion this peak around 100 msec was not present however, with 4° occlusion this component was again recorded.

8.3.3.ii Subject GB

On left upper quadant stimulation the only peak apparent was a positivity maximal over O3 at 108msec, see fig 8.12. The introduction of a central 1° scotoma lead to the appearance of a negativity at 78msec over the anterior left of the montage. An increase in the size of occlusion resulted in the appearance and increase in amplitude of an early contralateral positivity this was paralleled by a simultaneous reduction in amplitude of the negativity. On central 4° occlusion this contralateral positivity was no longer evident. The major positivity occuring around 100msec remained maximal over the ipsilateral hemisphere with increasing central occlusion, a negativity was also associated with this component on 4° occlusion. A later posterior positivity was also recorded after central 2° and 4° occlusion.

8.3.3.iii Subject EW

Left upper quadrant stimulation produced an early contralateral positivity, a posterior postivity and a later contralateral positvity, see fig. 8.13. On increasing central occlusion the positivity around 100ms remained maximal over the ipsilateral montage. The early contralateral positivity was no longer recorded with 4° occlusion, as for subject GB.

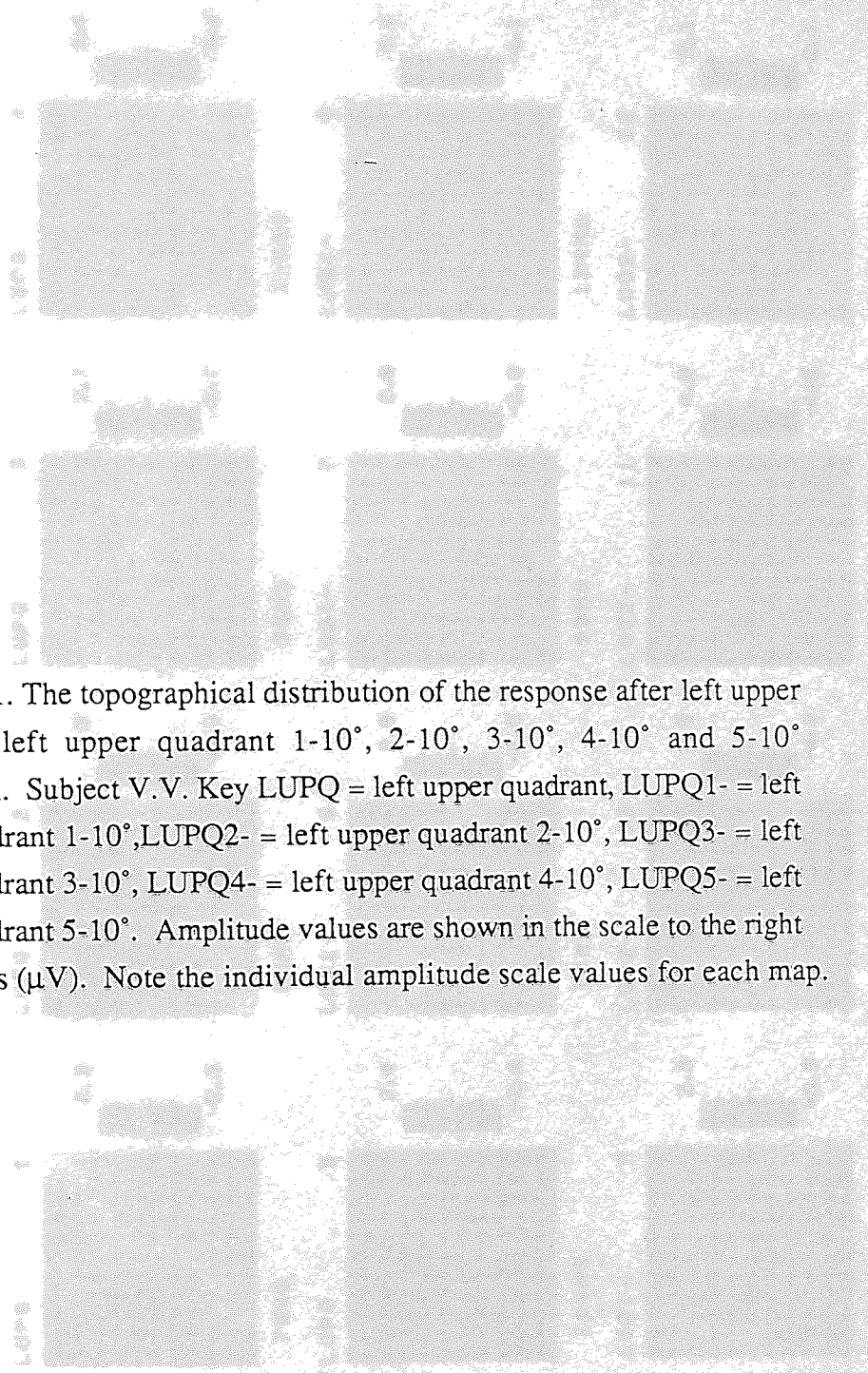
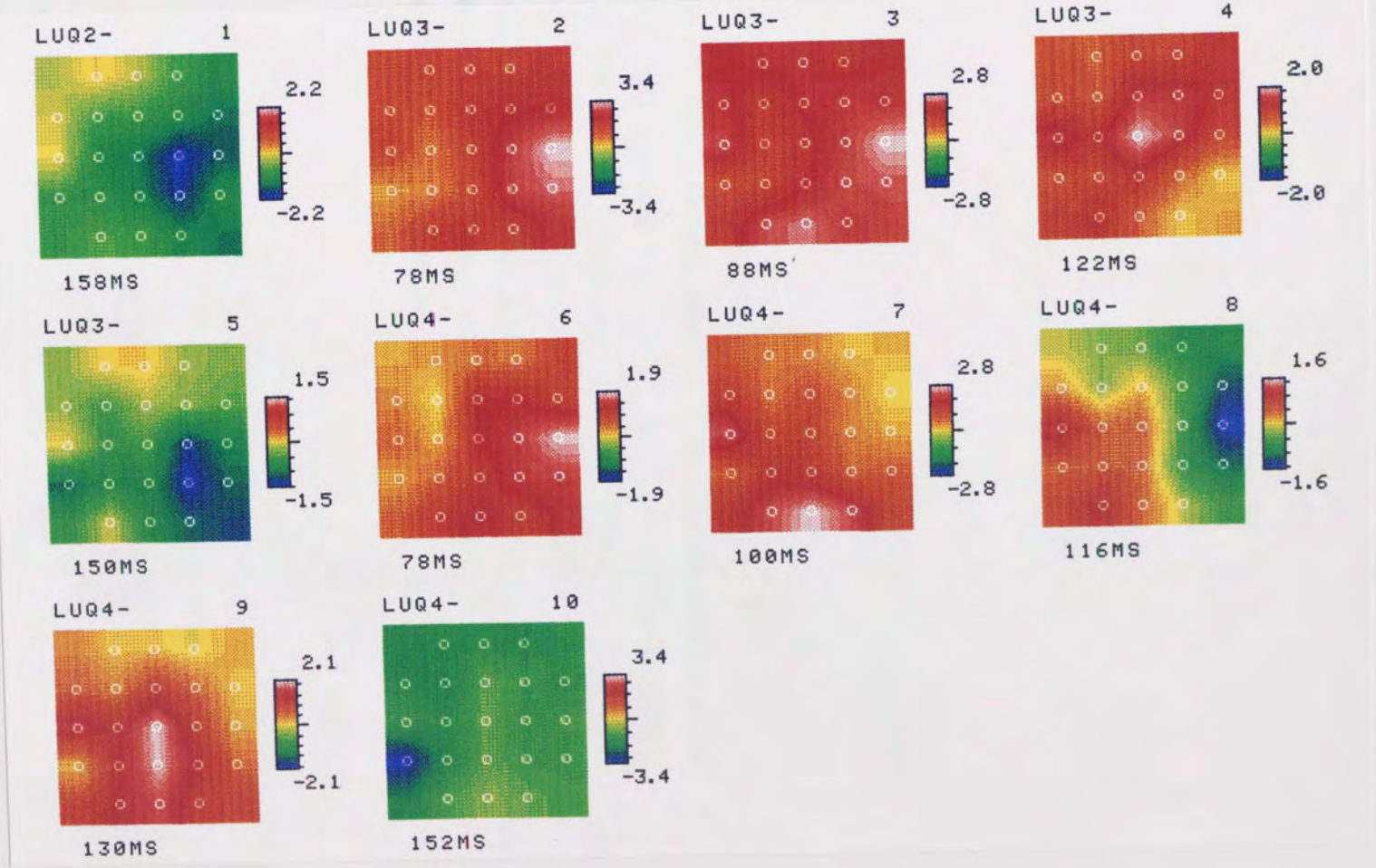


Figure 8.11. The topographical distribution of the response after left upper quadrant, left upper quadrant 1-10°, 2-10°, 3-10°, 4-10° and 5-10° stimulation. Subject V.V. Key LUPQ = left upper quadrant, LUPQ1- = left upper quadrant 1-10°,LUPQ2- = left upper quadrant 2-10°, LUPQ3- = left upper quadrant 3-10°, LUPQ4- = left upper quadrant 4-10°, LUPQ5- = left upper quadrant 5-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.

LUPQ 1
2.3
-2.3
70MS
LUPQ 2
2.0
-2.0
82MS
LUPQ 3
2.7
-2.7
96MS
LUPQ 4
2.6
-2.6
118MS
LUPQ 5
2.4
-2.4
164MS
LUQ1- 6
2.8
-2.8
76MS
LUQ1- 7
2.0
-2.0
98MS
LUQ1- 8
4.1
-4.1
124MS
LUQ1- 9
2.9
-2.9
178MS
LUQ2- 10
4.0
-4.0
78MS
LUQ2- 11
4.0
-4.0
98MS
LUQ2- 12
2.1
-2.1
130MS


LUQ2- 1
2.2
-2.2
158MS
LUQ3- 2
3.4
-3.4
78MS
LUQ3- 3
2.8
-2.8
88MS
LUQ3- 4
2.0
-2.0
122MS
LUQ3- 5
1.5
-1.5
150MS
LUQ4- 6
1.9
-1.9
78MS
LUQ4- 7
2.8
-2.8
100MS
LUQ4- 8
1.6
-1.6
116MS
LUQ4- 9
2.1
-2.1
130MS
LUQ4- 10
3.4
-3.4
152MS

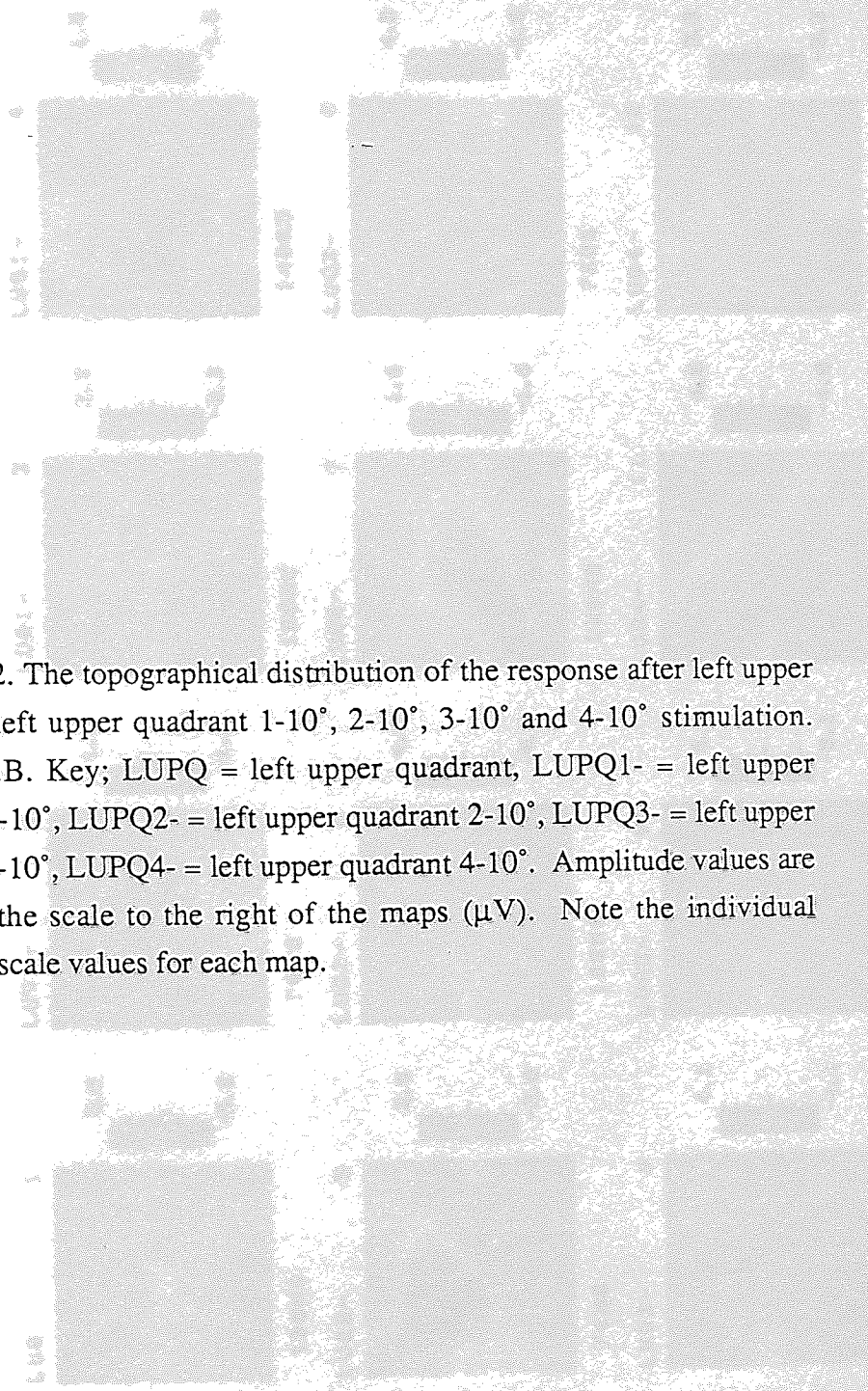
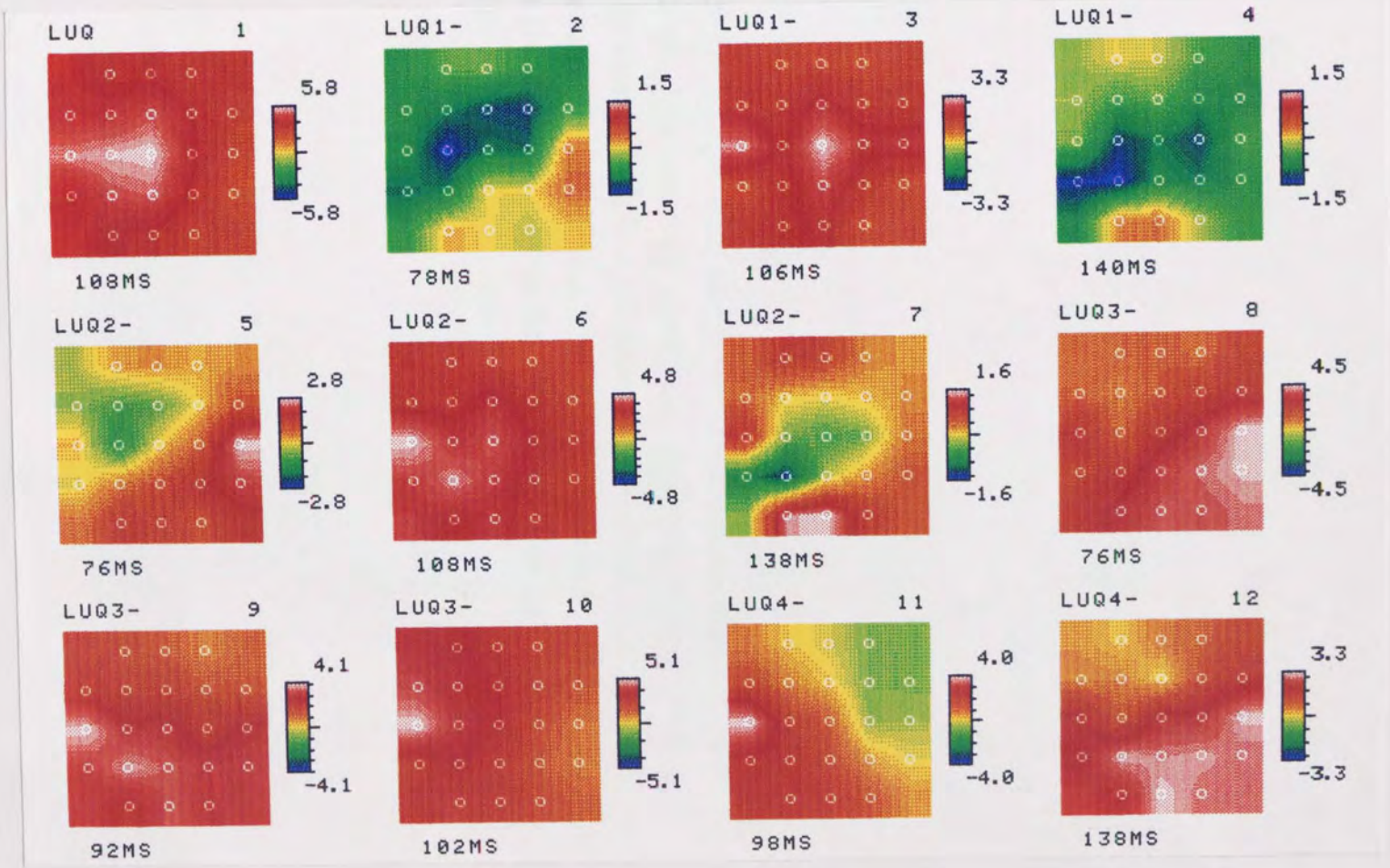


Figure 8.12. The topographical distribution of the response after left upper quadrant, left upper quadrant 1-10°, 2-10°, 3-10° and 4-10° stimulation. Subject G.B. Key; LUPQ = left upper quadrant, LUPQ1- = left upper quadrant 1-10°, LUPQ2- = left upper quadrant 2-10°, LUPQ3- = left upper quadrant 3-10°, LUPQ4- = left upper quadrant 4-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.


LUQ 1
5.8
-5.8
108MS
LUQ1- 2
1.5
-1.5
78MS
LUQ1- 3
3.3
-3.3
106MS
LUQ1- 4
1.5
-1.5
140MS
LUQ2- 5
2.8
-2.8
76MS
LUQ2- 6
4.8
-4.8
108MS
LUQ2- 7
1.6
-1.6
138MS
LUQ3- 8
4.5
-4.5
76MS
LUQ3- 9
4.1
-4.1
92MS
LUQ3- 10
5.1
-5.1
102MS
LUQ4- 11
4.0
-4.0
98MS
LUQ4- 12
3.3
-3.3
138MS

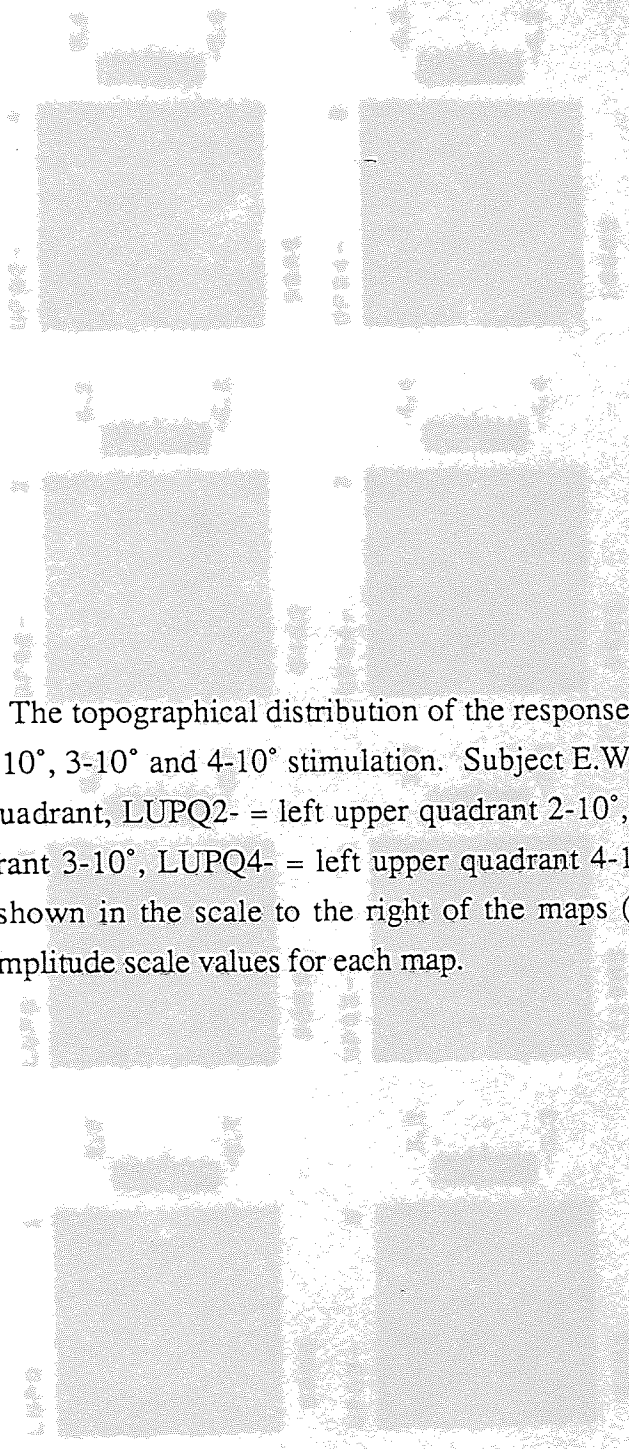
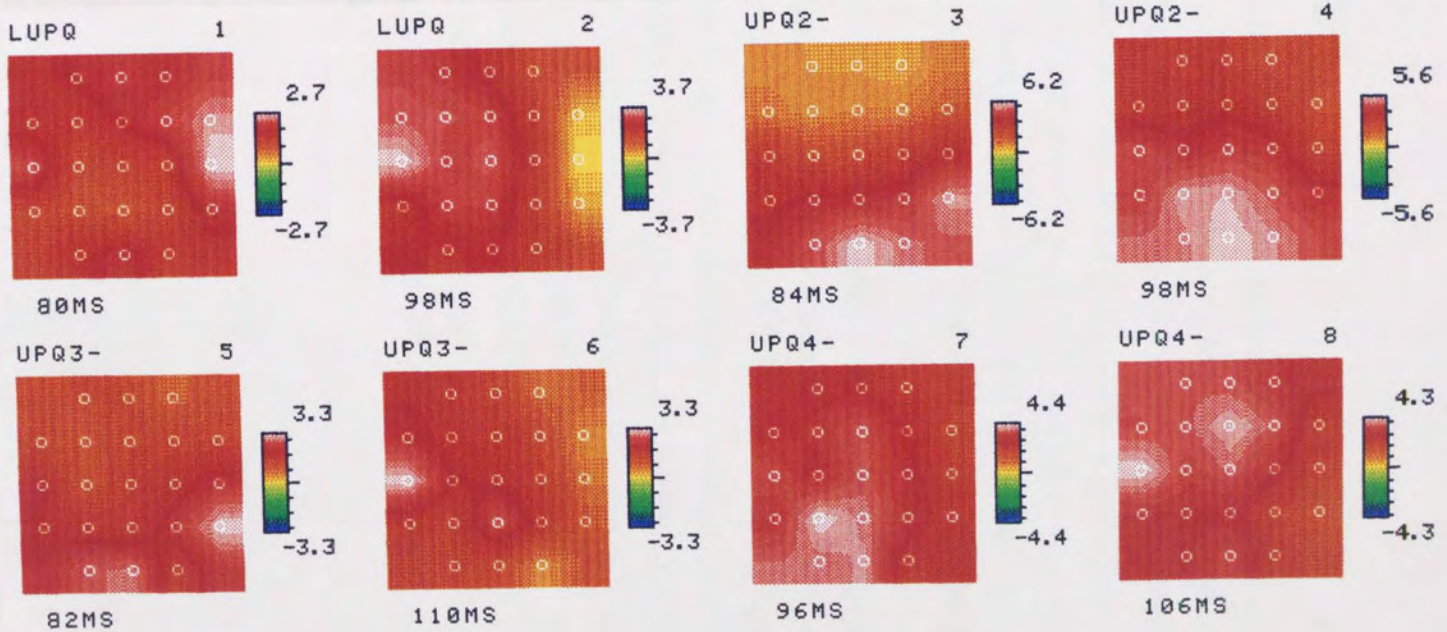


Figure 8.13. The topographical distribution of the response after left upper quadrant, 2-10°, 3-10° and 4-10° stimulation. Subject E.W. Key; LUPQ = left upper quadrant, LUPQ2- = left upper quadrant 2-10°, LUPQ3- = left upper quadrant 3-10°, LUPQ4- = left upper quadrant 4-10°. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.


LUPQ 1
2.7
-2.7
80MS
LUPQ 2
3.7
-3.7
98MS
UPQ2- 3
6.2
-6.2
84MS
UPQ2- 4
5.6
-5.6
98MS
UPQ3- 5
3.3
-3.3
82MS
UPQ3- 6
3.3
-3.3
110MS
UPQ4- 7
4.4
-4.4
96MS
UPQ4- 8
4.3
-4.3
106MS

8.3.4 Discussion

Reduction of the field size produces an increase in the variability of the responses, this may be a result of enhancement in asymmetries present in the cortical structures between individuals as a result of a reduction in summation effects. It appears that occlusion of the central field resulted in an increase in the amplitude of the early contralateral positivity upto 3° occlusion (i.e central 6° field), on increasing the central scotoma further this component was abolished in 2/3 subjects. This component may therefore be related to processing in the central/extra foveal areas. This component was however not present after full field stimulation, see chapter 5, this could be a result of response cancellation in the fissures. The amplitude of this component was also found to increase with a reduction in the check size, see chapter 7, further indicating a preferentially foveal source. The distribution of this peak is very localised over the extreme of the montage with a steep potential gradient, this may suggest a radial source. The major positivity occuring around 100ms became increasingly more diffuse with an increase in the scotoma size for subject V.V. this could be a result of smearing of the response by intervening cortical tissues as the site of generation becomes more anterior. The late contralateral positivity was eliminated with peripheral stimulation in subject V.V., this would fit with this component being generated in the central field.

8.4 Stimulation With Dioctants

8.4.1 Introduction

The classical model of the site of generation of the responses from the striate cortex is that of the cruciform model. On examination of the location of the cortical representations of the visual field it is perceived that the equivalent dipoles from quadrant stimulation are directed perpendicularly with respect to one another. The sum of these two could be affected to a greater extent activation of one of the octants and therefore produce a different distribution to another subject in which the response is more prominent from the other sector.

A stimulus that results in activation of the cortex with approximately the same orientation should produce a response that is not limited to a great extent by cancellation or vector summation effects (Harding 1991). This stimulus would be in the form of two upper and lower vertical octants in the case of the lateral half fields and upper or lower horizontal octants for altitudinal field stimulation. The response from this stimulus type was therefore investigated, in addition the response distribution was studied after stimulation with the constituent octants to examine any cancellation effects produced. A comparison of the response distribution from these types of stimuli has not previously been reported in the literature.

8.4.2 Method

The two types of stimulus (dioctants) were termed opposing (O) and non-opposing (N-O) relating to the position of the equivalent dipole generators on the cruciform model. Figures 8.14 and 8.15 illustrate the stimuli used and the cruciform model illustrates the approximate area of activation.

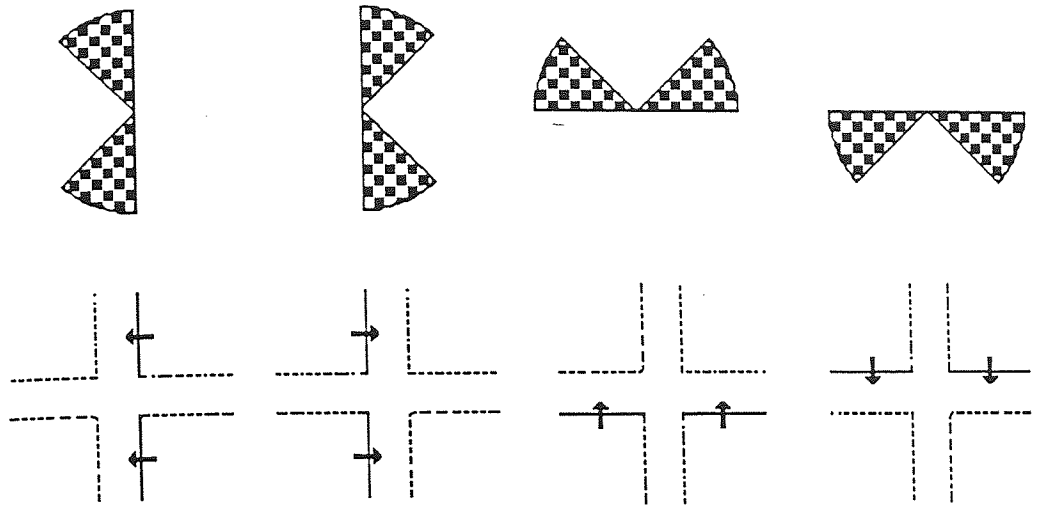


Fig 8.14 The visual field location and cortical projection of the non-opposing stimuli.

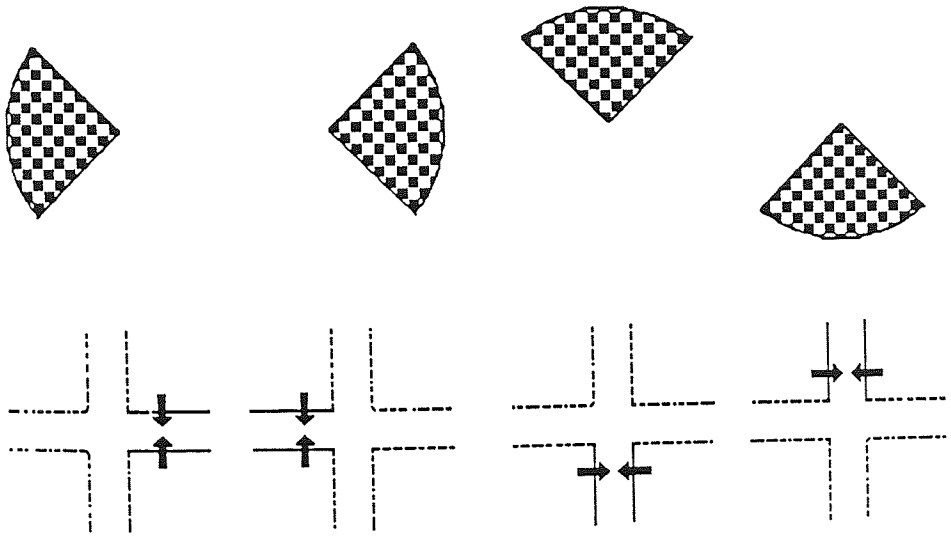


Fig 8.15 The visual field location and cortical projection of the opposing stimuli

Five subjects were used with the N-O dioctants, age range 22-27, three male and two female, three subjects were used for the O dioctants, age range 22-27, two male one female. The response was recorded as described in the methods section at the beginning of this chapter.

8.4.3 Results

8.4.3.i Morphological Variations Between Subjects

8.4.3.ia Subject RD (N-O dioctant)

No contralongitudinal components were recorded with the lower dioctant, however on upper dioctant stimulation there appeared to be an anterior negative deflection at the latency of the P100 this was followed by a late anterior positive, see fig.8.16. A late contralateral positivity was recorded on both left and right dioctant stimulation, in addition an early contralateral positivity was recorded with the right dioctant stimulus.

8.4.3.ib Subject MDH (N-O dioctant)

The upper dioctant stimulus produced an anterior negative-positive-negative complex. Contralateral components were recorded from both left and right dioctants, being maximal over O3 and O4, see fig. 8.17. P100 was maximal over the ipsilateral hemisphere with a slightly more anterior distribution after left field stimulation. When compared with the full half field responses the distributions were more lateralised after stimulation with the dioctant stimuli. On lower dioctant stimulation the response from the anterior region of the montage appears to be split by a negativity, at a similar latency to the major positivity, into a positive-negative-positive complex, both the positivities being maximal over the anterior montage.

8.4.3.ic Subject GB (N-O and O dioctant)

No contralongitudinal components were observed with the lower N-O dioctant, however an early posterior positive was observed with the other dioctant (O), see fig.8.18. Contralateral components were recorded with both the N-O and O dioctants for stimuli in the right field. In contrast no contralateral components were recorded with the left O dioctant. A low amplitude posterior positivity was recorded with the upper dioctant (O), in contrast a large anterior late positivity was observed with the N-O dioctant.

8.4.3.id Subject MB (N-O and O dioctant)

No contralongitudinal components were again observed with the lower N-O octant, in contrast with the lower O dioctant there was a low amplitude late positivity, see fig.8.19. Contralateral components were observed with all dioctant

stimuli when positioned in the lateral half fields. The response after stimulation with the N-O upper dioctant was a low amplitude anterior positivity, in contrast a large amplitude posterior positivity was produced after stimulation with the upper O dioctant.

8.4.3.ie Subject EW (N-O and O dioctant)

The contralateral components appeared to be of a larger amplitude with the O dioctant when placed in the lateral half fields. With the lower dioctant O there was a late positivity over the posterior region of the montage, in contrast with the N-O dioctant the positivity was anterior see fig.8.20. On upper O dioctant stimulation there appear to be contralongitudinal components over the anterior region of the montage, these were replaced by an anterior negative and contralongitudinal components over the posterior region when the N-O stimulus was used.

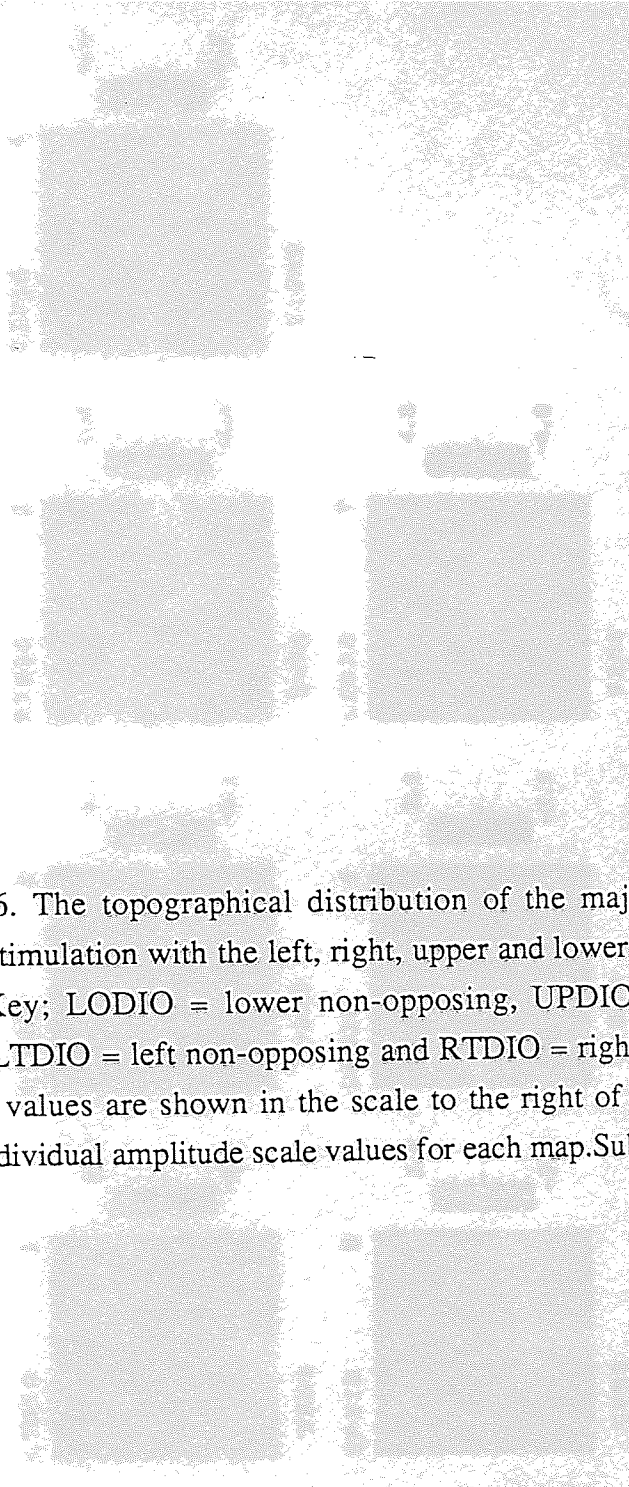
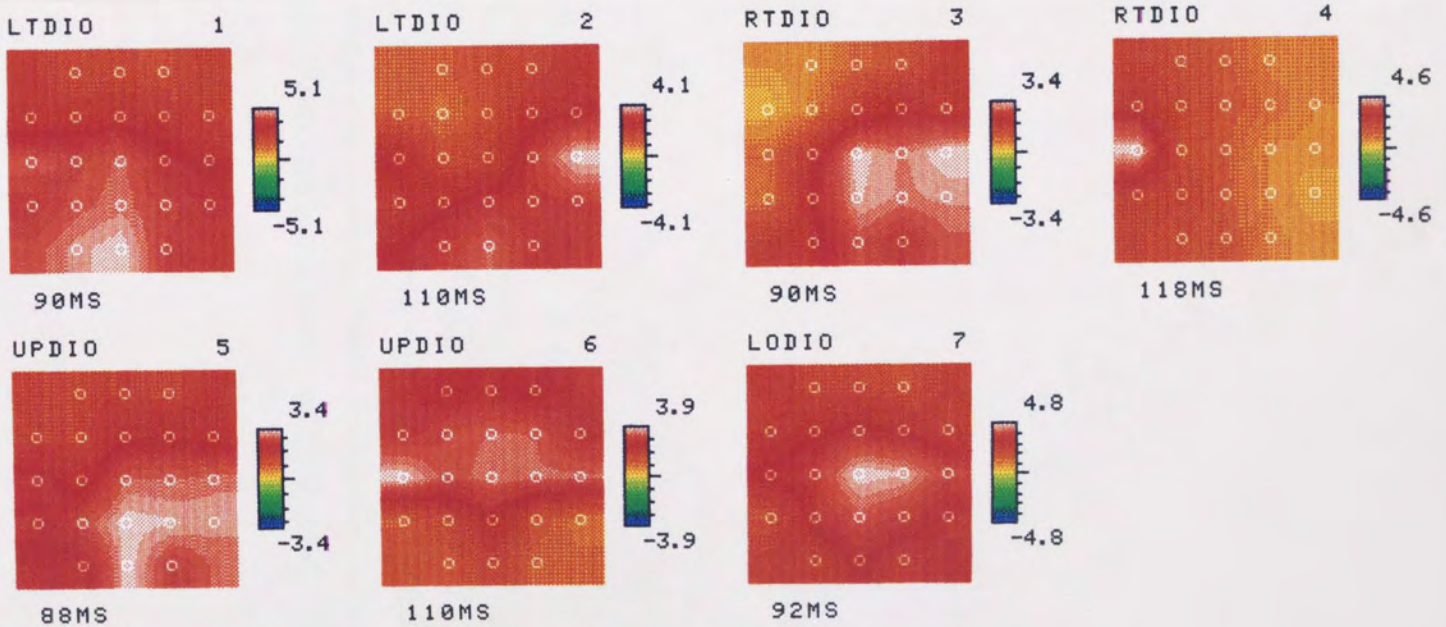


Figure 8.16. The topographical distribution of the major components following stimulation with the left, right, upper and lower non - opposing stimuli. Key; LODIO = lower non-opposing, UPDIO = upper non-opposing, LTDIO = left non-opposing and RTDIO = right non-opposing. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.Subject R.D.


LTDIO 1
5.1
-5.1
90MS
LTDIO 2
4.1
-4.1
110MS
RTDIO 3
3.4
-3.4
90MS
RTDIO 4
4.6
-4.6
118MS
UPDIO 5
3.4
-3.4
88MS
UPDIO 6
3.9
-3.9
110MS
LODIO 7
4.8
-4.8
92MS

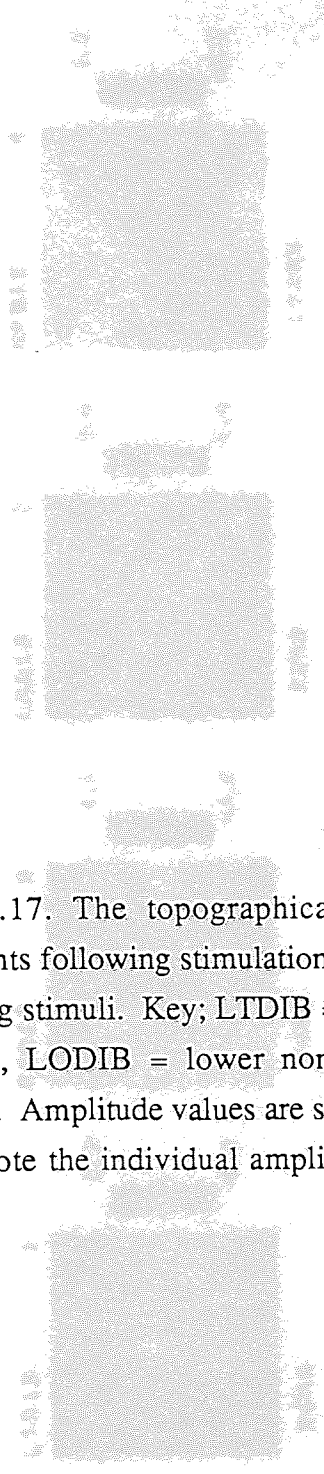


Figure 8.17. The topographical distribution of the major positive components following stimulation with the left, right, upper and lower non - opposing stimuli. Key; LTDIB = left non-opposing, RTDIB = right non-opposing, LODIB = lower non-opposing and UPDIB = upper non-opposing. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject MDH.

LTDIB
1
5.9
-5.9
96MS
RTDIB
2
4.6
-4.6
94MS
LODIB
3
5.8
-5.8
92MS
UPDIB
4
6.5
-6.5
112MS

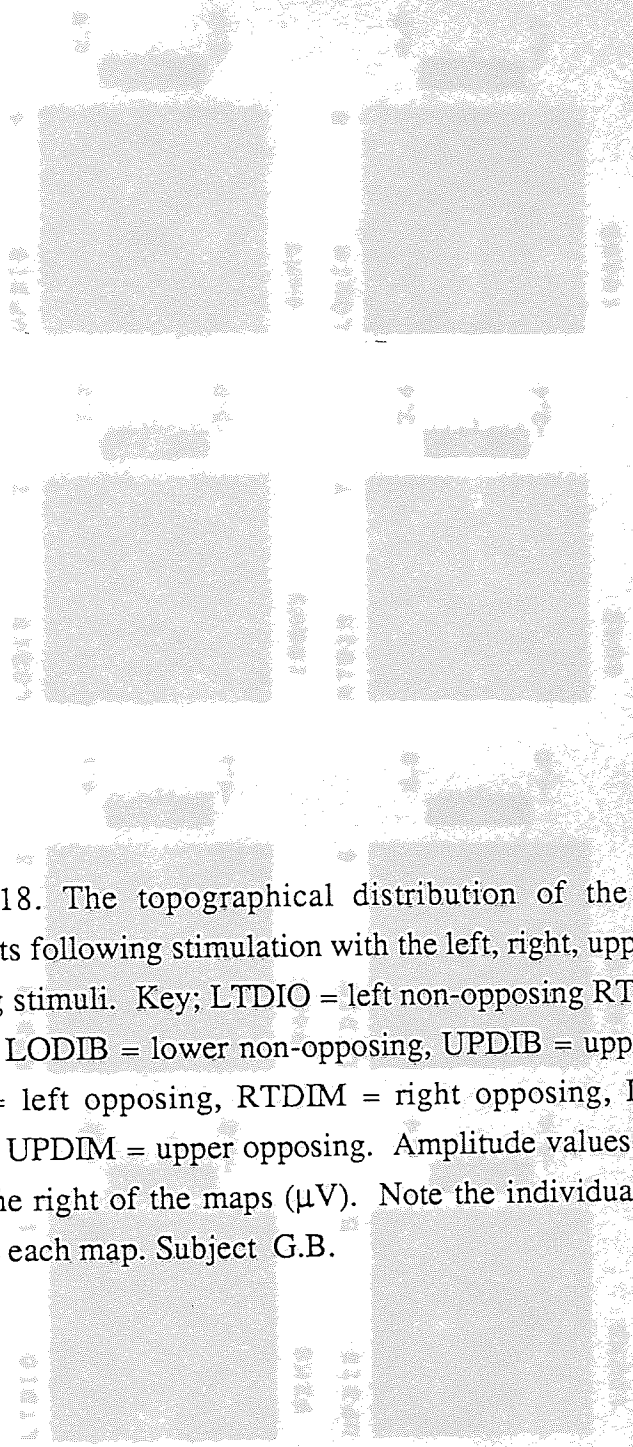


Figure 8.18. The topographical distribution of the major positive components following stimulation with the left, right, upper and lower non - opposing stimuli. Key; LTDIO = left non-opposing RTDIB = right non-opposing, LODIB = lower non-opposing, UPDIB = upper non-opposing, LTDIM = left opposing, RTDIM = right opposing, LODIM = lower opposing, UPDIM = upper opposing. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject G.B.

LTDIO 1
4.2
-4.2
92MS
RTDIB 2
4.1
-4.1
94MS
LODIB 3
3.9
-3.9
100MS
UPDIB 4
2.8
-2.8
84MS
UPDIB 5
4.4
-4.4
102MS
LTDIM 6
4.8
-4.8
108MS
RTDIM 7
3.4
-3.4
88MS
LODIM 8
4.7
-4.7
104MS
UPDIM 9
4.7
-4.7
100MS

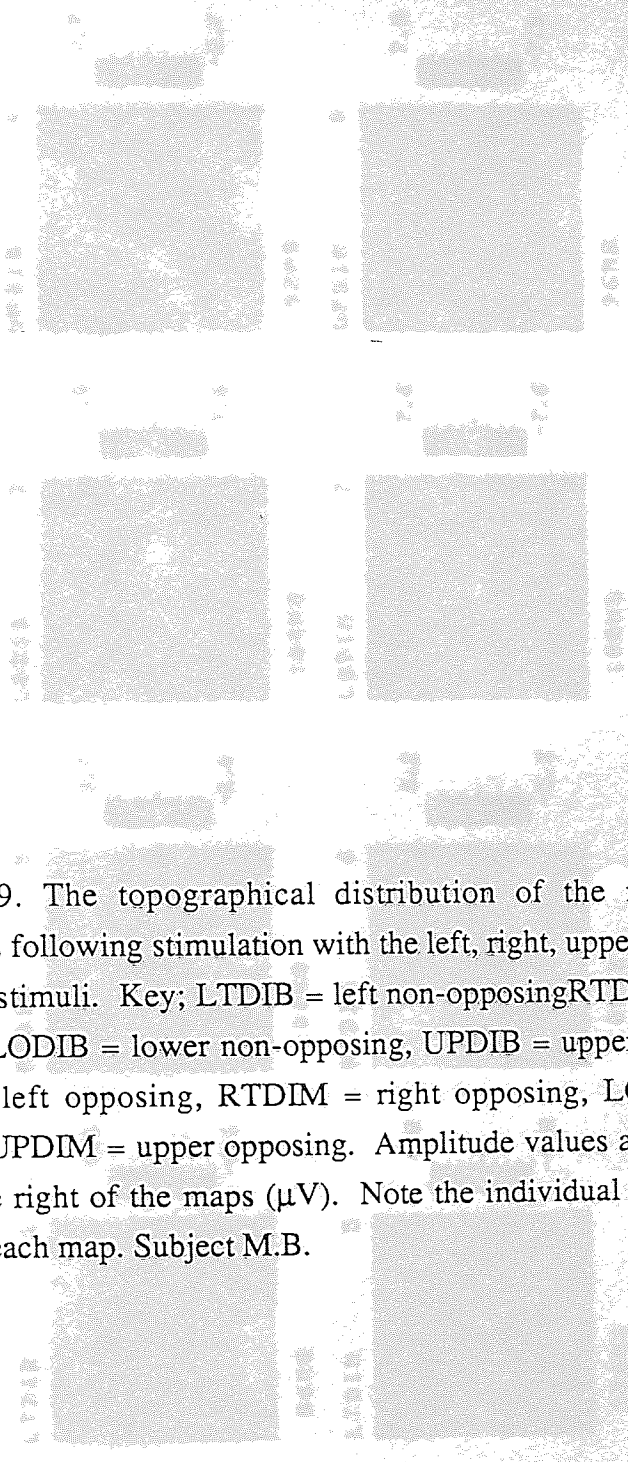


Figure 8.19. The topographical distribution of the major positive components following stimulation with the left, right, upper and lower non - opposing stimuli. Key; LTDIB = left non-opposingRTDIB = right non-opposing, LODIB = lower non-opposing, UPDIB = upper non-opposing, LTDIM = left opposing, RTDIM = right opposing, LODIM = lower opposing, UPDIM = upper opposing. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject M.B.

LTDIB 1
6.1
-6.1
96MS
RTDIB 2
2.6
-2.6
108MS
LODIB 3
7.6
-7.6
100MS
UPDIB 4
2.7
-2.7
92MS
LTDIM 5
4.0
-4.0
106MS
RTDIM 6
5.2
-5.2
108MS
LODIM 7
7.6
-7.6
100MS
UPDIM 8
7.9
-7.9
96MS
UPDIM 9
5.3
-5.3
132MS

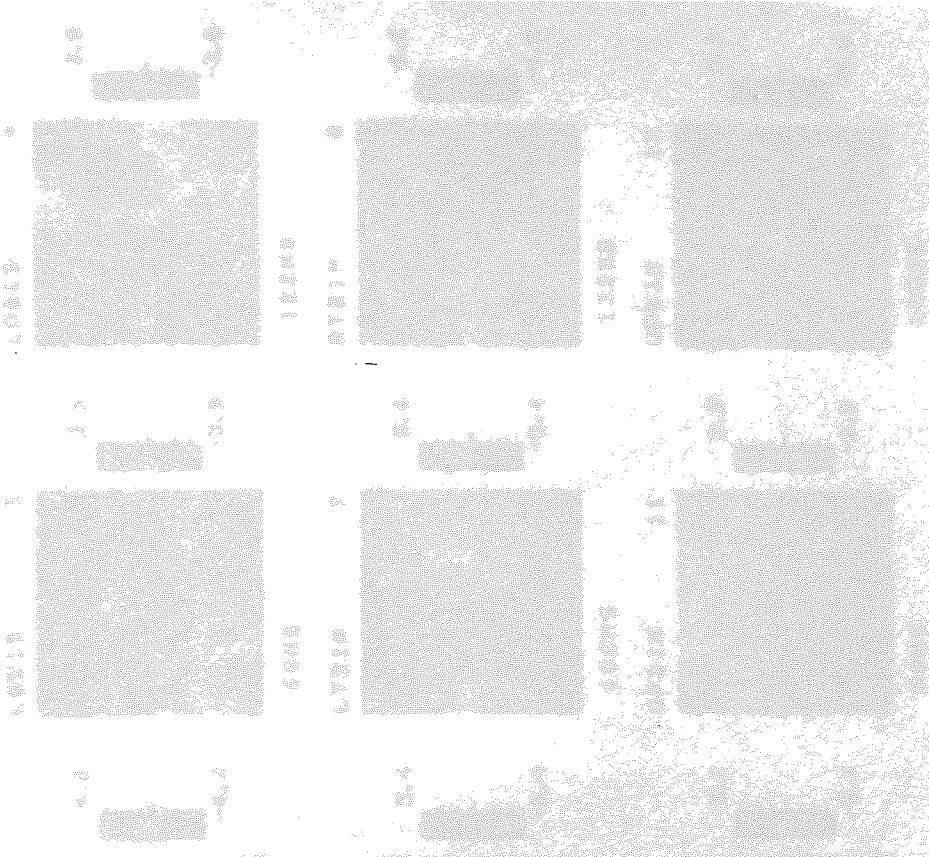


Figure 8.20. The topographical distribution of the major positive components following stimulation with the left, right, upper and lower non -opposing stimuli. Key; LTDIO = left non-opposingRTDIB = right non-opposing, LODIB = lower non-opposing, UPDIB = upper non-opposing, LTDIM = left opposing, RTDIM = right opposing, LODIM = lower opposing, UPDIM = upper opposing. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject E.W.

LTDIB 1 5.6 -5.6 106MS
RTDIB 2 4.2 -4.2 110MS
LODIB 3 3.9 -3.9 68MS
LODIB 4 3.0 -3.0 122MS
UPDIB 5 3.9 -3.9 76MS
UPDIB 6 5.4 -5.4 126MS
LTDIM 7 5.4 -5.4 106MS
RTDIM 8 4.2 -4.2 132MS
LODIM 9 4.2 -4.2 102MS
UPDIM 10 3.7 -3.7 82MS
UPDIM 11 2.9 -2.9 98MS
UPDIM 12 5.9 -5.9 128MS

8.4.4 Discussion

8.4.4.i Distributions Of Group Averaged Waveforms

8.4.4.ia Lateral Dioctants

The group mean waveforms and topographical distributions are shown in figures 8.21-24. The P100 appears to be more distinct when stimulating with the N-O stimulus for both left and right field stimulation. In the right half field the contralateral components spread over the ipsilateral hemisphere with the O dioctant, thus masking the P100 response. The contralateral components were more prominent on left field stimulation, both for the O and N-O dioctants. The early positivity was greatest following N-O octant stimulation, with only a vague negativity, see fig 8.29. The P120 component was more localised with the right N-O stimulus when compared with the O stimulus, no late positivity was shown in the left O response. An N145 was present in the group mean waveforms after N-O dioctant stimulation only, the distrbution was maximal ipsilaterally, as shown for the P100

8.4.4.ib Upper Field Stimulation

The group mean waveforms and topographical distributions are shown in figures 8.25 and 8.26. The distribution of the P100 response after upper O dioctant stimulation appears to be similar to that following full upper half field stimulation i.e. the maximum positivity was located over the posterior region of the montage. In contrast the maximum positivity after upper N-O dioctant stimulation was positioned over the anterior region. Typical contralongitudinal components consisting of a PNP complex were recorded over the posterior region of the montage with the N-O stimulus and over the anterior region with the O stimulus, see fig 8.29.

The distribution of these upper field responses would fit with a striate generator of the response and with the cruciform model of the striate cortex. This model would predict that the vertical octant dipoles which are projected ventrally onto the cortex may sum to produce a large amplitude response, in contrast the horizontal octant dipoles directed anteriorly, would not sum and may be atttenuated by the cortical structures as a result of signal generation being situated in the calcarine fissure.

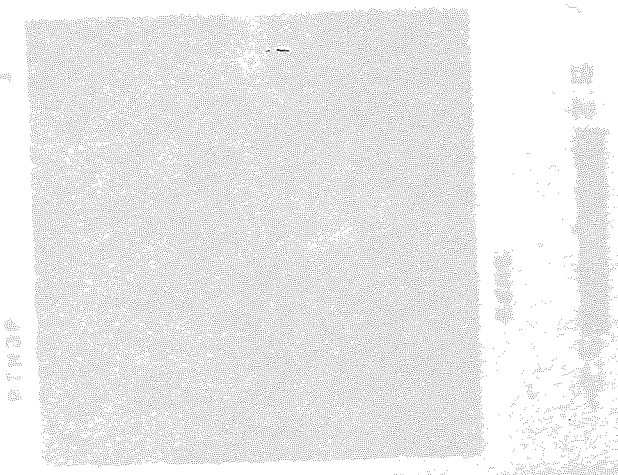
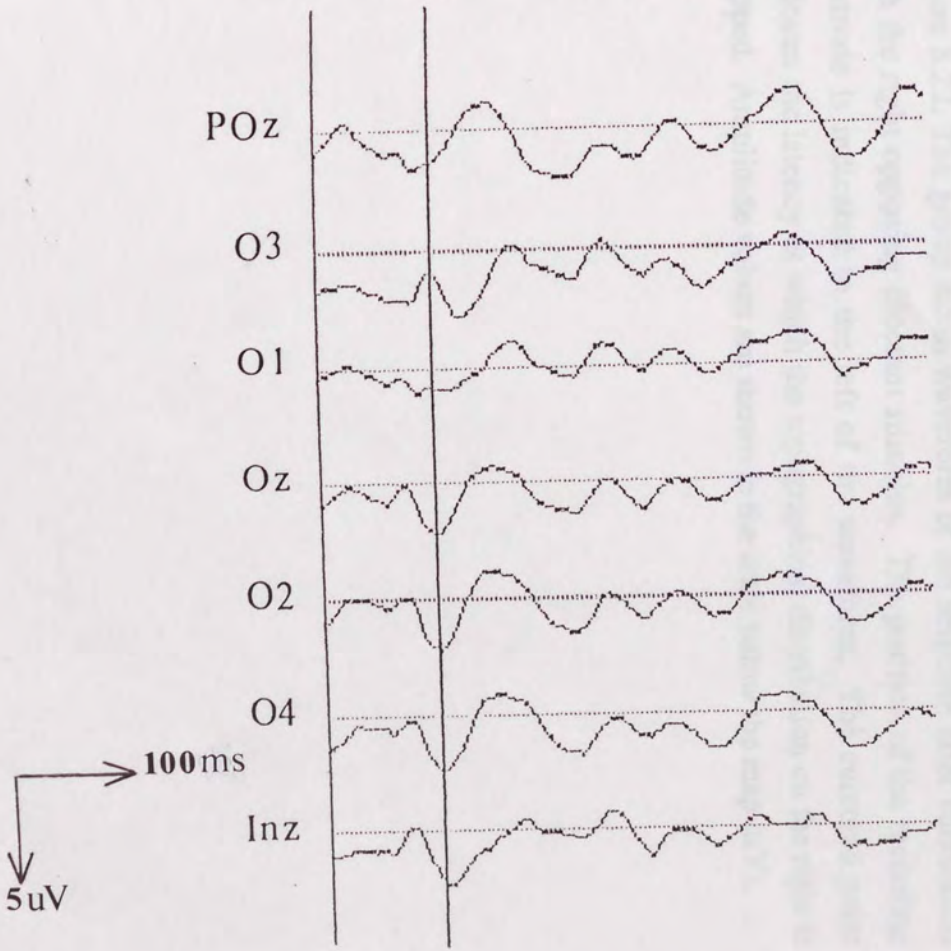
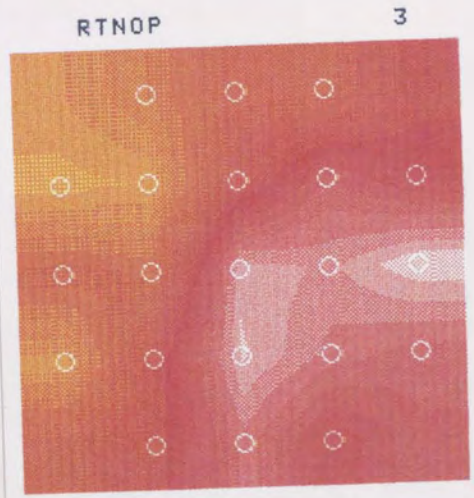


Figure 8.21. The group mean waveform of the response after stimulation with the right non-opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


POz
O3
O1
Oz
O2
O4
Inz
100ms
5uV


RTNOP
3
96MS
-2.5
2.5

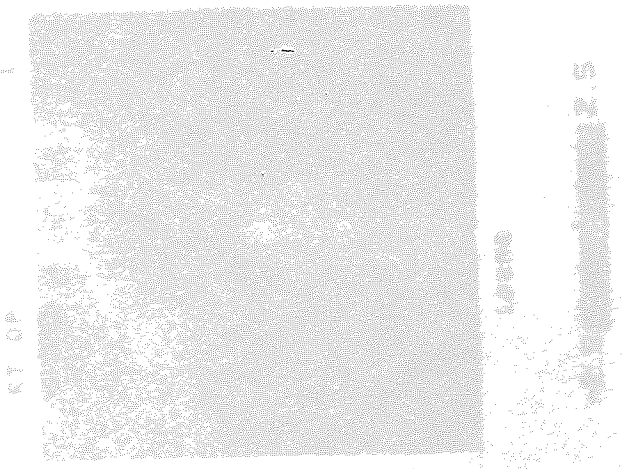
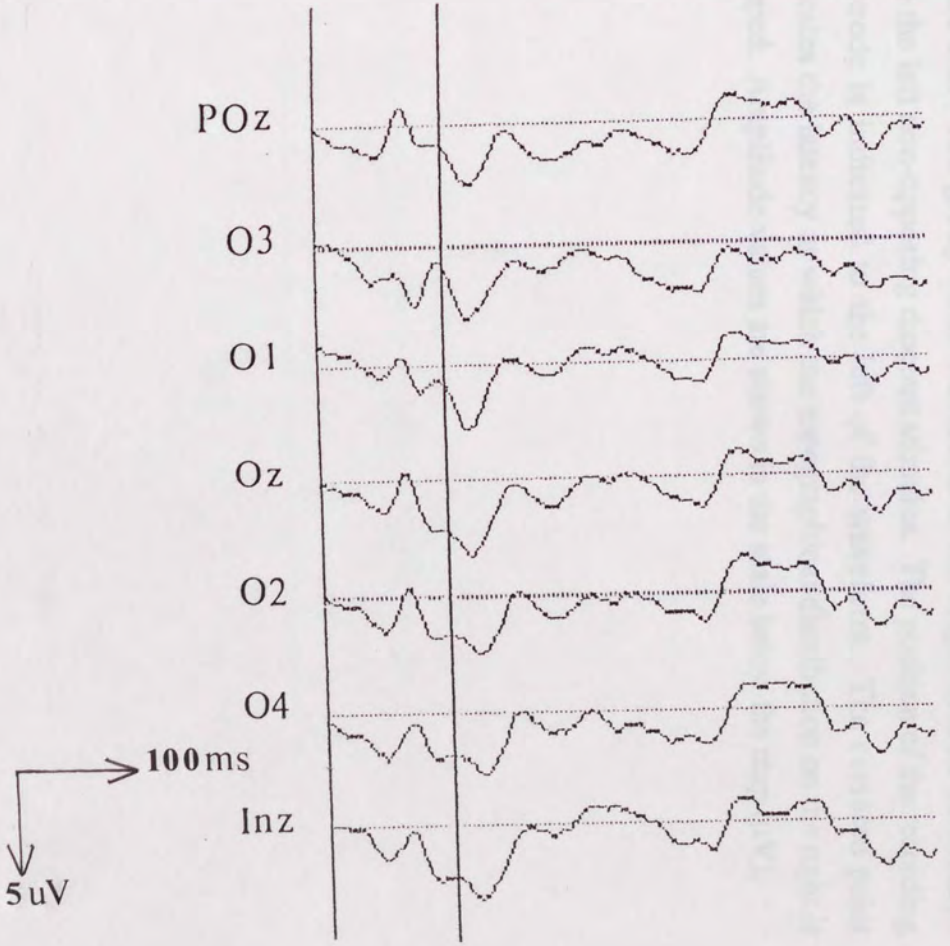


Figure 8.22. The group mean waveform of the response after stimulation with the right opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


POz
O3
O1
Oz
O2
O4
Inz
100ms
5uV

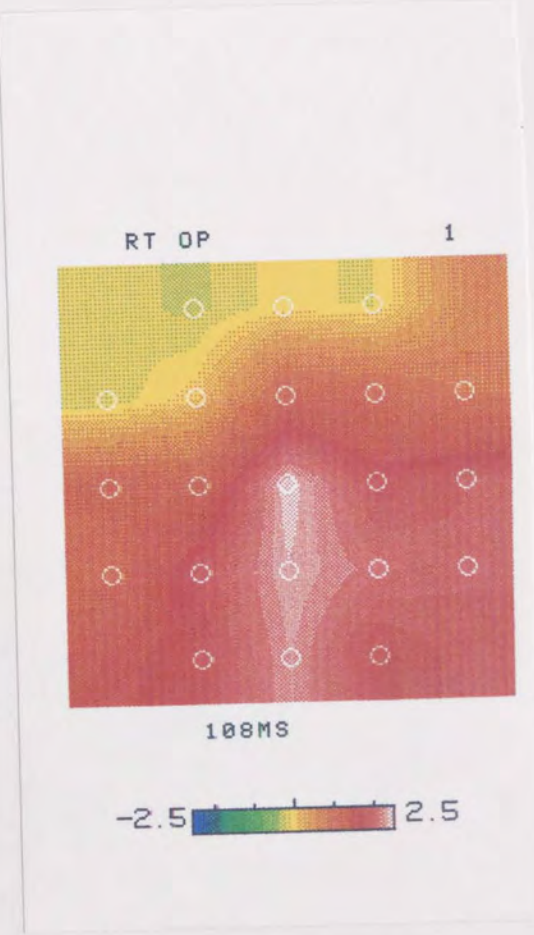
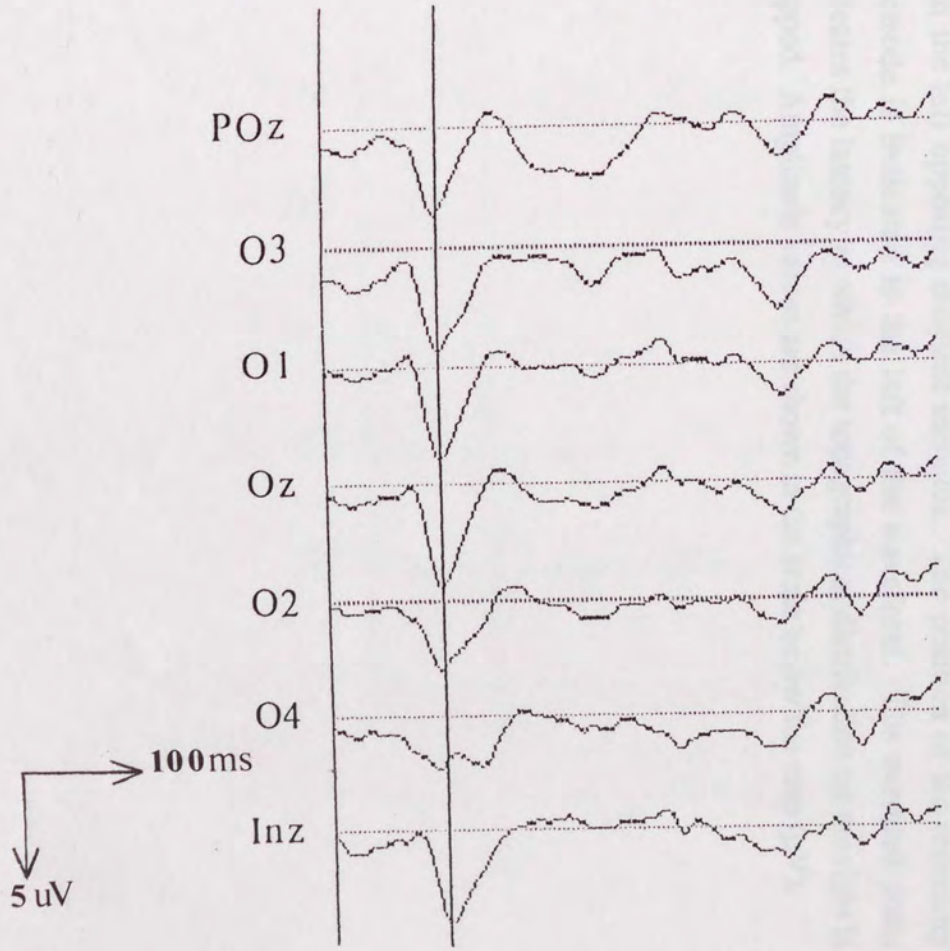
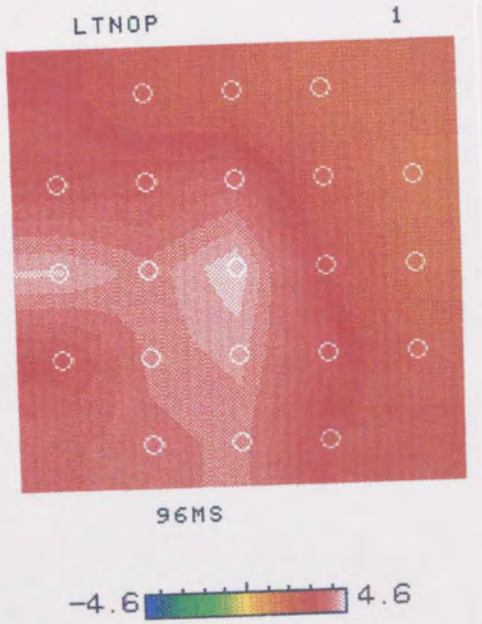

RT OP
1
108MS
-2.5
2.5

Figure 8.23. The group mean waveform of the response after stimulation with the left non-opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


POz
O3
O1
Oz
O2
O4
Inz
100ms
5uV


LTNOP
1
96MS
-4.6
4.6

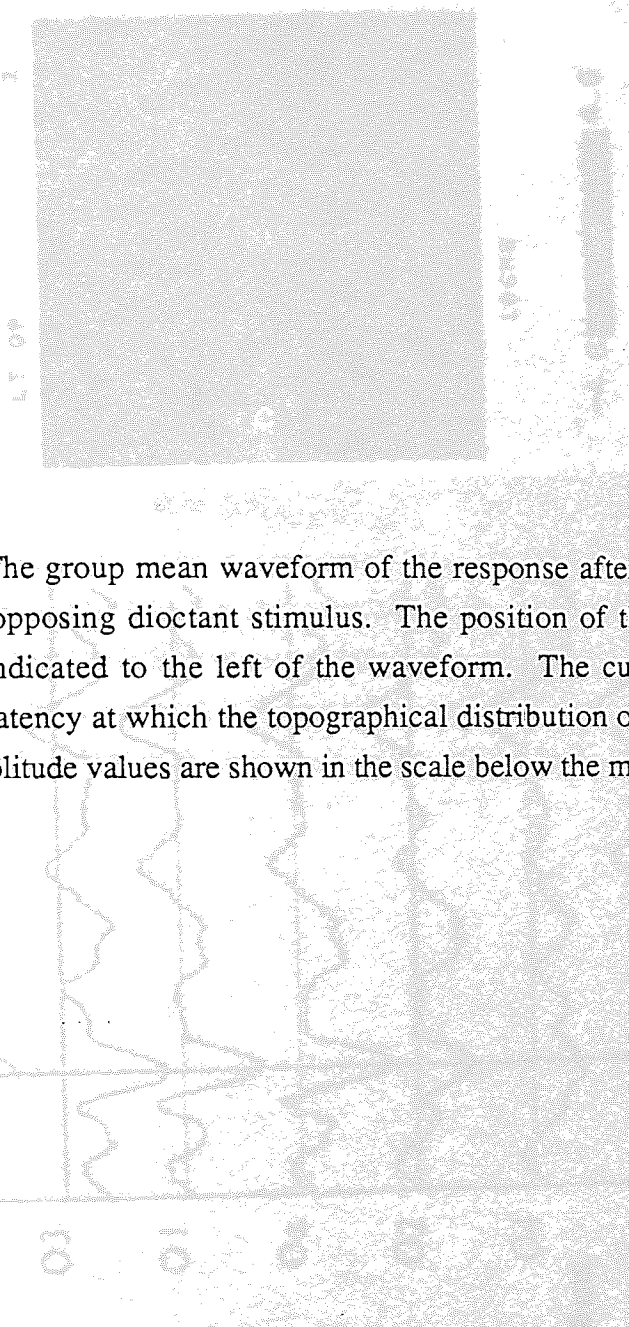
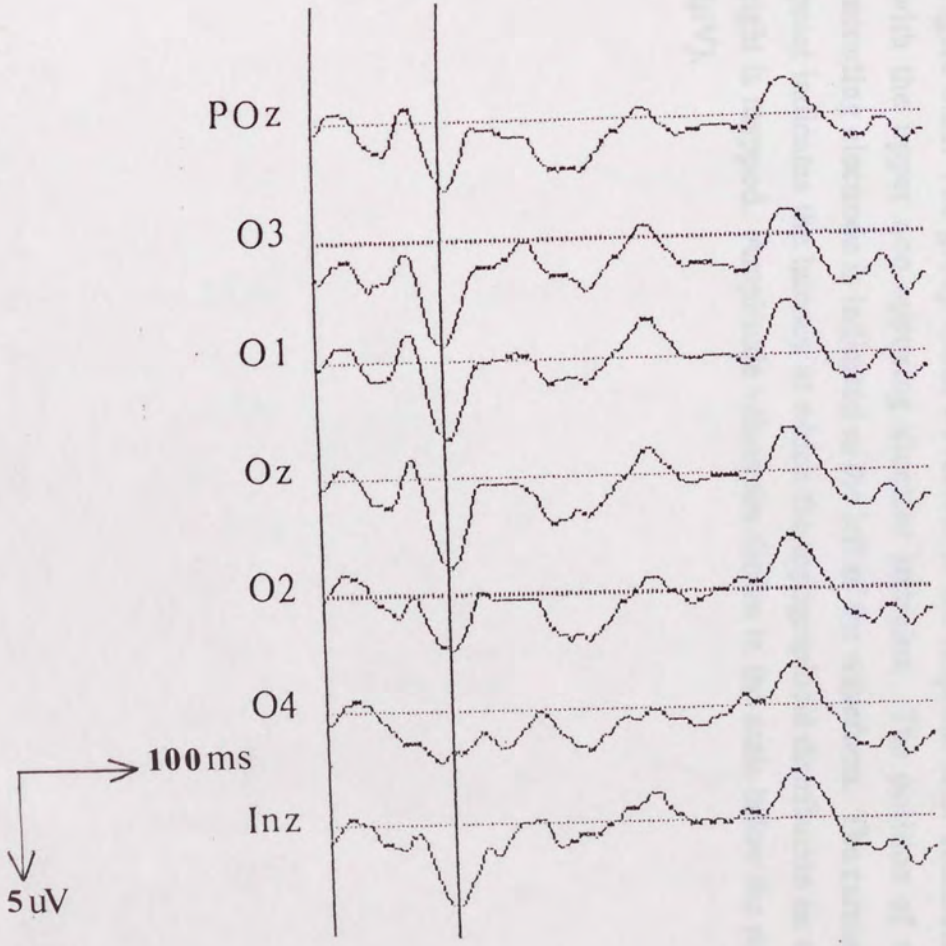


Figure 8.24. The group mean waveform of the response after stimulation with the left opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


POz
O3
O1
Oz
O2
O4
Inz
100 ms
5 uV

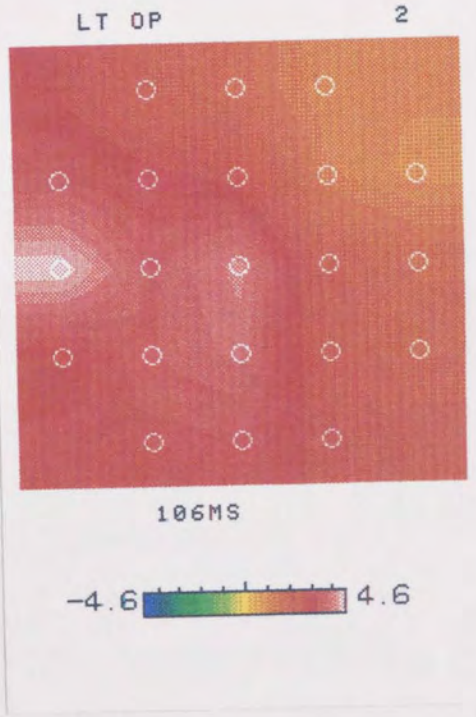
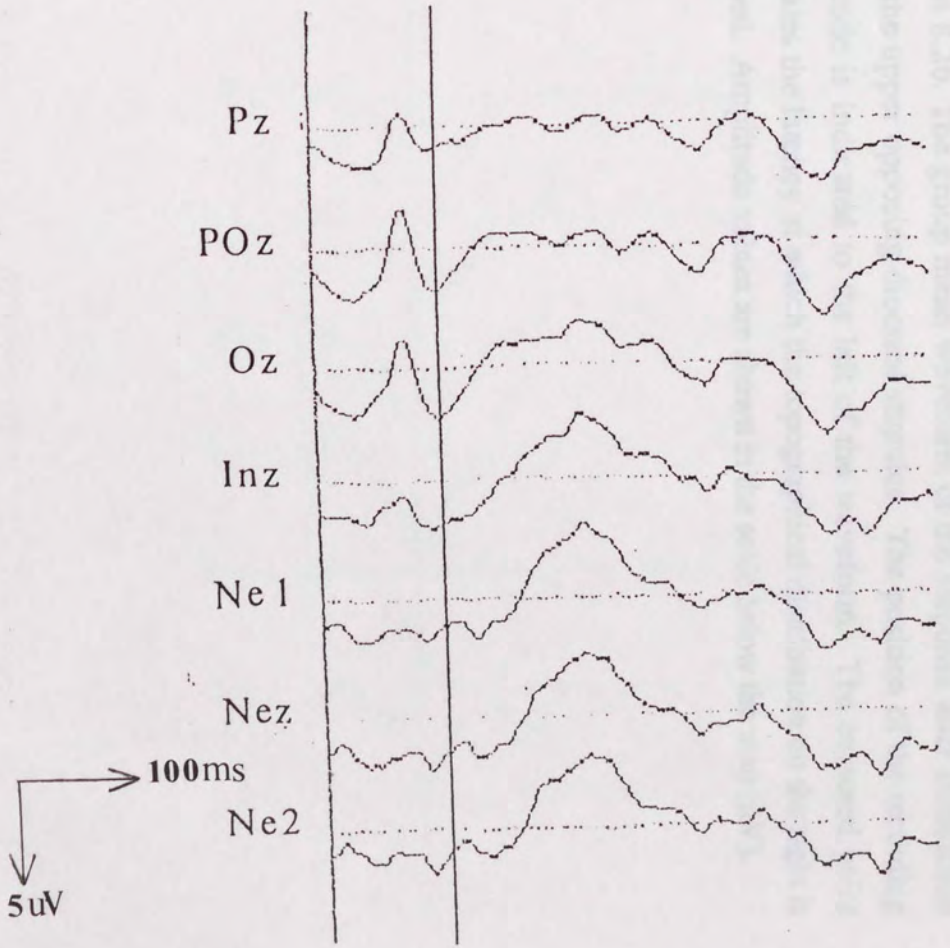
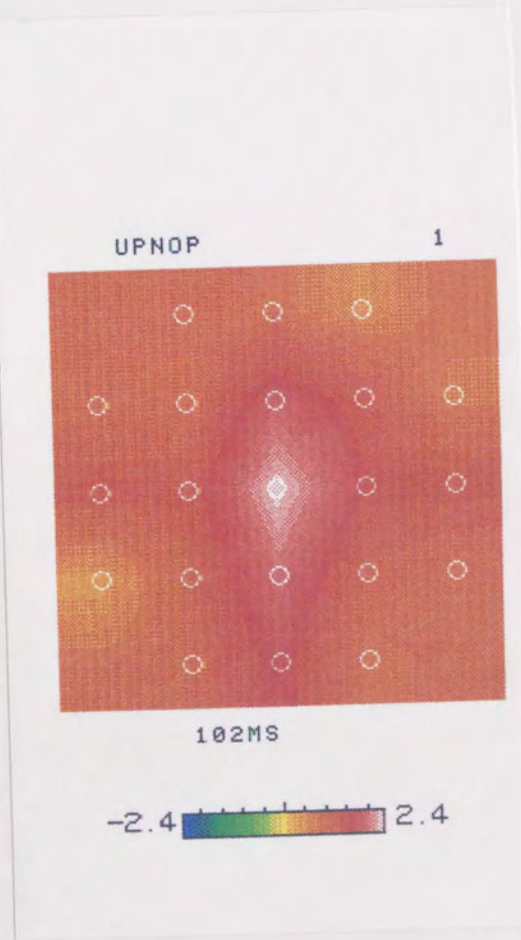

LT OP
2
106MS
-4.6
4.6

Figure 8.25. The group mean waveform of the response after stimulation with the upper non-opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


Pz
POz
Oz
Inz
Ne1
Nez
Ne2
100ms
5uV


UPNOP
1
102MS
-2.4
2.4

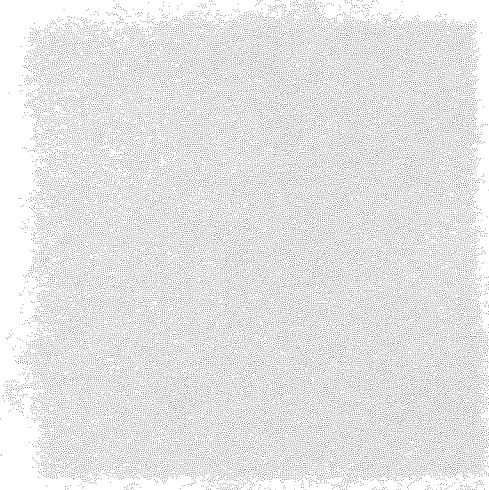
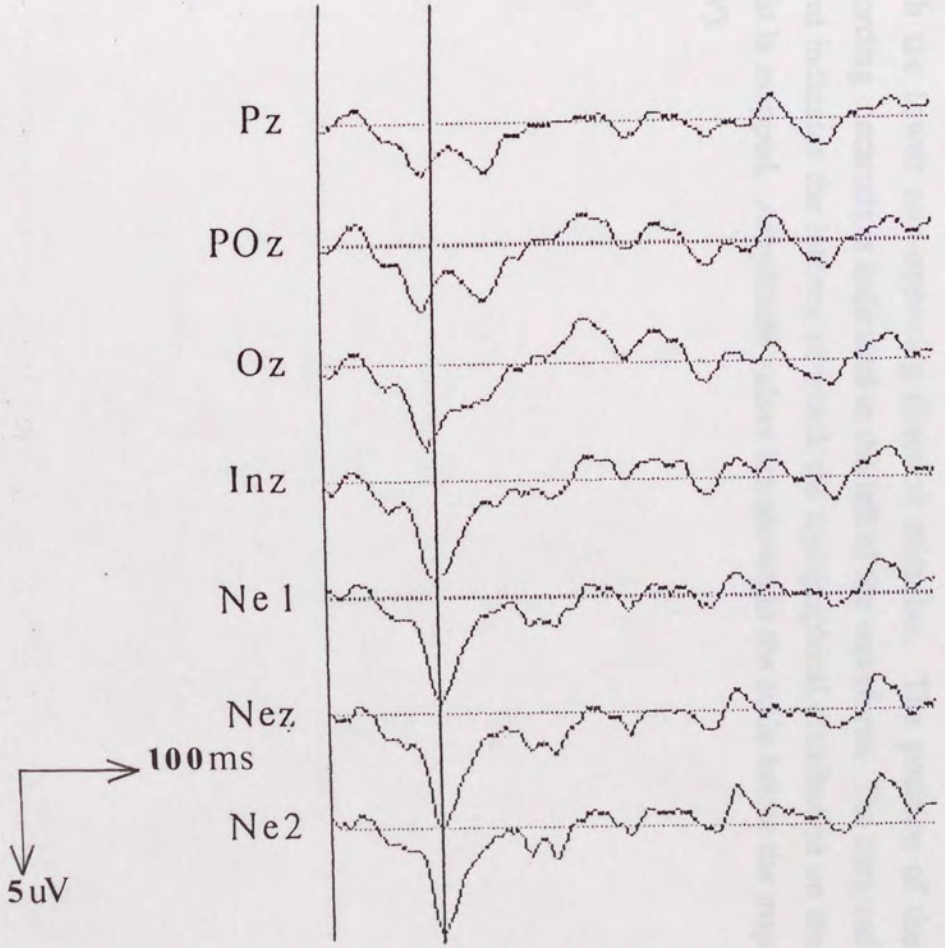


Figure 8.26. The group mean waveform of the response after stimulation with the upper opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


Pz
POz
Oz
Inz
Ne1
Nez
Ne2
100ms
5uV

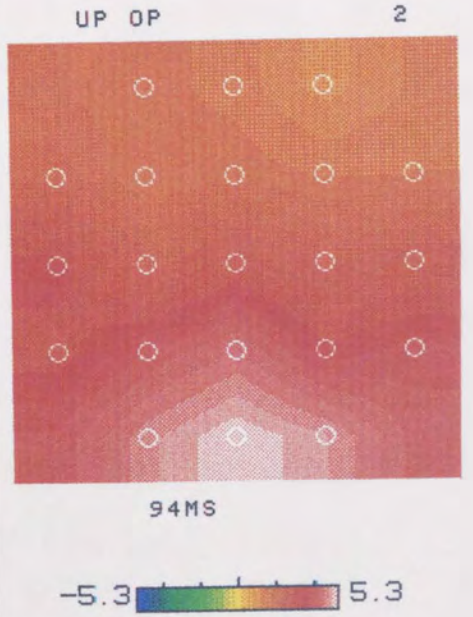
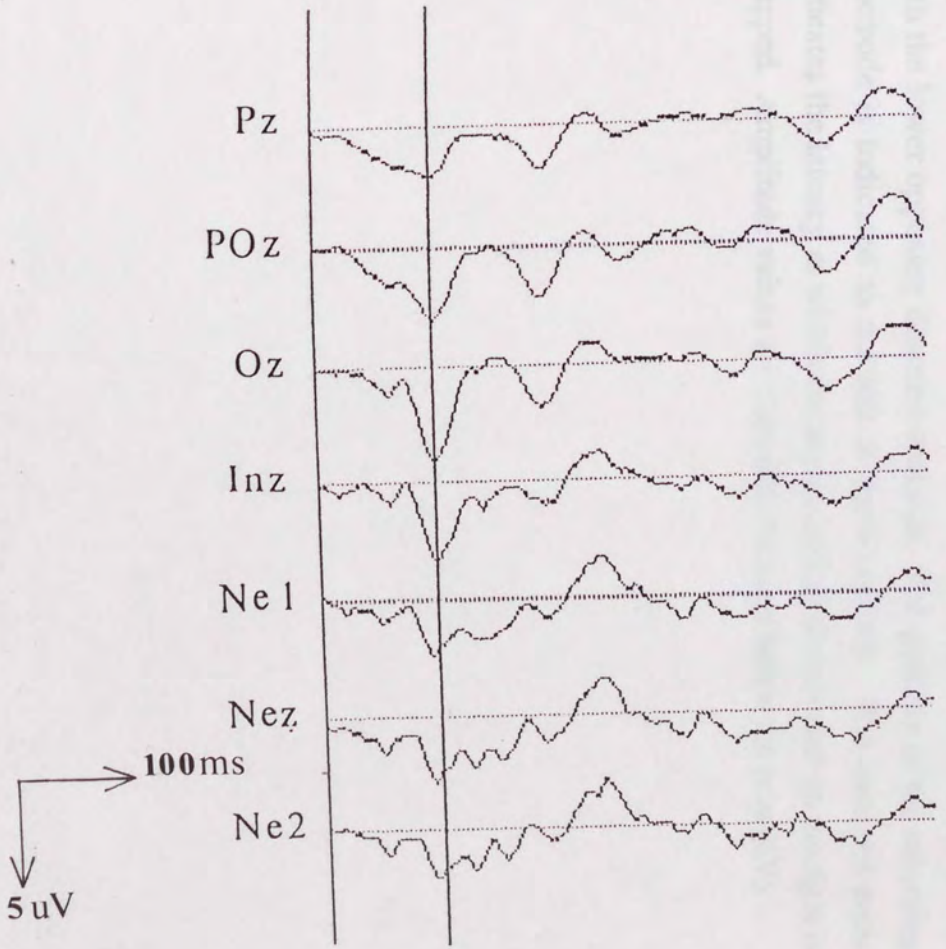
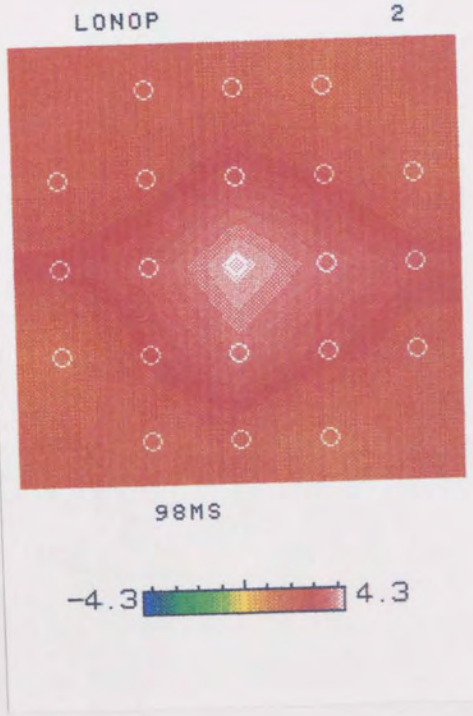

UP OP
2
94MS
-5.3
5.3

Figure 8.27. The group mean waveform of the response after stimulation with the lower non-opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


Pz
POz
Oz
Inz
Ne1
Nez
Ne2
100ms
5uV


LONOP
2
98MS
-4.3
4.3

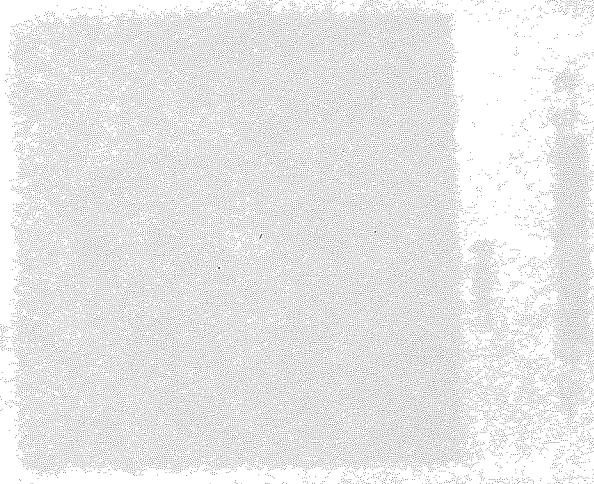
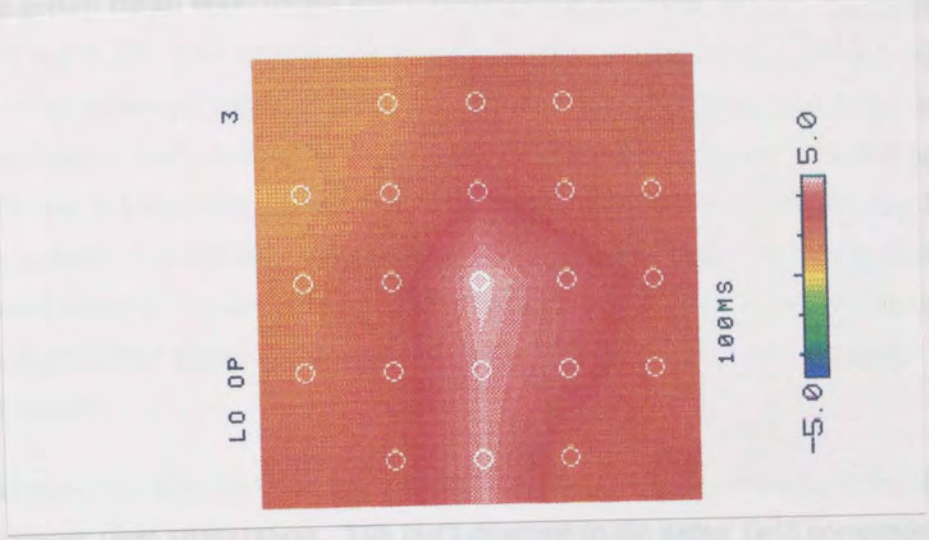
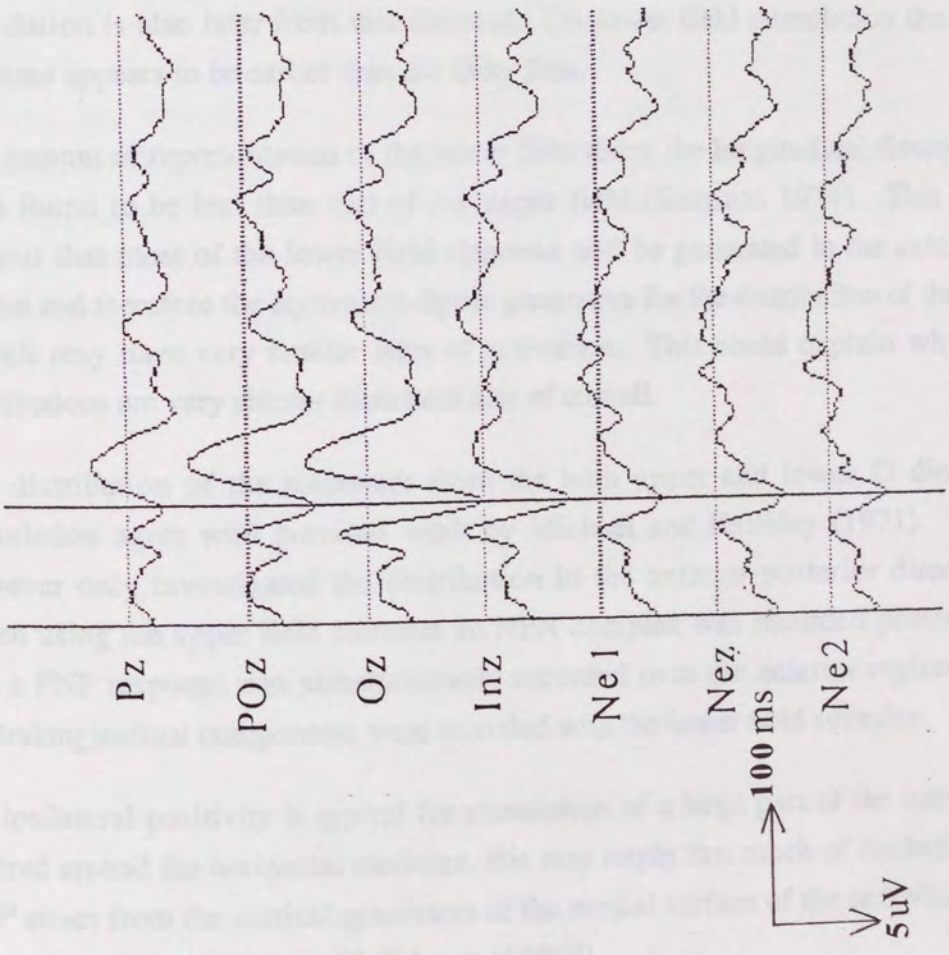


Figure 8.28. The group mean waveform of the response after stimulation with the lower opposing dioctant stimulus. The position of the recording electrode is indicated to the left of the waveform. The cursored point indicates the latency at which the topographical distribution on the right is mapped. Amplitude values are shown in the scale below the map (μV).


3
LO OP
100MS
5.0
-5.0


Pz
POz
Oz
Inz
Ne1
Nez
Ne2
100 ms
5uV

8.4.4.ic Lower Field Stimulation

The group mean waveforms and topographical distributions are shown in figures 8.27 and 8.28. The largest amplitude positive response was observed with the O dioctant stimulus, the distribution was however slightly skewed to the left. The distribution was more central, maximum over Oz, with the N-O dioctant. The N75 and N145 components were also larger with the O stimulus, see fig 8.29. No contralongitudinal components were present with either stimulus. As a consequence of the lack of contralongitudinal components it may be suggested that the equivalent dipoles generating the lower field response are more radially orientated.

The response after N-O dioctant stimulation was later than that from the O dioctant for upper field stimulation. The N-O dioctant in the upper field corresponds to the O dioctant stimuli for lateral half field stimulation, the response after lateral field stimulation is also later from this dioctant. On lower field stimulation the N-O response appears to be earlier than the O by 2ms.

The amount of representation of the lower field along the longitudinal fissure has been found to be less than that of the upper field (Stensaas 1974). This may suggest that most of the lower field response will be generated in the calcarine fissure and therefore the equivalent dipole generators for the distribution of the two stimuli may have very similar sites of activation. This could explain why the distributions are very similar from both sets of stimuli.

The distribution of the responses from the both upper and lower O dioctant stimulation agree with previous work by Michael and Halliday (1971). They however only investigated the distribution in the anterior-posterior direction. When using the upper field stimulus an NPN complex was recorded posteriorly and a PNP response was simultaneously recorded over the anterior region. No contralongitudinal components were recorded with the lower field stimulus.

An ipsilateral positivity is typical for stimulation of a large part of the half field centred around the horizontal meridian, this may imply that much of the half field VEP arises from the cortical generators of the medial surface of the occipital lobe rather than on the convexity (Halliday et al 1977).

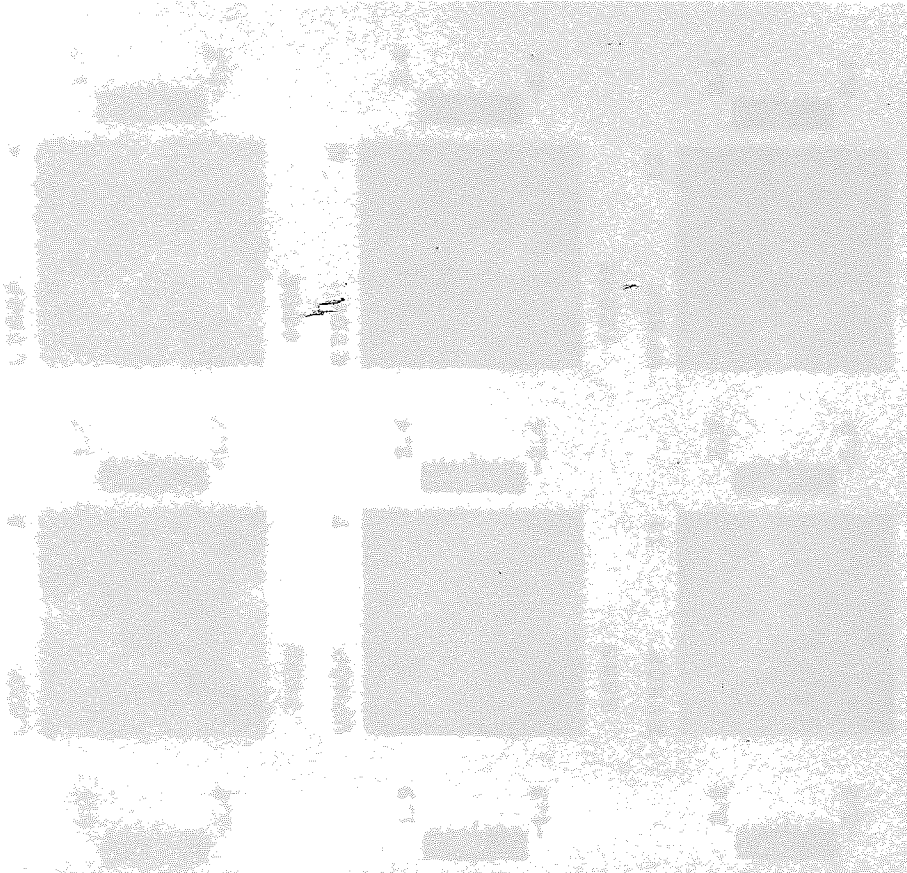
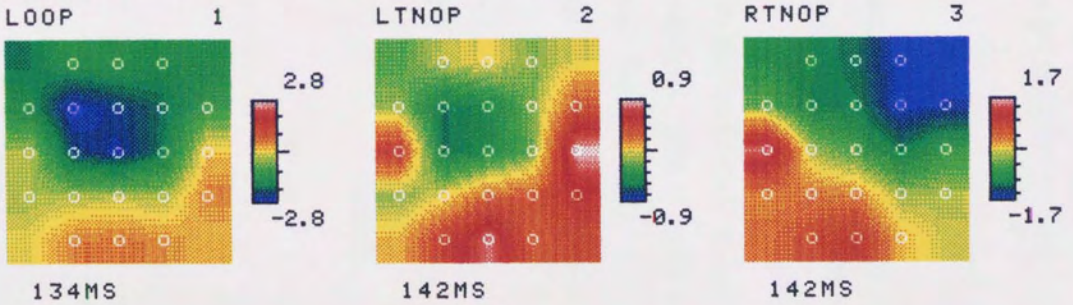


Figure 8.29. The topographical distribution, from the group mean waveforms, of the major components excluding the P100 following opposing and non-opposing dioctant stimulation. Key: LTOP = left opposing, RTOP = right opposing, LOOP = lower opposing, UPOP = upper opposing, LTNOP = left non-opposing, RTNOP = right non-opposing, UPNOP = upper non-opposing. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.

LTOP 1
1.4
-1.4
76MS
RTOP 2
1.6
-1.6
72MS
LOOP 3
1.7
-1.7
74MS
LTNOP 4
1.7
-1.7
68MS
RTNOP 5
2.2
-2.2
72MS
UPNOP 6
1.9
-1.9
72MS
UPNOP 7
2.4
-2.4
88MS
RTOP 8
3.4
-3.4
128MS
UPOP 9
2.4
-2.4
136MS
LTNOP 10
2.2
-2.2
126MS
RTNOP 11
2.9
-2.9
120MS
UPNOP 12
2.1
-2.1
122MS


LOOP
1
2.8
-2.8
134MS
LTNOP
2
0.9
-0.9
142MS
RTNOP
3
1.7
-1.7
142MS

8.5 Octant Stimulation

8.5.1 Introduction

The variation in distribution of the reponse could also be linked with the distribution of the response from the projections of the octants of the visual field on the cortex, as these are thought to project discretely to one area of the striate cortex and may be widely variable between the population.

8.5.2 Method

Six subjects participated in this study. Octant stimulation was produced with the octant being constituents of a full 20° field. Two subjects were stimulated with all eight octants (GB and RD), in the remaining four subjects the octants were only presented in the left half field. All subjects had visual acuities of 6/6 or better with correction if required. The check size was 27', as used previously. Fifty averages were performed for each stimulus with at least one repeat. The method of stimulation was the same as that used for previous studies.

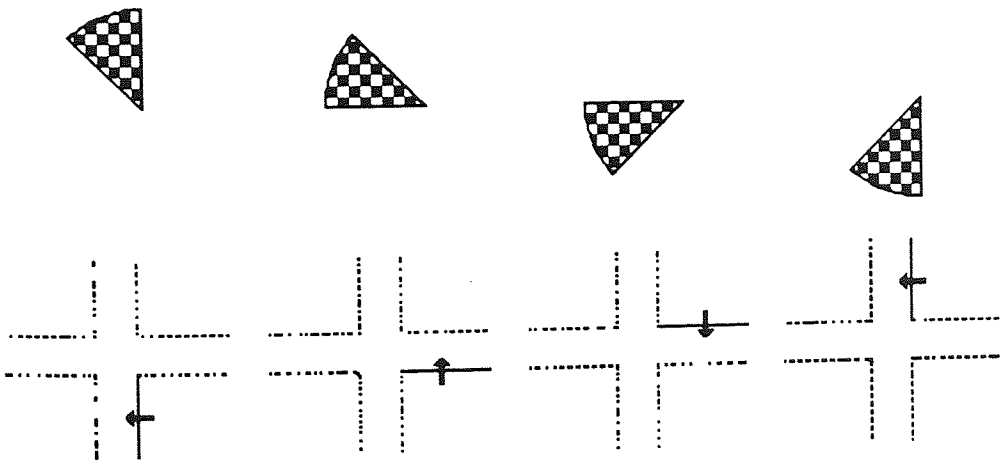


Fig. 8.30 The visual field location and cortical projection of the constituent octants of the left half field.

8.5.3 Results

Two of the subjects failed to give consistent responses, consequently the data is presented from four subjects (GB,RD,NE,NP).

8.5.3.i Individual Response Distributions

8.5.3.ia Subject RD

The early negativity was largest following upper octant stimulation although a low amplitude response was recorded after left lower horizontal octant stimulation. The following positivity was maximal over the centre of the montage with slightly varying lateralities for both lower octants, see fig 8.31. This peak was succeeded by a later positivity in the case of the vertical octants, this being maximal over the left with the right stimulus and over the posterior area with the left stimulus. The following negativity appeared to be maximal contralaterally. The response following upper horizontal stimulation appeared to be dominated by the contralateral positivity maximal over the O3/O4. An early posterior negativity was evident with right upper horizontal octant stimulation, this was replaced by a posterior positivity on left field stimulation. A contralateral anterior negativity was the last repeatable component. In contrast an early positivity was only recorded with left upper vertical stimulation. This was followed by a late anterior positivity and a contralateral negativity. The major positivity was positioned posteriorly when the stimulus was placed in the right field.

The domination of the upper horizontal octant response by the late contralateral positivity would fit with the distribution following upper dioctant stimulation in which the major positive response was maximal anteriorly.

8.5.3.ib Subject GB

The response following lower vertical octant stimuli in the right field was dominated by the early contralateral positivity, when positioned in the left field the positive response was maximal 16ms later. A low amplitude early negativity was also recorded. The major positivity following upper vertical octant stimulation was posterior with both left and right octants, see fig 8.32. For both upper and lower left horizontal octant stimulation the positive response was maximal ipsilaterally, the early negativity was larger with the upper stimulus whereas the later negativity was largest following lower field stimulation. An early contralateral positivity was recorded with right lower horizontal stimulation. Upper right horizontal octant stimulation also produced two positive peaks, the first was maximal posteriorly whereas the second larger component was maximal anteriorly.

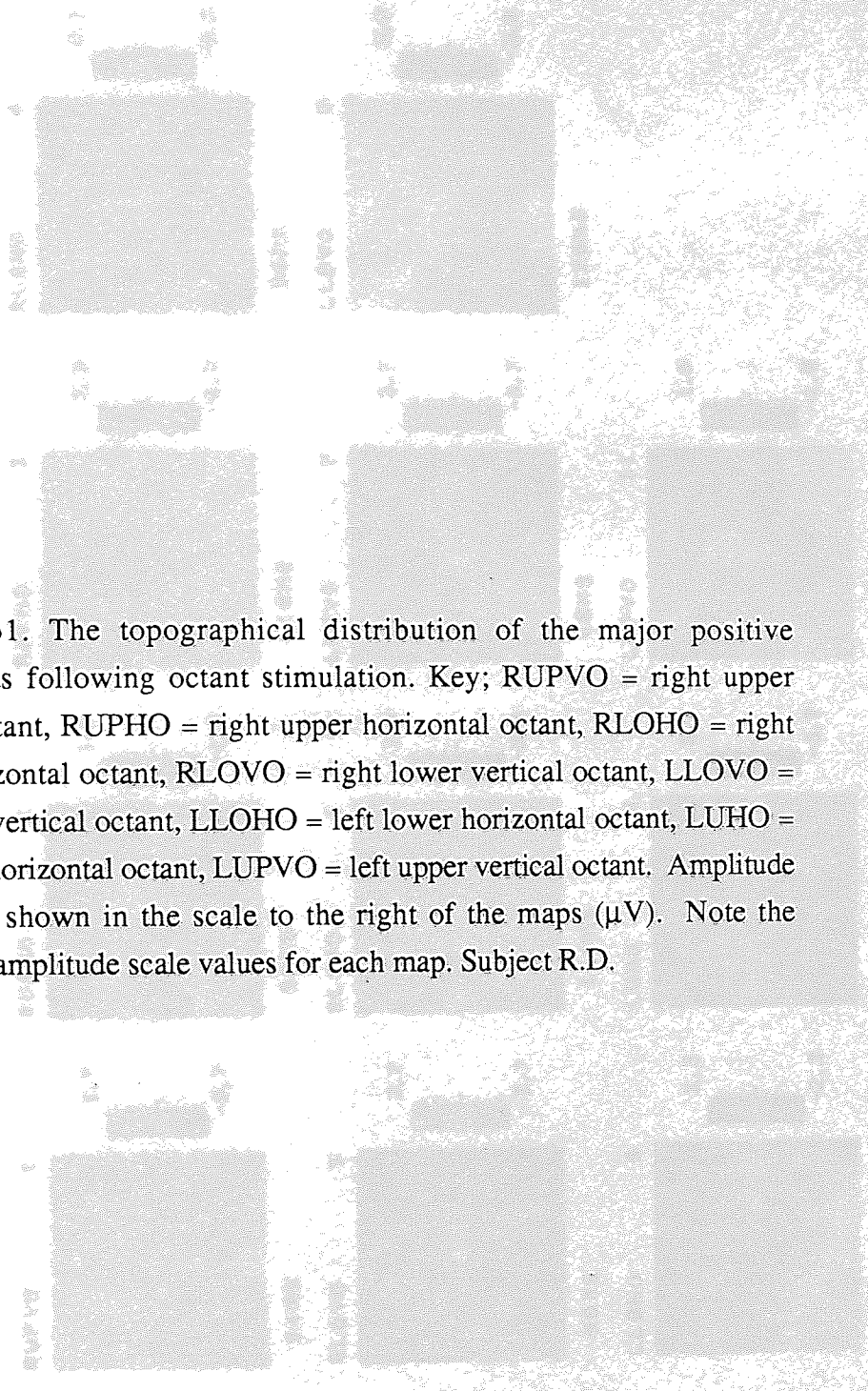
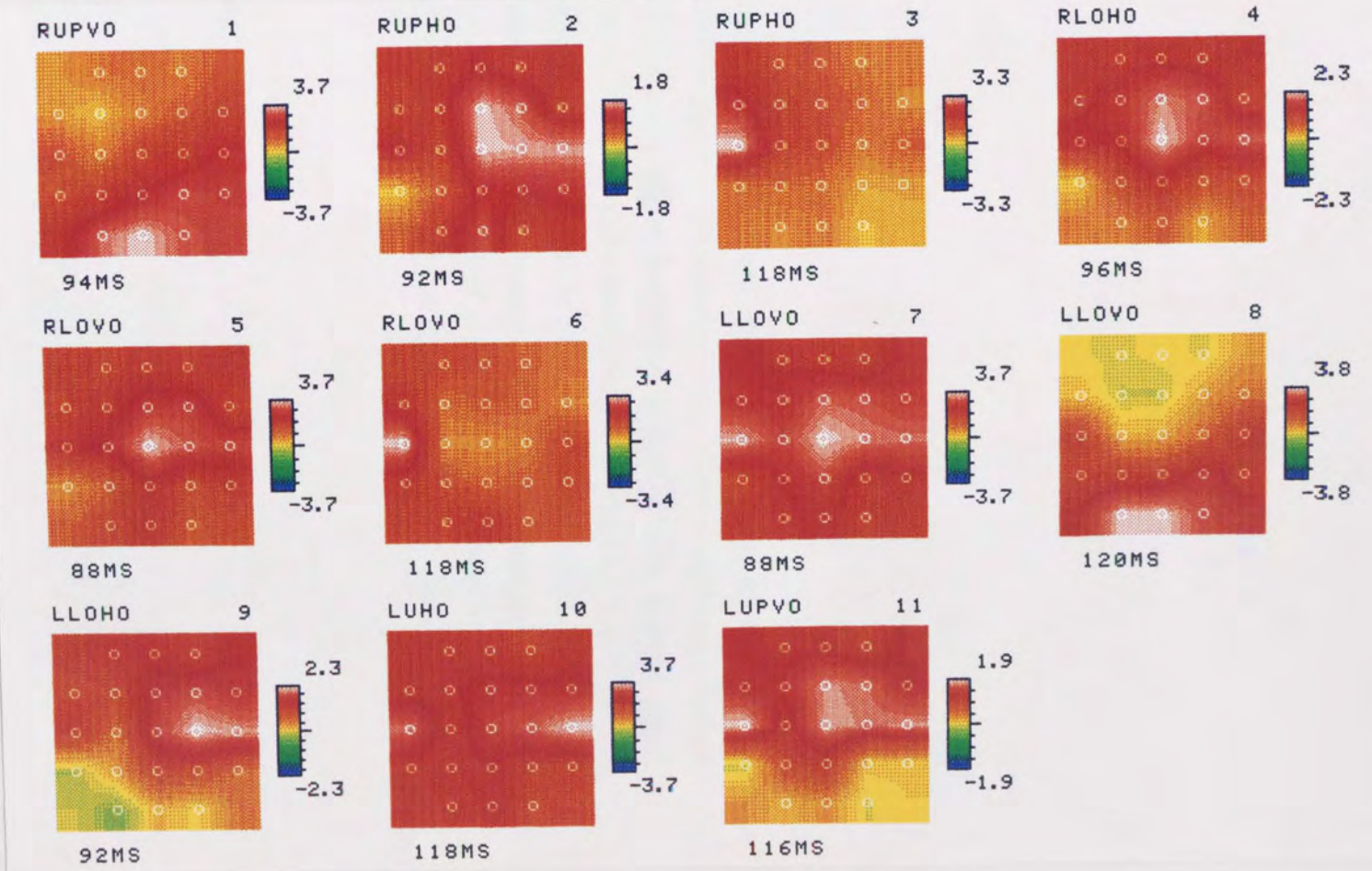


Figure 8.31. The topographical distribution of the major positive components following octant stimulation. Key; RUPVO = right upper vertical octant, RUPHO = right upper horizontal octant, RLOHO = right lower horizontal octant, RLOVO = right lower vertical octant, LLOVO = left lower vertical octant, LLOHO = left lower horizontal octant, LUHO = left upper horizontal octant, LUPVO = left upper vertical octant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject R.D.


RUPVO 1
3.7
-3.7
94MS
RUPHO 2
1.8
-1.8
92MS
RUPHO 3
3.3
-3.3
118MS
RLOHO 4
2.3
-2.3
96MS
RLOVO 5
3.7
-3.7
88MS
RLOVO 6
3.4
-3.4
118MS
LLOVO 7
3.7
-3.7
88MS
LLOVO 8
3.8
-3.8
120MS
LLOHO 9
2.3
-2.3
92MS
LUHO 10
3.7
-3.7
118MS
LUPVO 11
1.9
-1.9
116MS

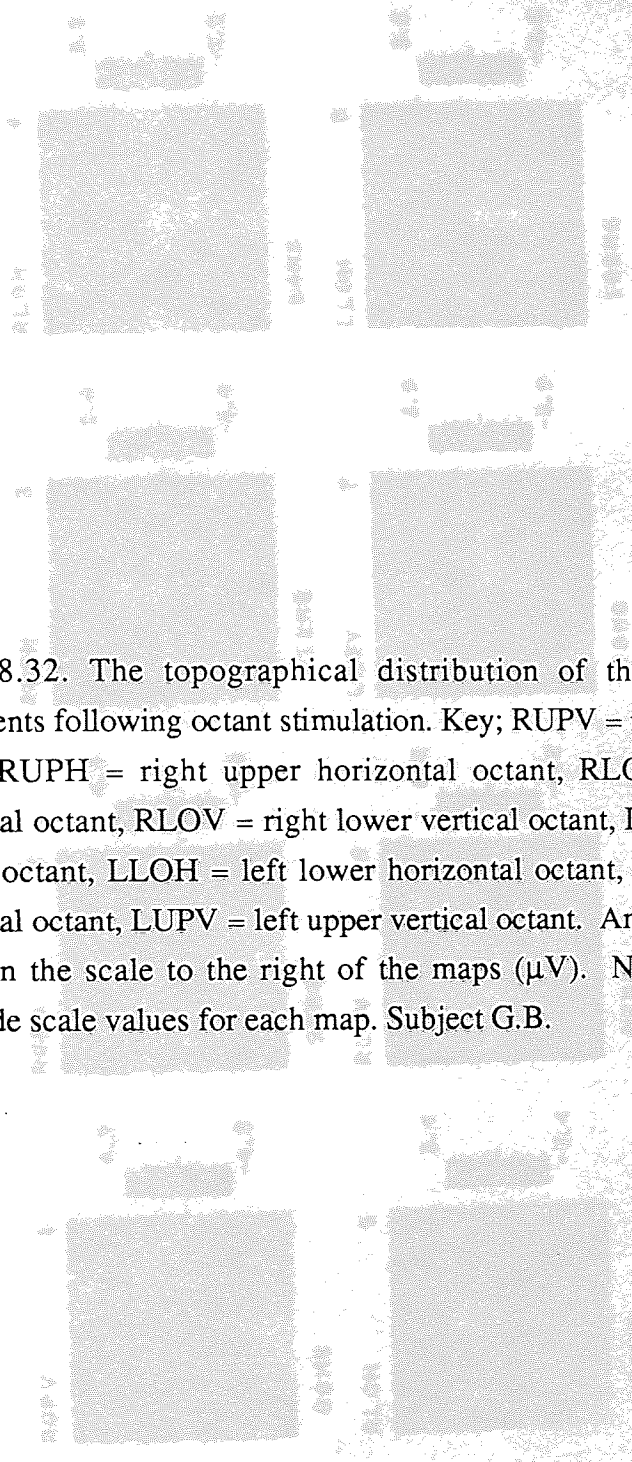
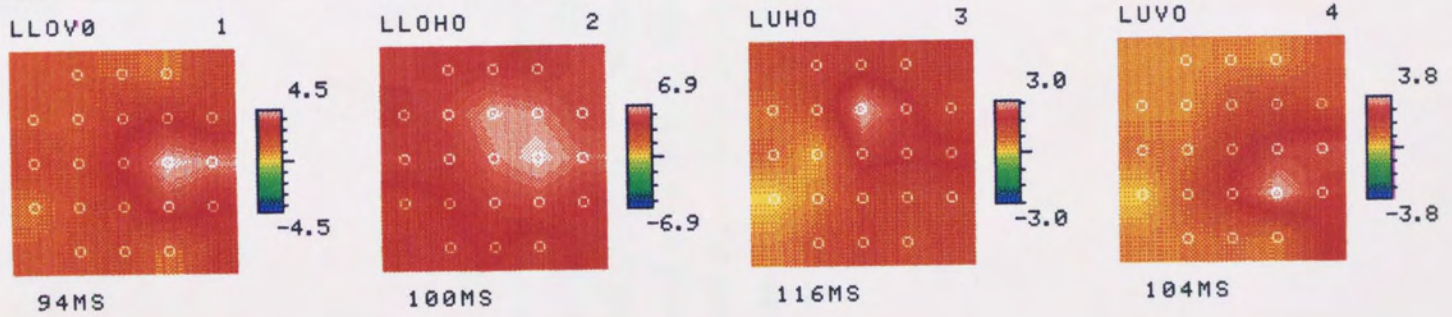


Figure 8.32. The topographical distribution of the major positive components following octant stimulation. Key; RUPV = right upper vertical octant, RUPH = right upper horizontal octant, RLOH = right lower horizontal octant, RLOV = right lower vertical octant, LLOV = left lower vertical octant, LLOH = left lower horizontal octant, LUH = left upper horizontal octant, LUPV = left upper vertical octant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject G.B.

RUPV
1
4.3
-4.3
88MS
RUPH
2
4.0
-4.0
90MS
RUPH
3
5.0
-5.0
112MS
RLOH
4
3.5
-3.5
84MS
RLOH
5
3.4
-3.4
104MS
RLOV
6
6.2
-6.2
82MS
LLOV
7
3.9
-3.9
98MS
LLOH
8
3.6
-3.6
108MS
LUPH
9
3.7
-3.7
110MS
LUPV
10
4.5
-4.5
108MS

Figure 8.33. The topographical distribution of the major positive components following octant stimulation. Key; LLOVO = left lower vertical octant, LLOHO = left lower horizontal octant, LUHO = left upper horizontal octant, LUPVO = left upper vertical octant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject N.E.


LLOV0
1
4.5
-4.5
94MS
LLOHO
2
6.9
-6.9
100MS
LUHO
3
3.0
-3.0
116MS
LUVO
4
3.8
-3.8
104MS

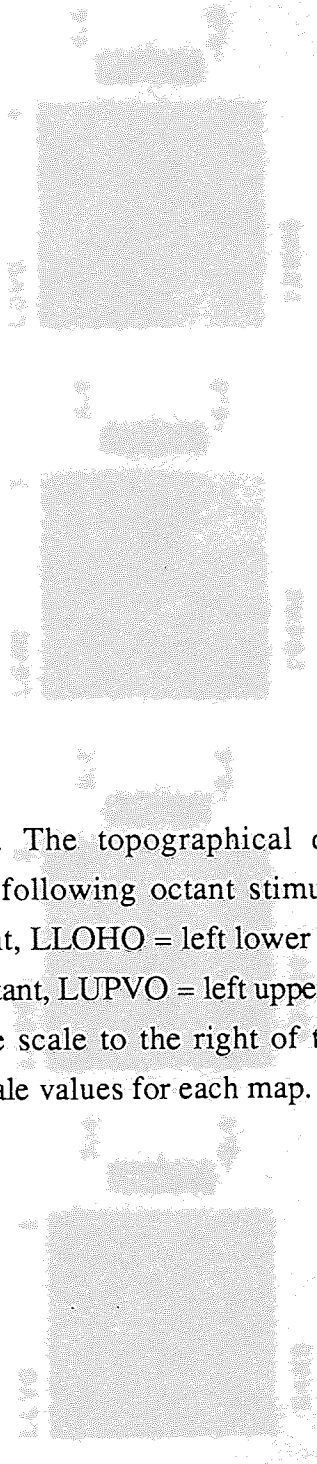


Figure 8.34. The topographical distribution of the major positive components following octant stimulation. Key; LLOVO = left lower vertical octant, LLOHO = left lower horizontal octant, LUHO = left upper horizontal octant, LUPVO = left upper vertical octant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map. Subject N.P.

LLVO
1
3.4
-3.4
94MS
LLOHO
2
2.6
-2.6
124MS
LUHO
3
6.8
-6.8
106MS
LUVO
4
4.6
-4.6
120MS

8.5.3.ic Subject NE

With lower left vertical octant stimulation the major positivity was maximal over O3, this was followd by a negativity maximal over a similar region, see fig. 8.33. No negativity was recorded following lower left horizontal octant stimulation, a late positivity was however evident over the left of the montage. An early negativity was observed after both upper octants were stimulated, these being maximal over the left anterior area. A positivity was then recorded with both stimuli, the peak was more anterior and later with the horizontal octant. In addition a later anterior positivity was evident after upper vertical octant stimulation. Following these peaks both stimuli produced a negativity maximal posteriorly, with a distribution similar to the negativity after upper half field stimulation.

8.5.3.id Subject NP

The lower octant responses resembled those of subject NE in that no early negativity was evident. The first peak was a positivity maximal over the anterior region, the latency of this peak was greater following horizontal octant stimulation, see fig. 8.34. This was followed by a negativity after vertical octant stimulation, which was similarly maximal over the anterior montage. An early negativity was recorded with both upper octants, the distributions were however not very similar. The major positive peak was more posterior after horizontal octant stimulation and was followed by a later posterior positivity. None of the octant stimuli produced a late negative response.

8.5.3.ii Distribution of the Group Mean Waveforms

The group mean waveforms and topographical distributions are show in figures 8.35 - 8.40. Lower octant stimulation produced a maximal positivity over the centre of the montage, no early negativity was evident with either of the octants. The late negativity was however present for both stimuli, see fig 8.41. Only one positive peak was observed with lower horizontal octant stimulation, in contrast a later posterior positivity was also produced with the vertical octant.

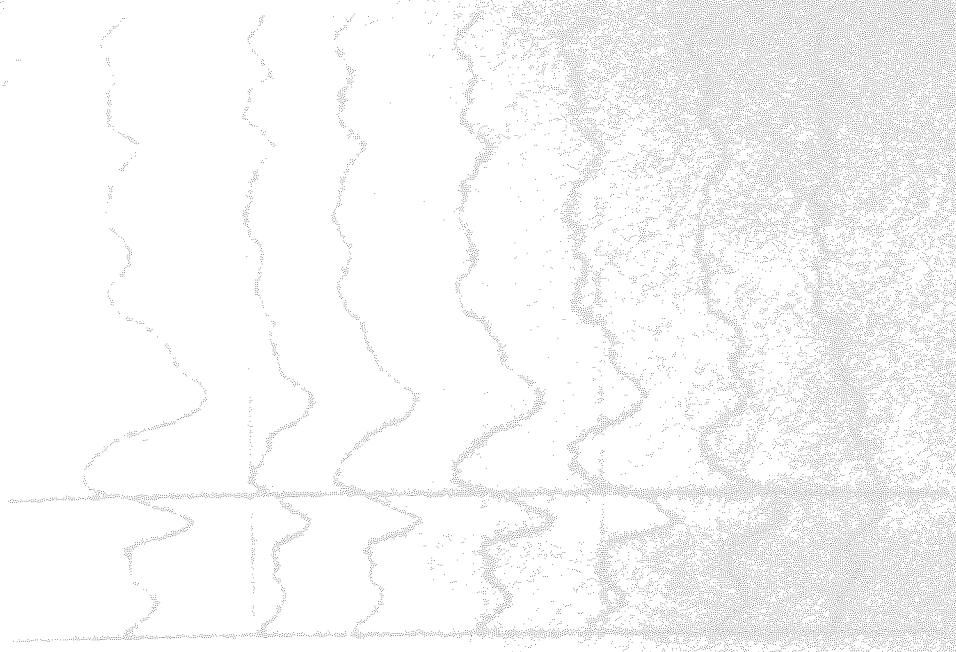


Figure 8.35. The group mean waveforms following left lower vertical octant stimulation. The position of the recording electrode is indicated to the left of the waveform. The cursor indicates the major positivities mapped in figure 8.36.

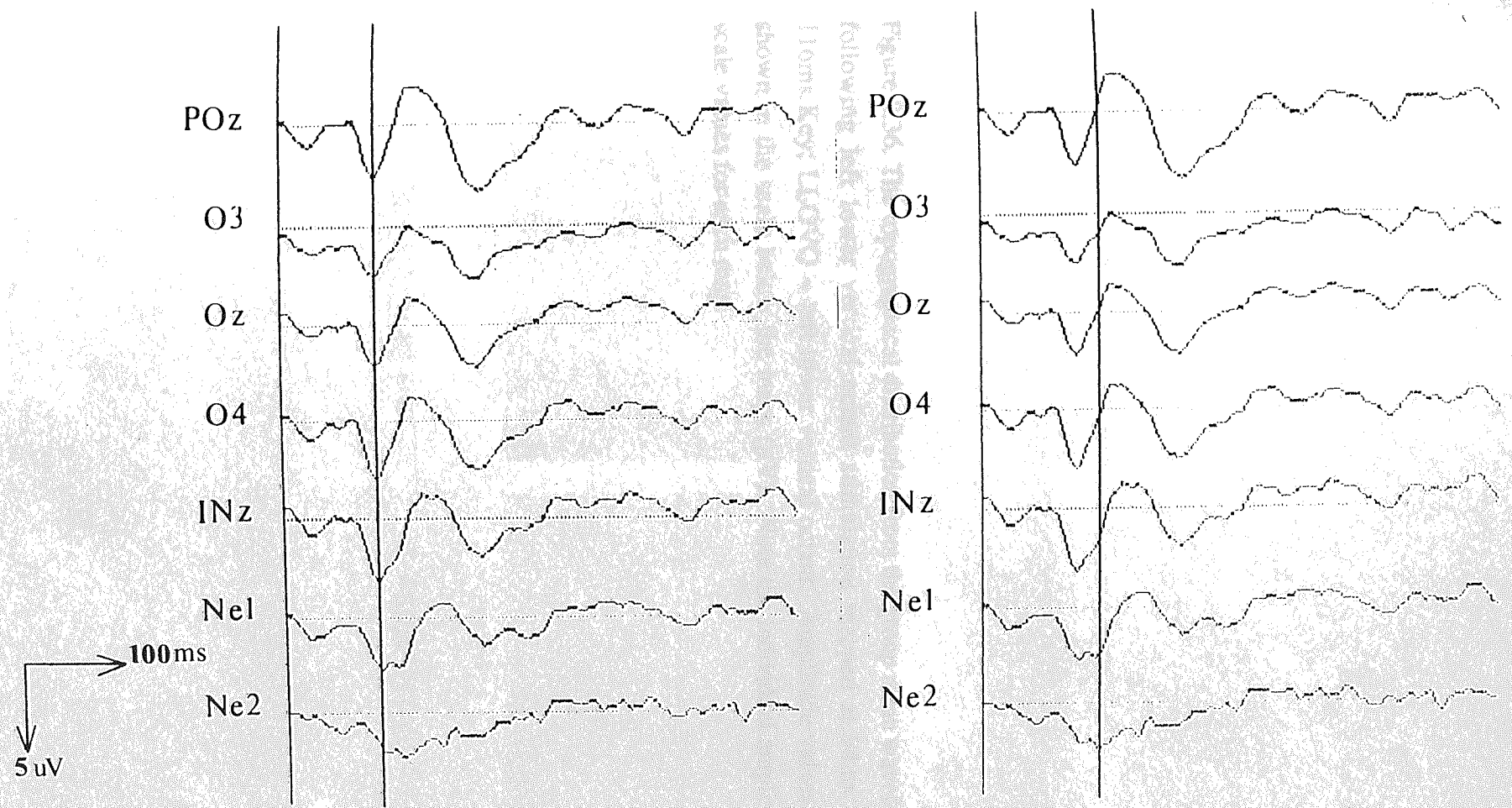
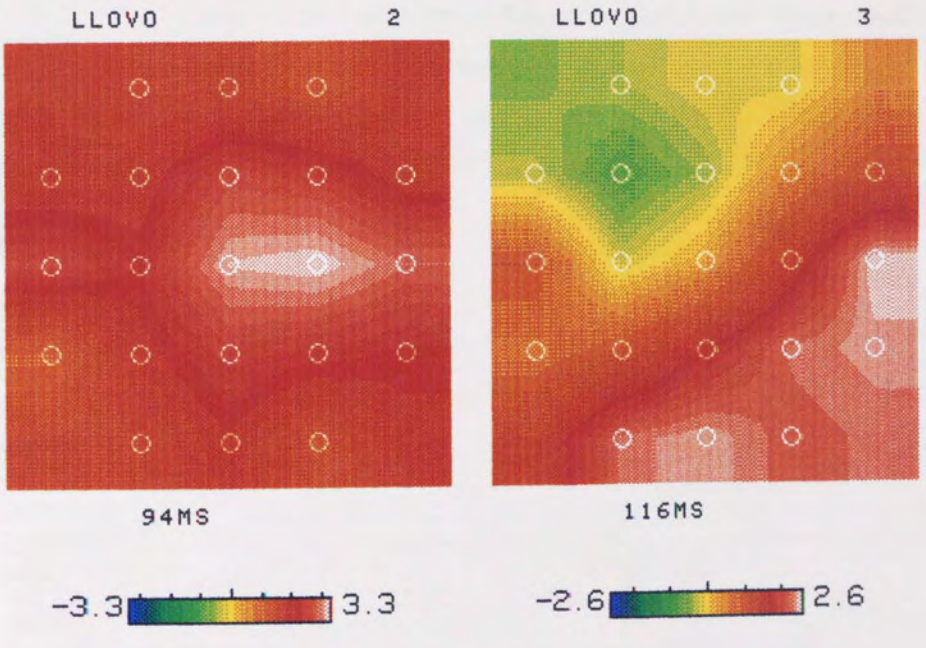

POz
O3
Oz
O4
INz
Nel
Ne2
100ms
5uV
POz
O3
Oz
O4
INz
Nel
Ne2

Figure 8.36. The topographical distribution from the group mean waveform following left lower vertical octant stimulation mapped at 94ms and 116ms.Key; LLOVO = left lower vertical octant. Amplitude values are shown in the scale below the maps (μV). Note the individual amplitude scale values for each map.


LLOVO 2
LLOVO 3
94MS
116MS
-3.3
3.3
-2.6
2.6

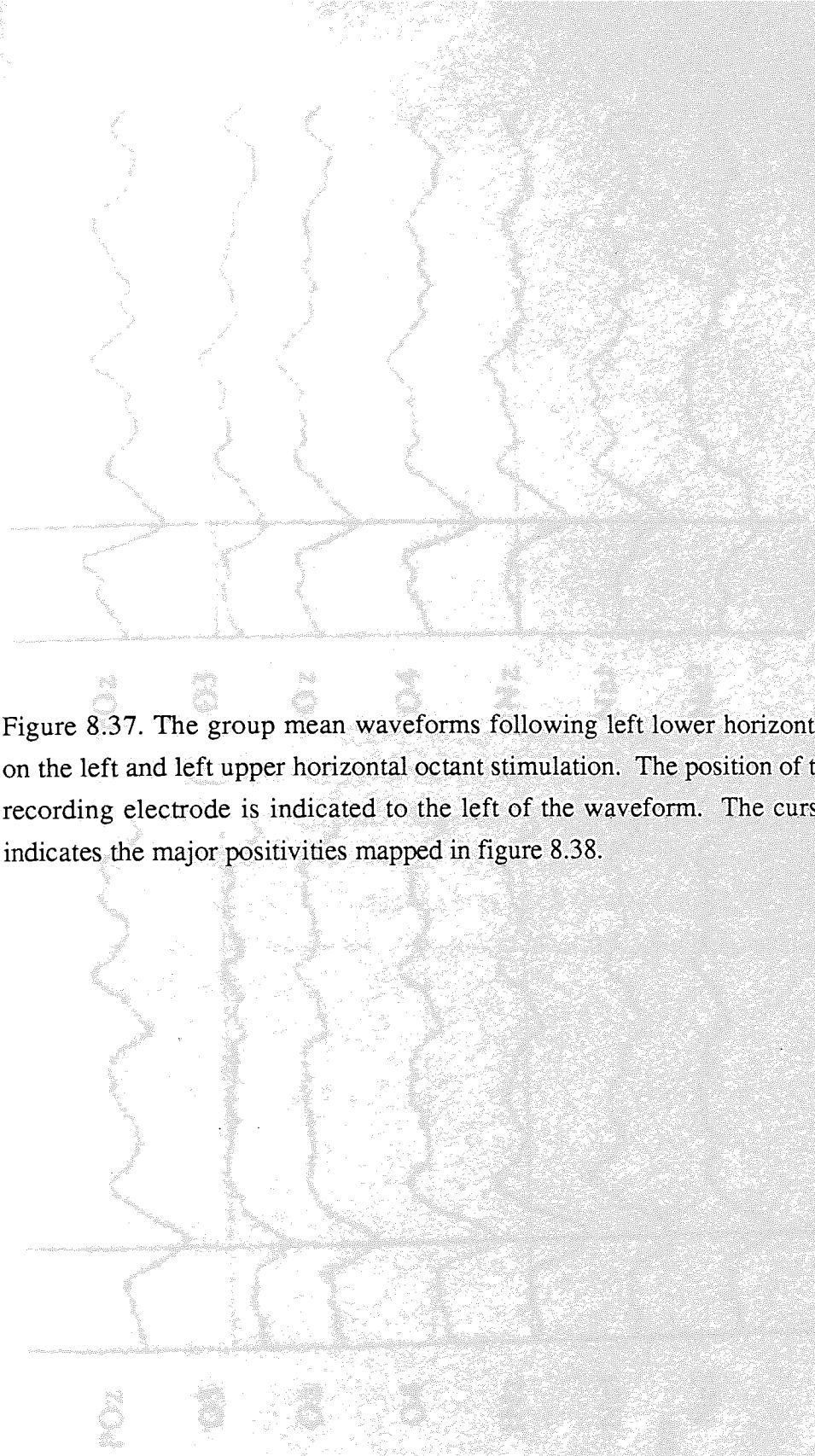


Figure 8.37. The group mean waveforms following left lower horizontal, on the left and left upper horizontal octant stimulation. The position of the recording electrode is indicated to the left of the waveform. The cursor indicates the major positivities mapped in figure 8.38.

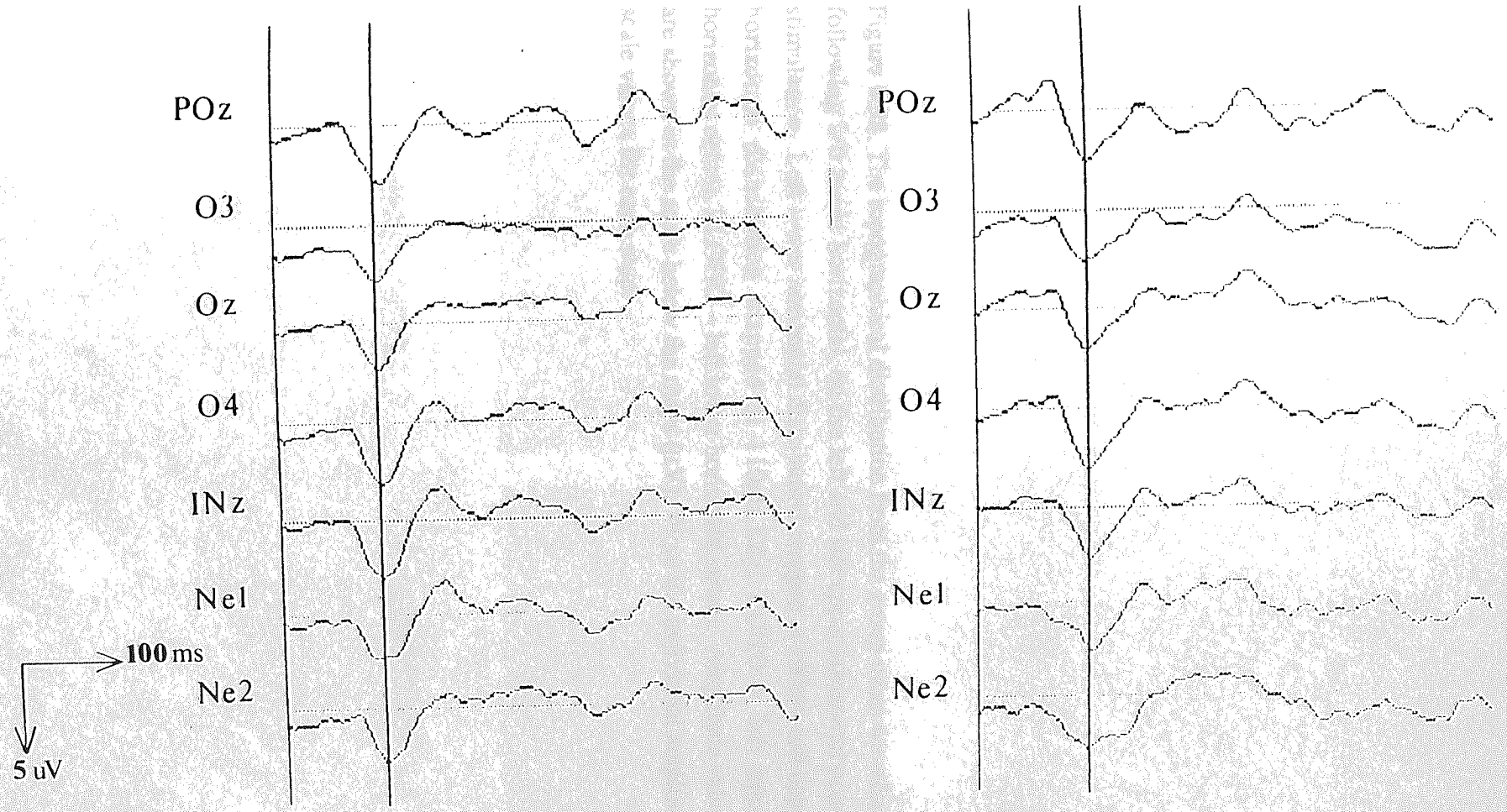
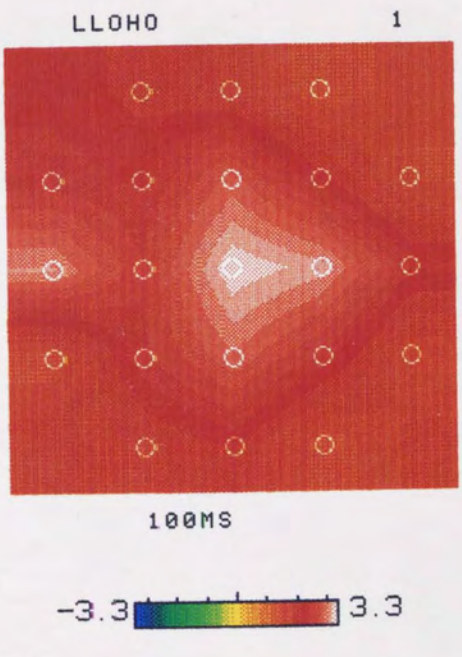
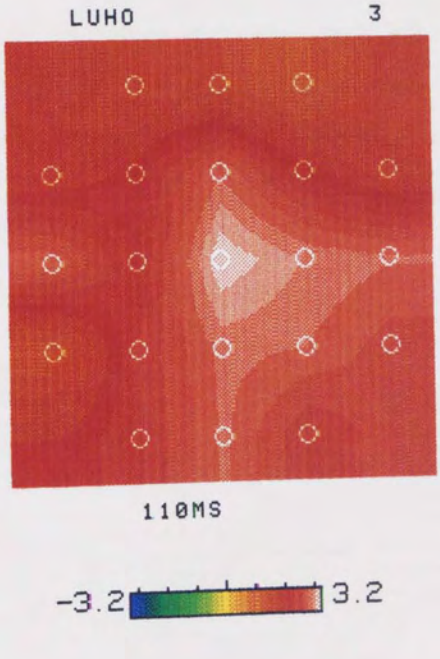

POz
O3
Oz
O4
INz
Ne1
Ne2
100 ms
5 uV
POz
O3
Oz
O4
INz
Ne1
Ne2

Figure 8.38. The topographical distribution from the group mean waveform following left lower horizontal, on the left and left upper horizontal octant stimulation. Left lower horizontal distribution mapped at 100ms, left upper horizontal distribution mapped at 110ms.Key; LUHO = left upper horizontal octant, LLOHO = left.lower horizontal octant. Amplitude values are shown in the scale below the maps (μV). Note the individual amplitude scale values for each map.


LLOHO
1
100MS
-3.3
3.3


LUHO
3
110MS
-3.2
3.2

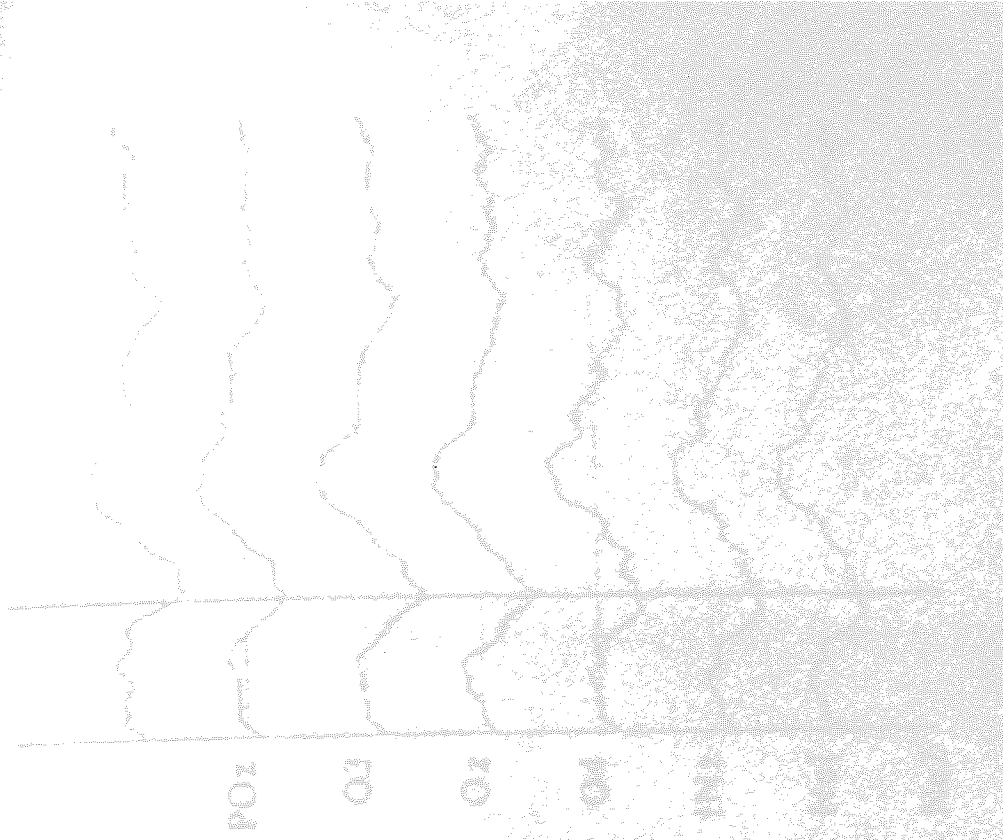
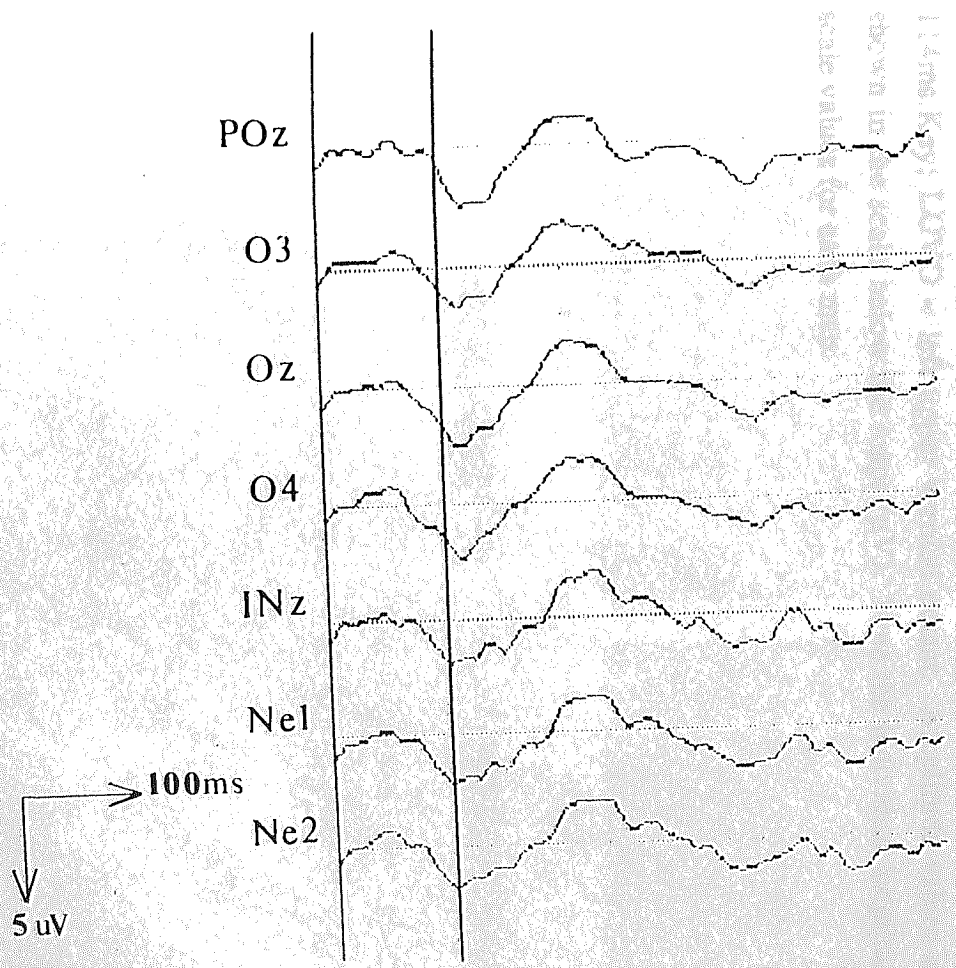


Figure 8.39. The group mean waveforms following left upper vertical octant stimulation. The position of the recording electrode is indicated to the left of the waveform. The cursor indicates the major positivities mapped in figure 8.40.


POz
O3
Oz
O4
INz
Nel
Ne2
100ms
5 uV

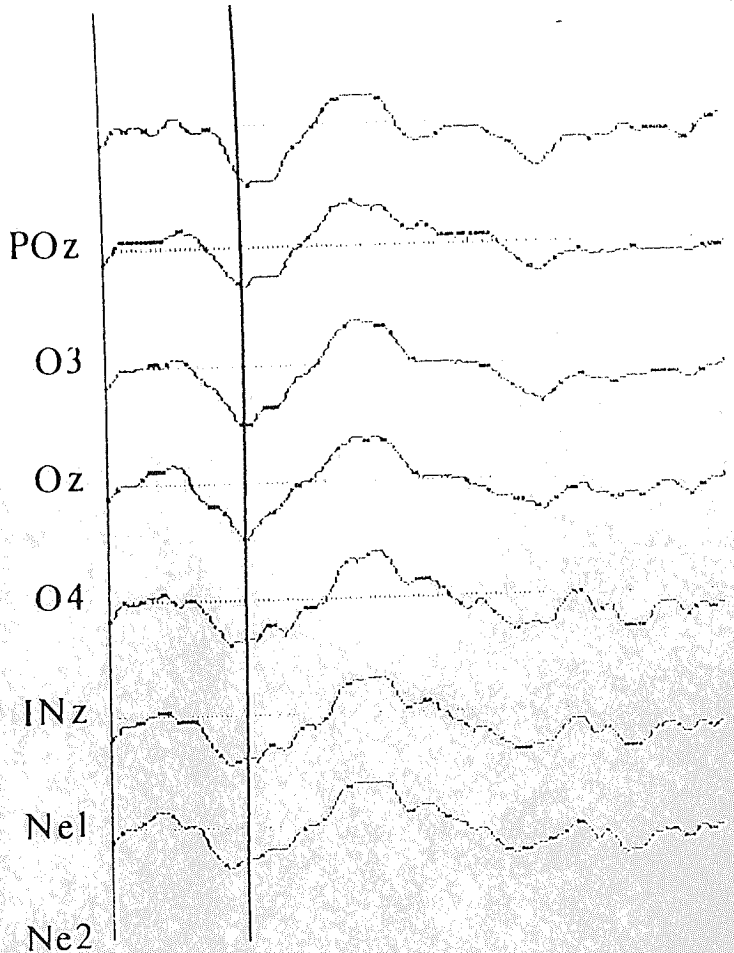

POz
O3
Oz
O4
INz
Nel
Ne2

Figure 8.40. The topographical distribution from the group mean waveform following left upper vertical octant stimulation mapped at 100ms and 114ms.Key; LUVO = left upper vertical octant. Amplitude values are shown in the scale below the maps (μV). Note the individual amplitude scale values for each map.

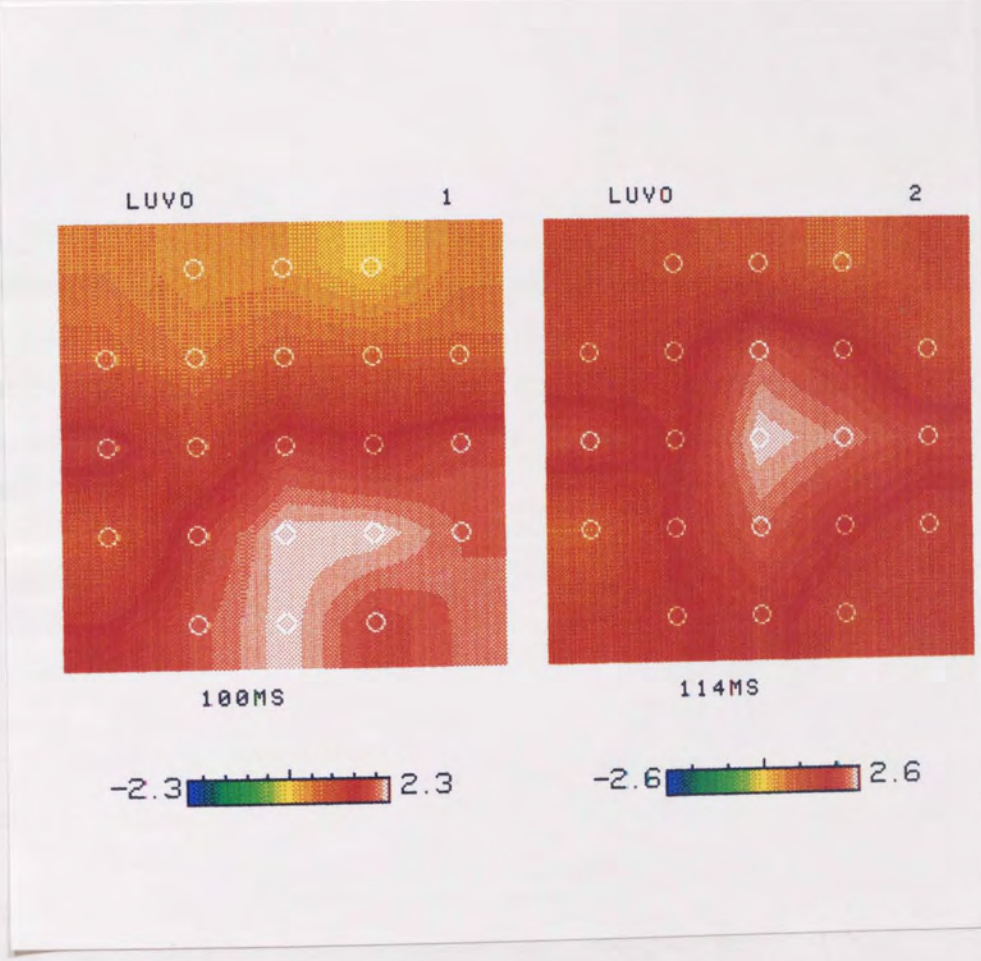

LUVO
1
100MS
-2.3
2.3
LUVO
2
114MS
-2.6
2.6

Figure 8.41. The topographical distribution, from the group mean waveforms, of the major peaks excluding the P100 after octant stimulation. Key; LUHO = left upper horizontal octant, LOHO = left lower horizontal octant, LOVO = left lower vertical octant. Amplitude values are shown in the scale to the right of the maps (μV). Note the individual amplitude scale values for each map.

LUHO
1
1.5
-1.5
76MS
LUHO
2
2.2
-2.2
138MS
LOHO
3
1.8
-1.8
160MS
LOVO
4
1.9
-1.9
132MS

The largest early negativity was recorded after upper horizontal octant stimulation. The positivity following this peak was maximal over the central right region of the montage. On upper vertical octant stimulation a posterior positive peak was initially recorded, this was succeeded by a more anterior positivity. A late negativity was the last component with both upper octants, this being more prominent with octants in the lower field.

8.5.4 Discussion.

Halliday and Michael (1970) demonstrated that, in spite of large intersubject variability all subjects showed a prominent wave with a peak latency of between 80 and 120ms after octant stimulation. The wave varied consistently and systematically in size and polarity depending on which octant was stimulated. Stimuli presented in the vertical octants gave the largest amplitude signals whereas the horizontal responses were small and sometimes ill defined, the horizontal octant response was probably attenuated due to the site of origin relative to the recording electrode. The major component produced by the upper octants was negative, in contrast the lower octants produced a positive response the amplitude of which was greater than the negative upper octant response. The polarity reversal was independent of the reference used, being present with both a mid frontal and mid cervical reference. In a few subjects the upper horizontal octant produced a positive peak, in others a transitional triphasic wave was seen. The response was maximal over the contralateral hemisphere. The polarity reversal was explained by a difference of afferent connections for the two quadrants to different layers of the cortex. They proposed that the site of generation was not in the calcarine fissure i.e. not located in the striate cortex.

Further investigatons showed that the upper vertical octants produced a positivity below the inion which was particularly associated with a contralateral negativity (Halliday et al 1977). Upper and lower horizontal octants produced ipsilateral positivities while with stimuli near the lower vertical octant the maximum positive tended to be over the midline or slightly contralateral. Corresponding octants in the left and right fields do not always produce complementary components.

In contrast to Halliday and Michael (1970) the amplitudes of the positive peaks from the group mean waveforms in this study were not lower following horizontal stimulation. Upper octant stimulation produced a major positive and not a negativity, in agreement with Halliday et al 1977. The positive peak appeared to be contralateral for all octants except the lower horizontal octant, the laterality of

this latter response supports the distribution observed by Halliday et al 1977. The distribution of the major positivity following upper and lower octant stimulation was similar, as was observed following upper and lower non-opposing dioctant stimulation. This would suggest a predominantly radial source in the vertical plane with a small tangential component. In addition the distribution following vertical octant stimulation appears to agree with that following vertical, i.e. opposing, dioctant stimuli in the upper and lower fields. A late negativity was recorded with octants situated in the lower field, this would fit with the prominence of this peak following lower half field stimulation. An early negativity was only observed following upper horizontal octant stimulation this would correspond with the dominence of this peak when responses following upper and lower field stimulation are compared.

Ossenblok and Spekreijse (1991) studied the pattern onset response after octant stimulation with principal component analysis and dipole fitting. Upper octant dipoles were mainly tangential, the lower octant's were mainly radial, in addition the sources for the upper octants were deeper. The laterality of the responses found in the present study, using a pattern reversal stimulus would suggest that the lower horizontal octant source was tangential, producing an ipsilateral distribution and the other octants were radial. This does not fit with the results of Ossenblok and Spekreijse and may be due to the different modes of stimulation studied. Orientations of the dipoles following vertical octant stimulation were shown to be very similar, both directed inferiorly. Horizontal octant stimulation gave dipoles directed away from the midline toward the contralateral side, the latencies were all similar. Equivalent dipole sources for stimuli near the horizontal meridian did not reverse in polarity whereas stimuli lying near the vertical meridian had a different orientation. A second component resulted in similar dipole positions for all octants, mainly tangential and clearly contralateral.

8.6 Summary

The early ipsilateral negativity appears to be more prominent after upper quadrant stimulation. In contrast the late negativity appears to be more prominent after lower quadrant stimulation. The position of the major positive response following quadrantic stimulation appears to fit with the component distributions following half field stimulation, i.e. ipsilateral to the lateral half fields and contralongitudinal to the altitudinal half fields. A later positive peak was recorded after upper quadrant stimulation.

The early negativity was again more prominent after upper octant stimulation, in addition the upper octants produced a late negativity. A late positivity was also found with both vertical octants, being maximal ipsilateral to the stimulating field. The octant responses although quite variable between subjects appear to correspond with the group dioctant distributions. A posterior positivity was recorded from two subjects after upper horizontal octant stimulation, the group mean response appears however to be dominated by a later anterior positivity. In contrast a posterior positivity was more dominant after upper vertical octant stimulation, although a later anterior comoponent was also evident.

Upper vertical dioctant stimulation produced a posterior positivity followed by an anterior positivity, part of a PNP complex. The upper horizontal dioctant response was a large anterior positivity with an earlier posterior positivity, again part of a PNP complex. In contrast to the upper vertical dioctant response the latency of the earlier positivity following upper horizontal dioctant stimulation was similar to that of the P80 peak. A late contralateral positivity was recorded with all lateral dioctants, the contralateral components do however appear to be most prominent with the right horizontal octants. The latency of the responses following horizontal octant stimulation appear to be later than those from the vertical octants this was however not significant when tested with a two factor split plot analysis of variance.

It therefore appears that the late anterior positivity observed following upper field stimulation may be the late positivity produced by the individual octants. The posterior positivity corresponds with stimulation of the vertical octants, which are projected more ventrally on the striate cortex. As a consequence of the reduced amplitude response produced after lower octant stimulation this late positive response does not appear to be evident in the lower half field response.

CHAPTER 9

Discussion and Conclusions

It has often been suggested that the position of the generators of the visual evoked potential can be deduced from the scalp potential distribution (Lehmann and Skrandies 1979, Lesevre 1979). As a consequence both the striate and extrastriate cortex have been proposed as the most probable generator sites for the major positive peak, P100 of the pattern reversal response.

The evoked potential recorded on the scalp is sensitive to sources lying both radially and tangentially to the scalp surface. In a flat radial source the largest amplitude potential is recorded over the midportion of the generator, in the case of a tangential source a dipolar distribution is achieved. These distributions are a result of the orientation of electrodes with respect to the configuration and orientation of the electric field, the amplitude of the potential measured at a point being proportional to the solid angle subtended by the dipole layer at that point (Gloor 1985). When the generator is distributed over both sulci and gyri the potential profile will be similar to that of the flat radial generator except that the gradient of the potential profile will be steeper, i.e. the distribution will appear more focal. As a consequence of tangential sources the response maxima may not be located over the expected region, this is classically demonstrated in the paradoxical ipsilateral lateralisation of the P100 component following lateral half field stimulation (Barrett et al 1976).

A technique has been developed in the last twenty years to record magnetic activity associated with electrical events in the brain. In contrast to the VEP this preferentially records activity generated by tangential sources. In addition this technique has advantages over recording the visual evoked potential in that smearing of the evoked current by interfaces between the source and the recording instrument does not affect the magnetic field and ambiguity due to the reference is avoided. An increased accuracy in source localisation is achieved when using the VEMR by virtue of the reduction in smearing effects.

The most consistent peak in the VEMR (visual evoked magnetic response) has been shown to be an outgoing field at approximately 100msec. In the present

study the distribution of the outgoing field of the VEMR to half field stimulation was maximal over the correct hemisphere following left half field stimulation and more over the midline following right half field stimulation, see fig 9.1. There was however intersubject variability in these distributions, see fig 6.4 -6.7 .

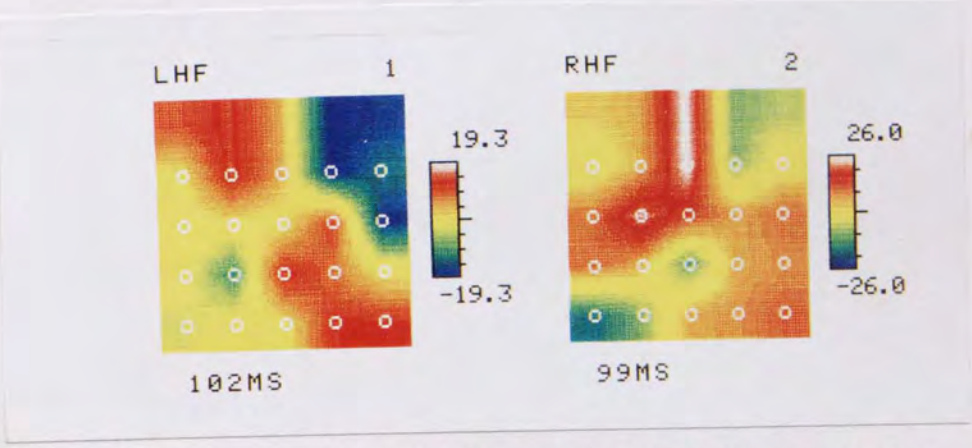


Fig 9.1 Topographical distribution of the VEMR following left (LHF) and right (RHF) half field stimulation. The distribution illustrates the major outgoing (positive) and ingoing (negative) field at the latency shown below the map. Stimulation is with 70' checks, subject R.A. Note individual amplitude scales to the right of the maps (μV).

The latency of the VEMR was found to be longer than the corresponding VEP, the latencies of both responses did however possess significantly similar trends when the size of the stimulating check was reduced, i.e. an increase in latency with a reduction in check size. This suggests that we are recording different aspects of the same electrical event.

Dipole fitting procedures were attempted on the half field VEMR responses, see fig. 9.2. The successful fits provide evidence that the generator of the VEMR was within the striate or neighbouring cortex.

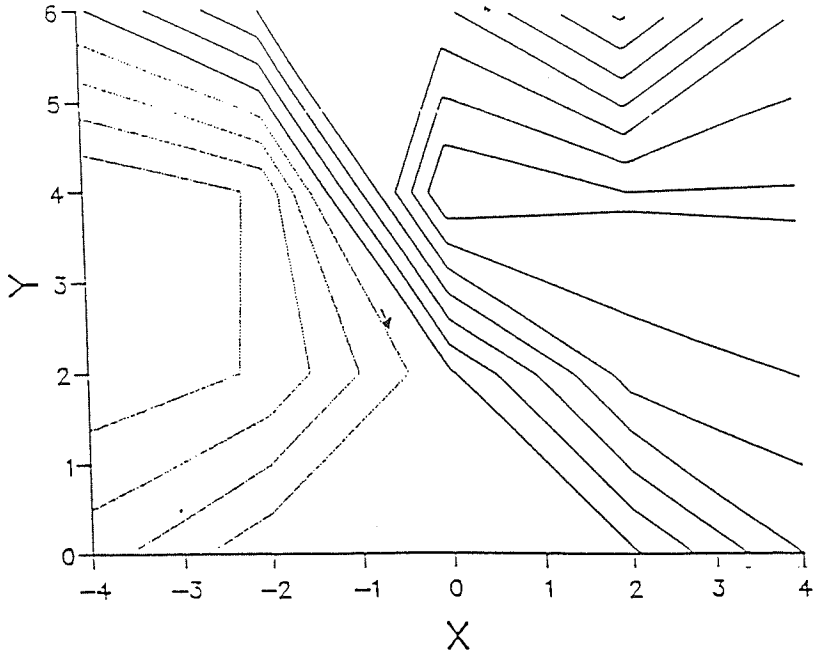


Fig. 9.2 The dipole fit to the distribution recorded after full field 22' stimulation, subject R.A.

The striate visual cortex has being schematically represented as a '+' this is termed the cruciform model, see fig. 9.3.

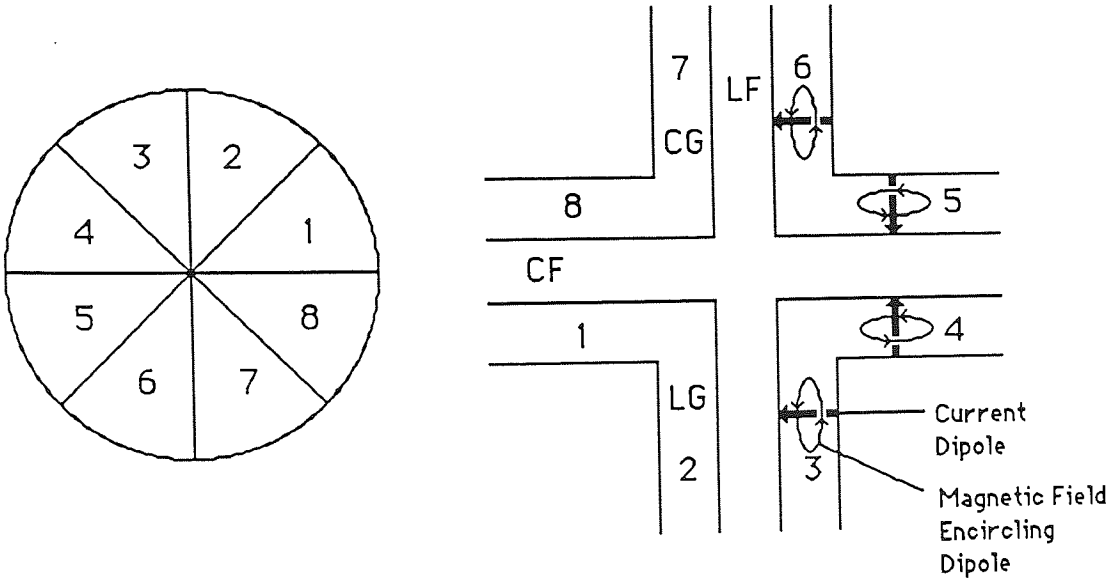


Fig.9.3. The cruciform model demonstrating the projection of the visual field on the striate cortex. Activation of the left half field will result in the illustrated dipoles being activated and the concomitant magnetic field. LF= longitudinal fissure, CF= calcarine fissure, CG= cuneal gyrus and LG= lingual gyrus. After Harding, Janday and Armstrong (1991).

The effect of stimulating different areas of the visual field on the components of the pattern reversal response has been investigated. Full field, half field, central and peripheral stimuli were initally studied. Further investigation into the generator sites of the VEP led to the development of a stimulus that should maximally stimulate specific cortical regions indicated by the cruciform model. Stimuli were used that would maximally stimulate opposing and non-opposing regions of the striate cortex. These stimuli consisted of two octants positioned in either the upper, lower, left or right half field. In addition further reduction in the stimulus size was attempted and the distributions following octant stimulation in the left half field were investigated.

The half field VEP consists of six peaks; N75, P80, P100, N105, P120 and N145. Intracortical studies in monkeys have proposed that all the components up to and including the P100, the P120 is not however mentioned, are a result of activation in the striate cortex. The N145 peak was thought to be due to activation of the extrastriate areas (Schroeder et al 1988, 1990, 1991).

The topographical distributions at the time of maximal global field power in the group mean waveforms were compared with those at the peak latency after stimulation with different field sizes and locations. In some cases no peak in the field plot was found to correspond with a peak in the potential profile, this could be either a result of the low response amplitude or a shallow potential gradient. For example with upper quadrant stimuli no early peak was found in the field plot, see table 8.5. For the majority of stimuli the latency of the maximal field strengths corresponded with peaks in the potential profile, thus resulting in a similar topographical distribution. This is in agreement with Hamburger and van der Burgt (1991) who found no significant difference between the amplitude, latency or topographical distribution of the major components when comparing the two methods.

The N75 component, the first repeatable response in the VEP, was found to be more prominent when stimuli were located in the upper field, although no significant effect of field on amplitude was demonstrated. The maxima of N75 were located ipsilaterally following half field stimulation, this distribution would fit with a tangential source positioned according to the cruciform model, with responses along the opposing regions of the cortex cancelling, leaving those along

the non-opposing cortex to produce the response, see fig 9.4. The responses following quadrant stimulation follow the trends illustrated with lateral and altitudinal half fields, see fig. 9.5.

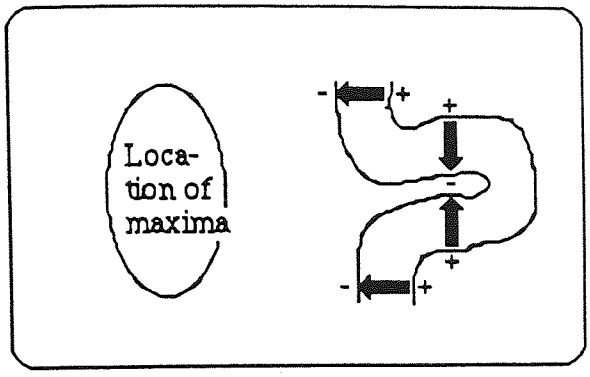


Fig 9.4. The location of the approximate dipoles thought to evoke N75 after stimulation of the left half field. The location of the maximal potential distribution on the scalp is illustrated.

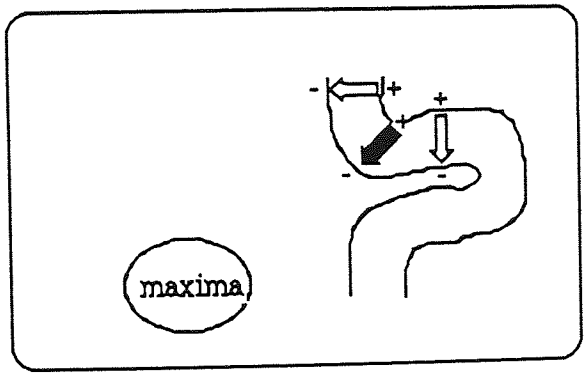


Fig 9.5 The location of the approximate dipoles thought to evoke N75 after stimulation of the left lower quadrant. The location of the maximal potential distribution on the scalp is illustrated.

The amplitude of N75 was found to reduce with a reduction in field size and the numbers of subjects producing the response declined. In contrast central full field occlusion produced an increase in the response amplitude, this however failed to reach significance. Previous findings have suggested that the N75 response is preferentially generated in the extrafoveal region of the cortex which is in agreement with the results obtained (Maugiere 1985). Thus for this check size the extrafoveal area is preferentially stimulated. It has been proposed that N75 is dependent on the spatial frequency content of the stimulus (Onofrj 1991) and

therefore with a much smaller check size the amplitude may not be significantly affected with central stimulation.

N75 was maximal following opposing dioctant stimulation when stimulating the lateral and lower fields, in contrast the response was greater following non opposing stimulation of the upper field. The cortical projections of these stimuli are located along the calcarine fissure for the lateral and upper fields, the lower projection is positioned along the longitudinal fissure, see fig. 9.6. This would suggest that the upper vertical octant minimally contributes to N75.

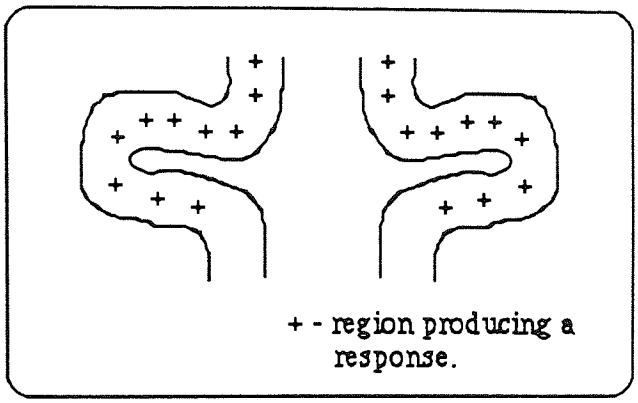


Fig. 9.6. Diagram to illustrate the regions of cortex producing the N75 component following dioctant stimulation.

N75 was most prominent following upper horizontal octant stimulation, a vague negativity was shown after lower vertical octant stimulation. The distribution was similar to that found after stimulation with the left upper quadrant, being maximal in the same position as the stimulating field, see fig. 9.7. This distribution would also appear to correspond with that found following dioctant stimulation. The topographical distribution could be predicted from the cruciform model of the striate cortex.

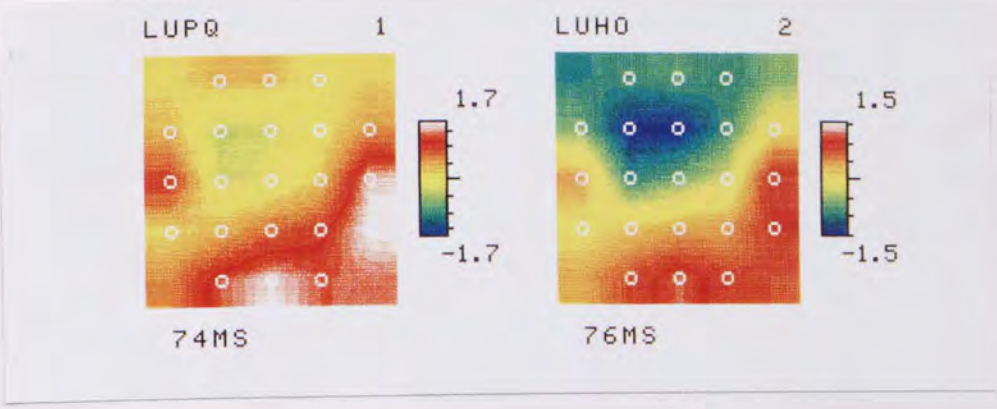


Fig. 9.7. Topographical distribution of the group mean waveform mapped at the peak of N75 following left upper quadrant (LUPQ) and left upper horizontal octant stimulation (LUHO). A positivity is also shown on the map this being more prominent following quadrant stimulation, this peaks slightly later than the N75 component. Note individual amplitude scales to the right of the maps (μV).

The peak P80 was not present in the full field response. On lateral half field stimulation however P80 was produced and was maximally distributed contralateral to the field. In addition on upper and lower half field stimulation the response was maximal posteriorly for the upper field and anteriorly for the lower field, i.e. also contra to the stimulating field. Occlusion of the central region of the left upper quadrant led to an increase in the amplitude of this response, suggesting that this response is either being masked by the central response or is a result of extrafoveal stimulation, see fig. 9.8.

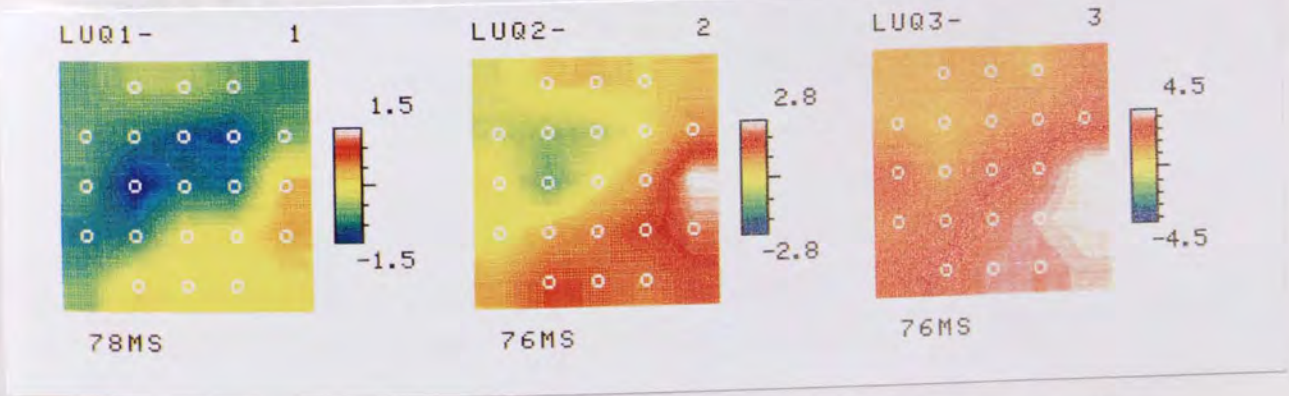


Fig 9.8. Topographical distribution mapped at the peak of N75 following progessive central occlusion of the left upper quadrant (LUQ), by 1°, 2° and 3°. Subject G.B. Note individual amplitude scales to the right of the maps (μV).

This component has been proposed to be related to N75, as the 'other end of the dipole' producing N75 (Onofrj 1990). The present study is in agreement with this proposal in that the amplitude of P80 was found to increase with peripheral stimulation, both quadrant and half field, with a concurrent reduction in the amplitude of N75, see fig 9.9. The increase in amplitude of P80 is more prominent on lower field compared with upper field stimulation.

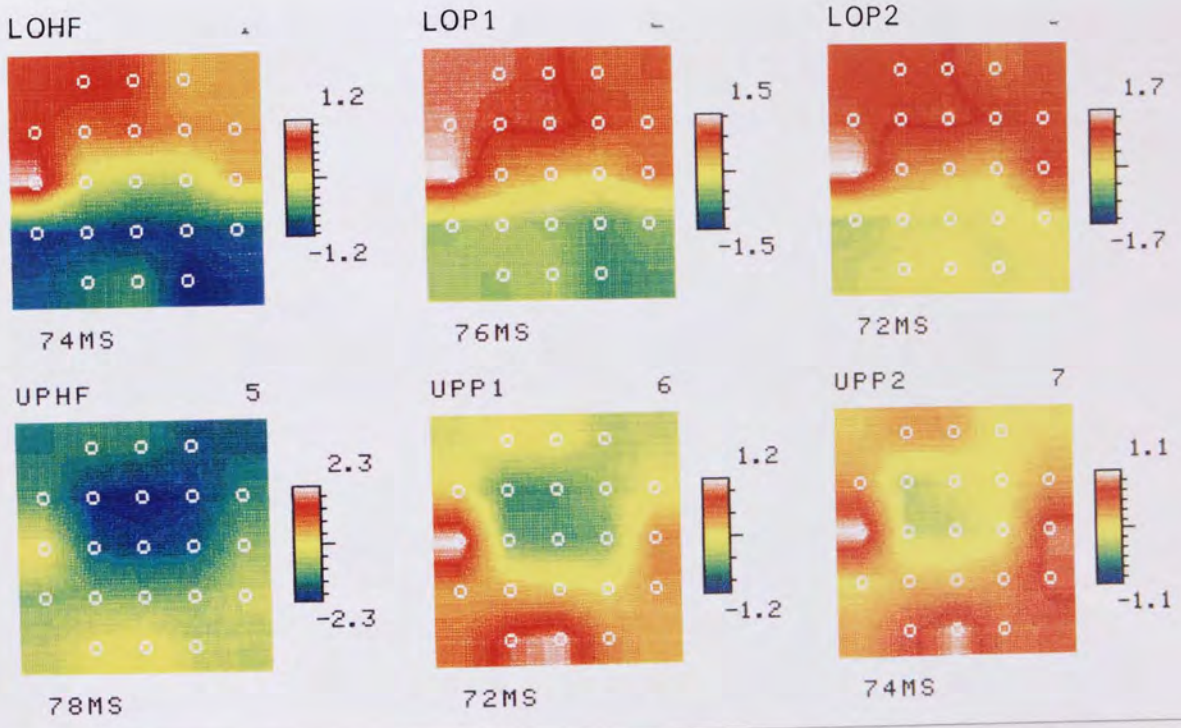


Fig 9.9. Topographical distribution mapped at the peak of N75 following full lower (LOHF) and upper half field (UPHF) stimulation and progessive central occlusion by 1° (LOP1, UPP1) and 2° (LOP2, UPP2). Note individual amplitude scales to the right of the maps (μV).

This would correspond with the proposal that on increasing peripheral stimulation the location of activity is moving around the occipital pole then along both the calcarine and longitudinal fissures, see fig 9.10. A discrepancy in the peak latencies of these components was however found, this is in agreement with previous studies.

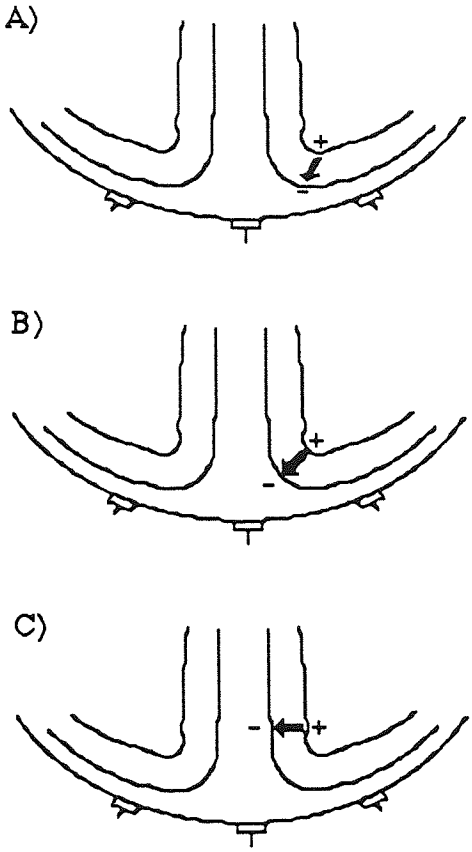


Fig 9.10. The relationship of N75/P80 with increasingly more peripheral stimulation. A - central stimulation, B and C - peripheral stimulation.

On examination of the cruciform model it would be expected that most of the P100 response after half field stimulation would be a result of stimulation along the longitudinal fissure. Thus resulting in a maximal distribution over the ipsilateral hemisphere. Lateral half field stimulation did produce an ipsilateral maximal positive P100 in contrast, the distribution of the P100 following altitudinal half field stimulation was maximal over the correct hemisphere, contralongitudinal, after upper field stimulation and over the midline for lower field stimulation.

In addition to the different distribution, the upper half field P100 response has previously been shown to be significantly later than that following lower half field stimulation. In the present study however a significant shift in latency failed to be demonstrated. The amplitude was however significantly reduced following upper field stimulation when compared with both the lower and full field response. No significant amplitude asymmetry was demonstrated when the ERG (electroretinogram) responses from the upper and lower half fields were

compared. This may suggest that the reduction in amplitude of the upper field response is a result of the more ventral position of the upper field projection onto the striate cortex rather than differential receptor and ganglion cell distributions.mm

When recording the upper half field response the major positivity was maximal over the posterior region of the montage, this suggests that clinical recording of the VEP from electrodes positioned over O1 and O2 will not maximally record the response from the upper field. Application of electrodes over the inion and below should ensure detection of the upper field response.

Reduction of the overall field size and central occlusion led to a significant reduction in the amplitude of P100 ($p<0.01$). The fact that the amplitude was reduced with both central occlusion and a reduction in field size would imply that the response amplitude was related to the area of stimulation.

The P100 distribution following lower quadrant stimulation appeared to fit with the cruciform model of the cortex with the maximal response being over the ipsilateral hemisphere. The distribution following upper quadrant stimulation was again maximal ipsilaterally and also posteriorly, see fig 9.11. This distribution would be more likely to fit with that following stimulation of the vertical octant.

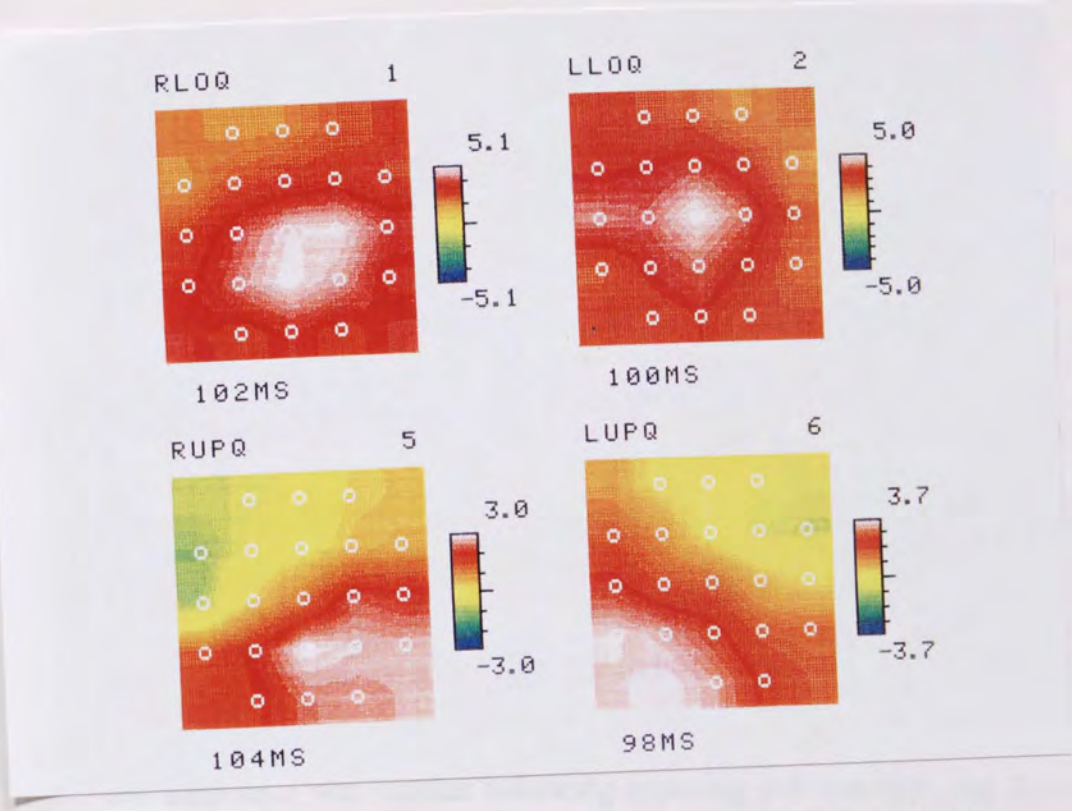


Fig. 9.11. Topographical distribution mapped at the peak of P100 in the group mean waveform following lower right (RLOQ) and left (LLOQ) and upper right (RUPQ) and left (LUPQ) quadrant stimulation. The peak latencies are shown below the maps. Note individual amplitude scales to the right of the maps (μV).

The upper and lower half field P100 distributions became increasingly similar with a reduction in field size, i.e. the upper field response became more anterior, see fig 9.12. In addition the distribution became maximal over the extremes of the montage, this may correlate with the cortical projection of the central field being located more over the occipital pole. The response therefore appeared to be moving out of the longitudinal and calcarine fissures and over the occipital pole.

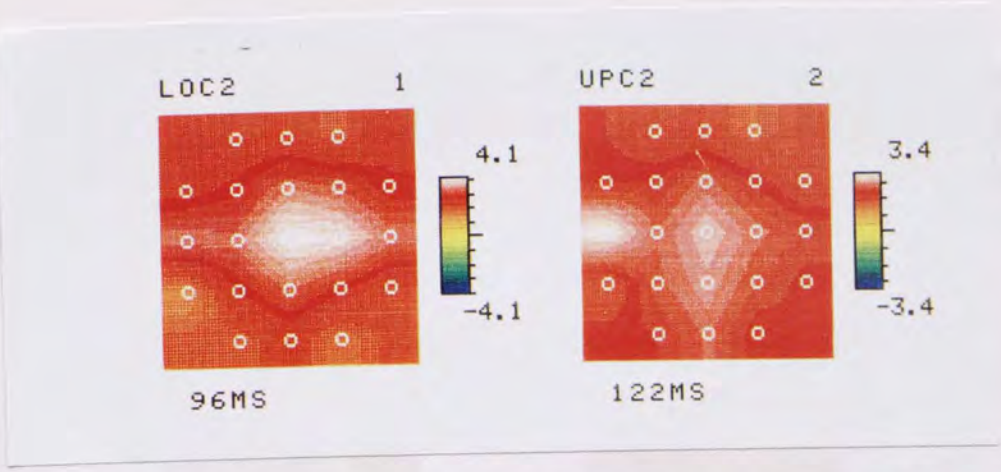


Fig. 9.12. Topographical distribution mapped at the peak of the major positivity following lower central (LOC2) and upper central (UPC2) 2° stimulation. The peak latencies are shown below the maps. Note individual amplitude scales to the right of the maps (μV).

The P100 amplitude was similar following opposing and non-opposing dioctant stimulation for the lateral fields, both distributions being maximal ipsilaterally, see fig 9.13.

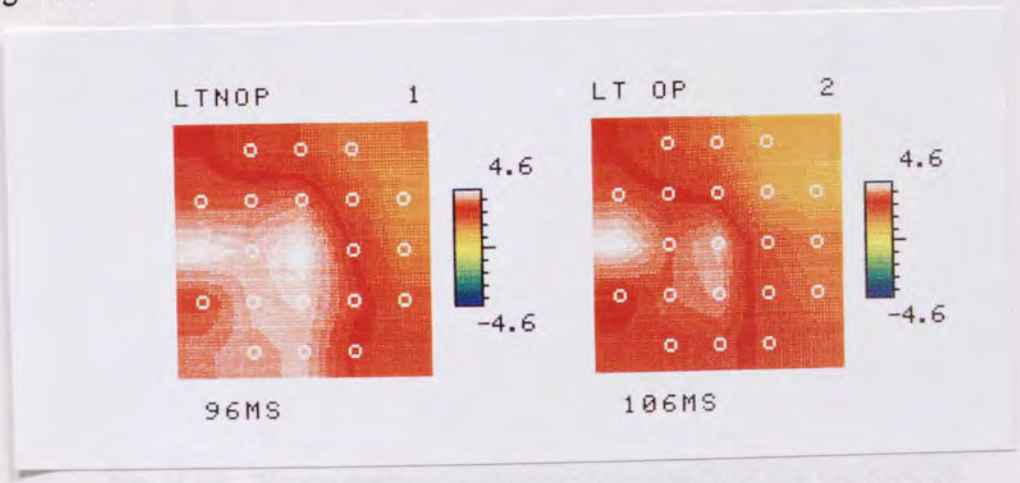
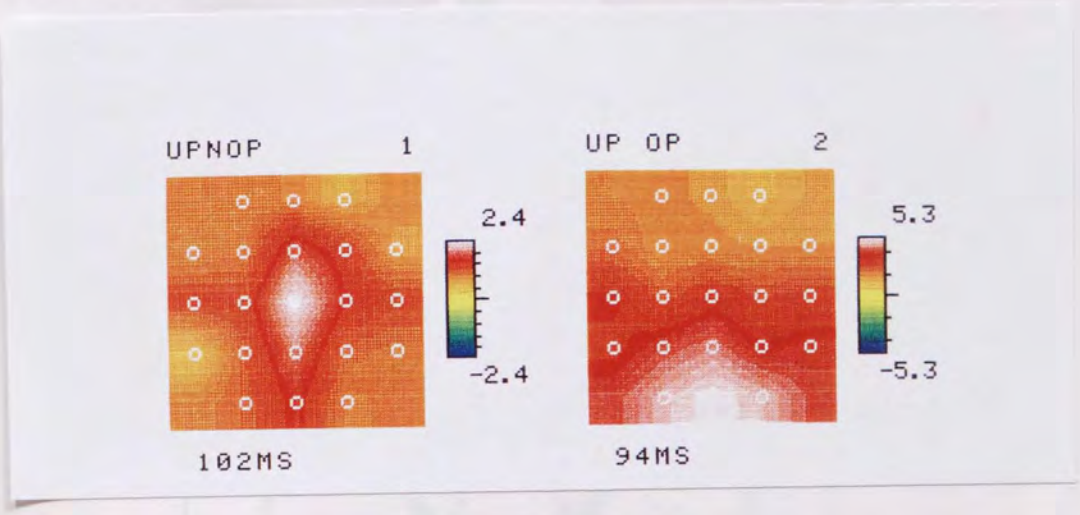


Fig. 9.13. Topographic distribution mapped at the peak of the P100 in the group mean waveform following opposing (LTOP) and non-opposing (LTNOP) dioctant stimulation in the left field. The peak latencies are shown below the maps. Note individual amplitude scales to the right of the maps (μV).

On right opposing dioctant stimulation the P100 of the group mean waveform was however masked by the contralateral N105. If these stimuli were stimulating the regions of the striate cortex proposed by the cruciform model then the response should be of reduced amplitude and more contralateral following opposing dioctant stimulation, this was not apparent with the lateral stimuli. In contrast the distribution following upper non-opposing and opposing dioctants did appear to

relate to the cruciform model with the upper non-opposing dioctants producing an anterior positivity and the opposing dioctants producing a posterior response, see fig 9.14. The distribution following lower field stimulation was not as dependent on the site of stimulation, both responses being maximal over Oz, see fig 9.15.



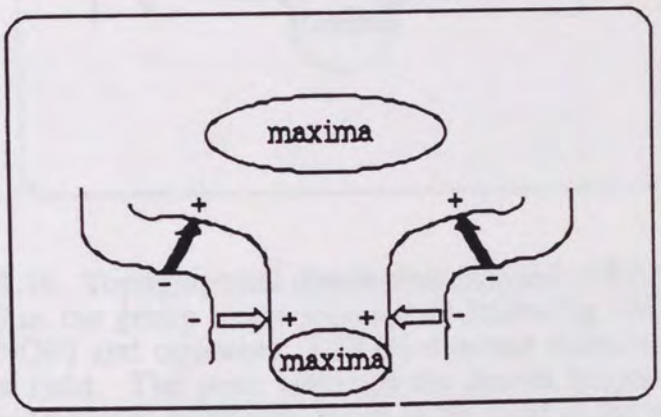
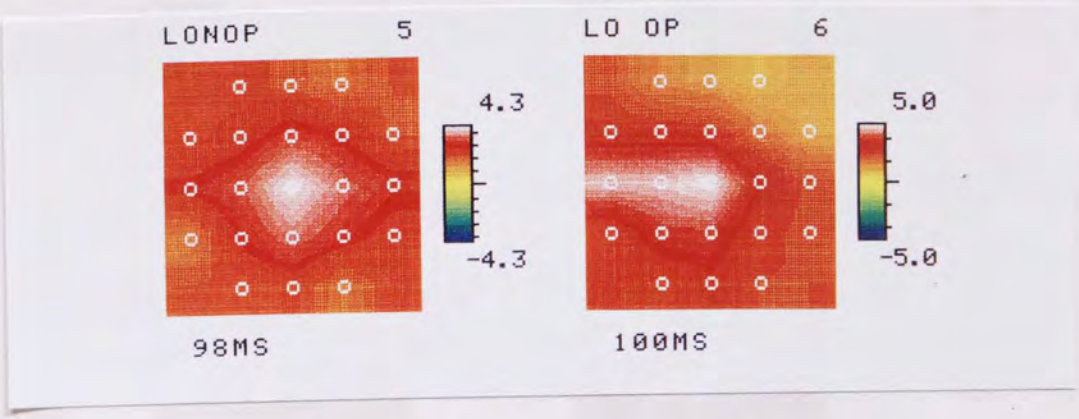


Fig. 9.14. Topographical distribution mapped at the peak of the P100 in the group mean waveforms following non-opposing (UPNOP) and opposing (UPOP) dioctant stimulation in the upper field. The peak latencies are shown below the maps. Note individual amplitude scales to the right of the maps (μV). The diagram illustrates the region of striate cortex activated and the equivalent dipoles produced after stimulation with the dioctant stimuli in the upper field. Black dipoles represent activation following non-opposing stimulation, white represent opposing dioctant stimulation.



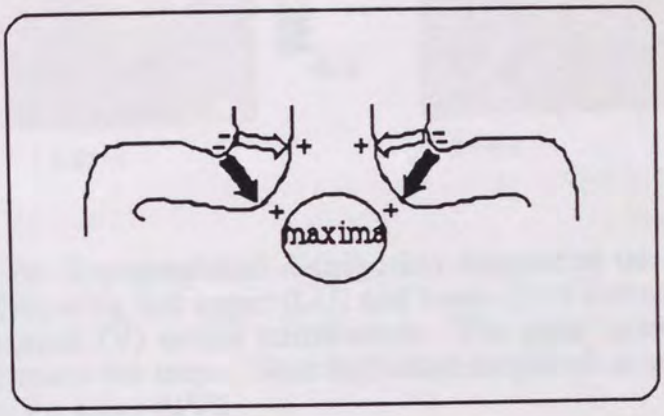


Fig. 9.15. Topographical distribution mapped at the peak of the P100 in the group mean waveforms following non-opposing (LONOP) and opposing (LOOP) dioctant stimulation in the lower field. The peak latencies are shown below the maps. Note individual amplitude scales to the right of the maps (μV). The diagram illustrates the region of striate cortex activated and the equivalent dipoles produced after stimulation with the dioctant stimuli in the lower field. Black dipoles represent activation following non-opposing stimulation, white represent opposing dioctant stimulation.

A similar central distribution was found after stimulation of both the upper and lower horizontal octants. A central distribution was also observed following lower vertical octant stimulation, in contrast the positivity was more posterior following upper vertical octant stimulation,see fig 9.16. The distribution of P100 would therefore correspond with the cruciform model of the striate cortex, with more anterior distributions following lower octant stimulation, these projecting to the more dorsal striate cortex.

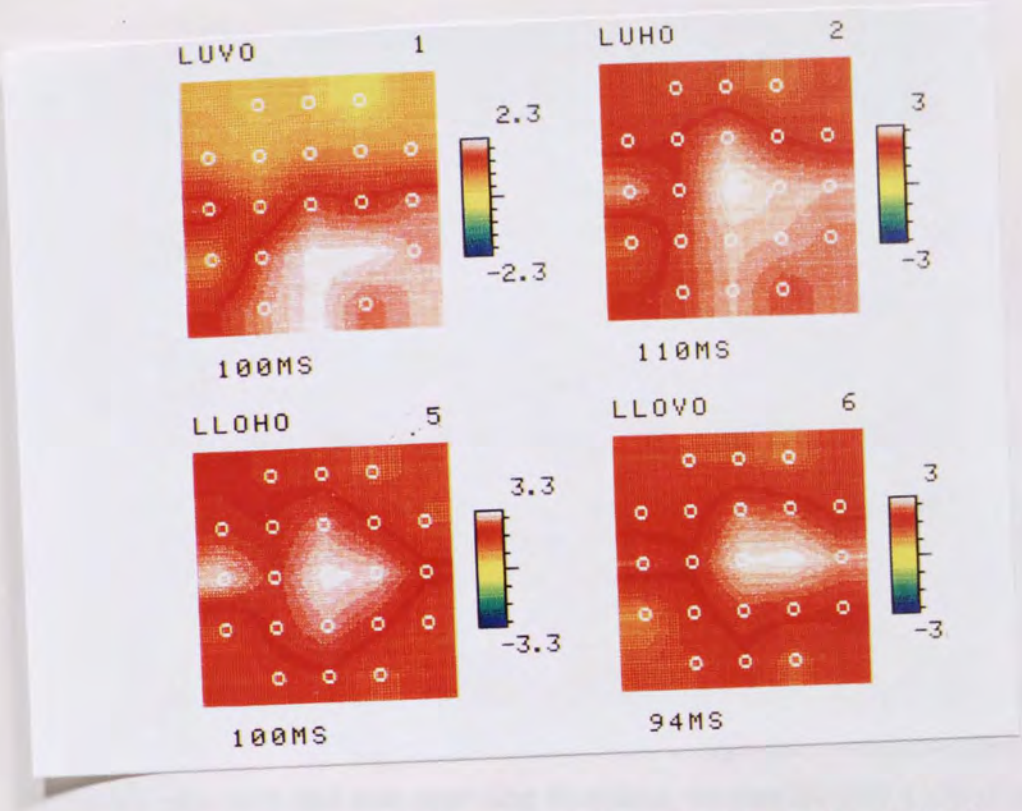


Fig. 9.16. Topographical distribution mapped at the peak of P100 following left upper (LU) and lower (LO) horizontal (H) and vertical (V) octant stimulation. The peak latencies are shown below the maps. Note individual amplitude scales to the right of the maps (μV).

The P100 distribution therefore appears to correspond with the cruciform model and with a generator in the striate cortex.

The lateralisation of the late positivity, P120, was opposite to that of the P100 following half field stimulation, the distribution was however similar to that of P80 on lateral half field stimulation, see fig.9.17. P120 was present in the group waveform and mean maps of all quadrant responses. This component appeared to reduce with central occlusion of the upper and lower half fields and upper quadrants.

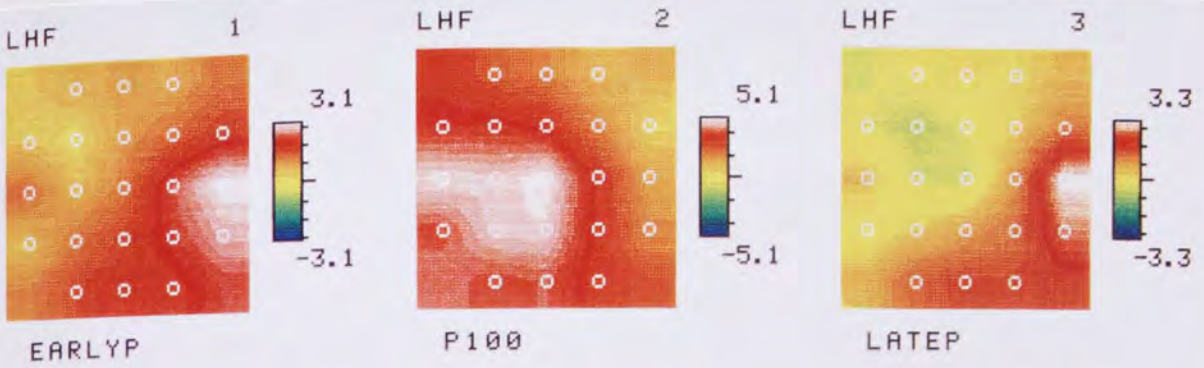


Fig. 9.17. Topographical distribution from the group mean maps following left half field stimulation. The maps are distributions of the P80, (EARLYP), P100 and P120 (LATEP). Note individual amplitude scales to the right of the maps (μV).

P120 appears to be more prominent following stimulation of the vertical dioctants when positioned in the lateral fields. The P120 component was obtained with both the upper opposing and non-opposing dioctants, in contrast only a vague positivity was recorded with the lower non-opposing dioctant. The distribution of P120 was similar to that found when stimulating the full half fields, i.e. contralateral for the lateral fields and ipsilongitudinal for the altitudinal half fields.

This late positivity was demonstrated following stimulation of the cortex by vertical octants but no later positivities were shown after horizontal octant stimulation. The P120 component does not appear to correspond to the cruciform model of the striate cortex. The late positivity being maximal over the extremes of the montage may fit with V2 being the more likely site of generation. The vertical meridian of V2 is positioned adjoining the striate cortex, with the central projection over the lateral cortical surface. This does not however fit with the ipsilongitudinal distribution of this peak following altitudinal half field stimulation.

N145 was found to possess a similar distribution to that of the major positivity, P100 following half and quadrant stimulation. The amplitude was also significantly larger following lower half field stimulation ($p<0.05$). However, in contrast to the P100 there was no significant latency or amplitude effect of central occlusion or reduction of field size. So, while the two components did possess similar distributions they did not display exactly the same latency and amplitude trends. It has previously been proposed that the N145 component originates in the

extrastriate regions (Schroeder 1990) and therefore has a different source to the P100. The topographical distribution of this component is very similar to that of the P100 thus indicating a similar source region. The fact that the same trends with respect to latency and amplitude were not observed following central and peripheral stimulation implies that different processing in the same cortical area should not be excluded.

A large N145 component was produced after stimulation with the lower opposing dioctant stimulus, this would correspond with the significantly larger response following lower field stimulation. N145 was also recorded following left and right non-opposing dioctant stimulation, see fig 9. The vertical octants would therefore appear to produce a greater N145 response.

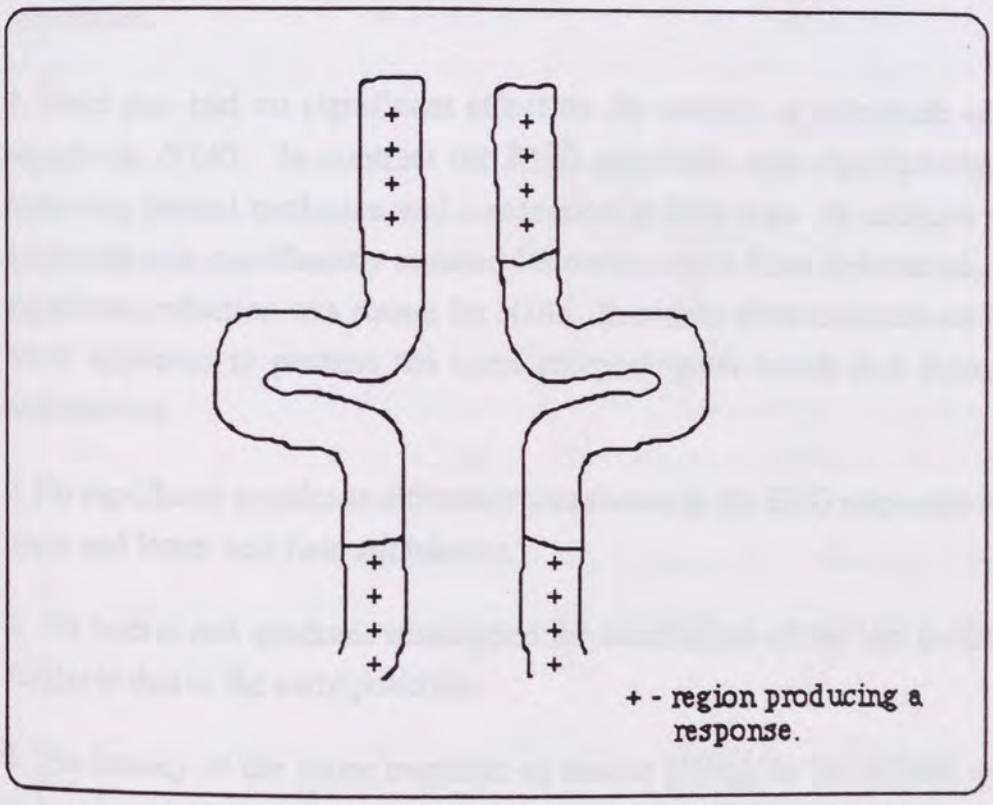


Fig. 9.18. Possible region of extrastriate cortex generating the N145 component.

Following octant stimuli N145 was again maximal following lower field stimulation, a low amplitude response was shown after upper field stimulation. The distribution was more localised with lower vertical octant stimulation and maximal over the left anterior region, the distribution being diffuse over the anterior right area of the montage following lower horizontal stimulation. The

response distribution following lower vertical octant stimulation was similar to that after lower opposing dioctant stimulation.

As a result of different optimal stimuli and the fact that there was no significant effect of field location on the amplitude and latency of N145 it can not be ruled out that N145 may be generated in a different visual area when compared with the P100, as suggested by intracortical studies (Schroeder et al 1990).

In Summary

1. Investigation of the pattern reversal response from the altitudinal half fields failed to demonstrate any latency shift in the major positive P100 response following upper field stimulationwhen compared with both lower and full field stimulation.

2. Field size had no significant effect on the latency or amplitude of the late negativity, N145. In contrast the P100 amplitude was significantly reduced following central occlusion and a reduction in field size. In addition the P100 amplitude was significantly reduced following upper field stimulation, a similar significant reduction was shown for N145. In certain stimulus locations P100 and N145 appeared to possess the same morphological trends and topographical distributions.

3. No significant amplitude difference was shown in the ERG responses following upper and lower half field stimulation.

4. On lateral and quadrant stimulation the distribution of the late positivity was similar to that of the early positivity.

5. The latency of the major response, at around 100ms, in the VEMR was found to be longer when compared with the equivalent VEP. The same trend of an increase in the response latency with a reduction in check size was however found for both responses.

6. The topographical distribution of the VEMR and subsequent dipole fits following full and lateral half field stimulation would imply that the source of the major positivity recorded after pattern reversal stimulation is located in the striate or neighbouring extrastriate cortex.

7. The distribution and morphology of the P100 peak after octant and double octant stimulation suggest a source located in the striate cortex.

8. The distribution of the major positive response following half field stimulation can be explained from the distribution of responses following octant stimulation along the vertical and horizontal meridia. Both the lateral and altitudinal half field responses appear to be more dependant on stimulation of the vertical meridia.

Future Plans

Design of stimuli which will preferentially stimulate individual visual areas may lead to a greater insight into the functional characteristics of these different visual areas of the human brain. Implementation of magnetometry in this area may give an increase in source localisation accuracy. Consideration of both the location of the stimulated area with respect to the magnetometer and the size of the stimulated area must be made. The use of a multichannel system would be advantageous, not only to reduce the length of recording sessions but reduce the variablility within the recordings.

Simultaneous recordings of both the VEMR and VEP may assist with component classification, in addition further investigation of radial versus tangential sources may be made. In addition the VEMR following quadrant stimulation may give further insight into the sources producing the VEP distribution. The use of the dioctant stimuli in conjunction with the magnetometer may reinforce the proposal that the initial upper half field response is the result of sources positioned along the longitudinal fissure.

Investigation of the stimulus characteristics of the peaks in the pattern reversal VEP may assist in source location, e.g.the effects of contrast, spatial frequency and chromatic stimuli.

The topographic distributions of the responses to both central and peripheral stimulation may be made more confused and complicated with central and peripheral interactions. Study of central and peripheral stimulation with dioctant stimuli may produce simpler and more conclusive maps to interpret.

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APPENDICES

APPENDICES

Subject	FULL FIELD	FF P100	FF P120	FF N145
RD	72	94	116	152
CD	76	102		150
BE	78	106	114	144
PF	74	102		132
MN	74	98		138
JN	76	110		164
NP	74	98		140
KR	84	114		156
TS	76	98		134
DT	76	100		128
VV	78	106		140
EW	76	114	118	160
Mn (S.D.)	76.17 (3.01)	103.5 (6.56)	116 (2)	144.8 (11.52)

Subject	RHF IN75	RHF CP80	RHF IP100	RHF CN105	RHF P120	RHF IN145
RD	68	78	94		118	150
CD	76		110			158
BE	76	76	108	102		154
PF	74		102			138
MN	74		100		126	154
JN	70		104			162
NP	72	83	94	98	110	132
KR			116	98		162
TS	74	74	104	106		138
DT	78	78	94	108		124
VV	76		106			144
EW	74		116		136	178
Mn (S.D.)	73.8 (2.89)	79.83 (5.81)	104 (7.77)	104.44 (6.23)	122.5 (11.17)	149.5 (15.04)
Subject	LHF IN75	LHF CP80	LHF IP100	LHF CN105	LHF P120	LHF IN145
RD	74	86	92		118	150
CD	76		102			152
BE	78	78	104	104	134	148
PF	76		100			142
MN		84	100		114	140
JN	78	78	94			150
NP	76	84	96	102	118	138
KR	88		116		126	150
TS	74	78	100			132
DT	76	88	104			142
VV	80	90	106	112		140
EW	76	80	114	102		158
Mn (S.D.)	77.45 (3.91)	82.89 (4.59)	102.33 (7.23)	103.43 (4.58)	122 (8)	145.17 (7.31)

A.1 Table of individual subject latencies (ms) for all the major components following full field (FF) left half field (LHF) and right half field (RHF) stimulation. Key C = Contralateral and I = ipsilateral.

Subject	FF N75	FF P100	FF LATE P	FFN145
RD	-2.21	6.37	5.01	-3.54
CD	1.41	9.94		-10.06
BE	-5.16	10	7.29	-7.28
PF	-2.87	6.9		-3.85
MN	-0.16	12.36		-11.73
JN	-2.2	5.89		-1.88
NP	1.19	8.06		-6.17
KR	0.45	11.26		-8.5
TS	-4.66	10.4		-8.07
DT	-7.54	10.74		-6.22
VV	-6.5	7.05		-10.18
EW	-4.64	8.87	2.29	-4.59
Mn(S.D.)	-2.74 (3.02)	8.98 (2.11)	4.86 (2.50)	-6.84 (3.02)

Subject	RHF IN75	RHF CP80	RHF IP100	RHF CN105	RHFP120	RHF IN145
RD	-0.02	3.44	6.17		4.63	-2.5
CD	-2.7		5.06			-8.34
BE	-2.56	6.07	6.38	-1.8		-4.85
PF	-1.39		4.82			-1.25
MN	-1.93		6.28		2.25	-8.65
JN	-2		3.93			-3.28
NP	-0.22	3.04	4.58	0.63	1.27	-3.88
KR			11.32	2.93		-5.2
TS	-7.23	2.24	6.71	6.23		-3.65
DT	-2.47	3.74	4.77	-4.47		-5.51
VV	-3.68	4.66	6.55			-6.05
EW	-3.6		5.75		4.28	-2.75
Mn (S.D.)	-2.53 (1.95)	3.87 (1.34)	6.03 (1.89)	0.704 (4.14)	3.39 (1.85)	-4.66 (2.25)

Subject	LHF IN75	LHF CP80	LHF IP100	LHF CN105	LHF P120	LHF IN145
RD	0.81	2.07	4.28		2.65	-1.85
CD	-0.49		5.74			-5.75
BE	-3.43	2.77	5.67	-1.35	4.59	-6.78
PF	-2.18		3.46			-6.43
MN	-0.69	6.47	8.34		3.87	-7.29
JN	-0.54	3.21	6.58			-2.06
NP	-0.11	3.6	4.95	-0.46	3.13	-3.58
KR	-1.39		6.42		4.41	-2.57
TS	-1.47	5.14	8.51			-3.15
DT	-3.38	2.32	8.53			-2.92
VV	-2.84	3.33	2.74	-2.61		-5.25
EW	-1.28	2.95	6.67	-2.85		-3.06
Mn (S.D.)	-1.48 (1.37)	3.54 (1.41)	5.99 (1.92)	-1.82 (1.12)	3.69 (0.95)	-4.22 (1.95)

A.2 Table of individual subject amplitudes (μV) for all the major components following full field (FF) left half field (LHF) and right half field (RHF) stimulation. Key C = Contralateral and I = ipsilateral.

Subject	FF4 N75	FF4 P100	FF4 N145	FF7 N75	FF7 P100	FF7 P120	FF7 N145
RD	78	102	150	78	96	116	144
CD	80	104	158	78	106		148
BE		110	146	82	110	114	142
PF		102	136	72	98		134
MN	70	102	136		102		138
JN	78	110	154	56	116		150
NP	70	98	130	76	92		134
KR	60	116	170	94	110		174
TS	78	100	128	78	106	124	142
DT	78	100	134	78	102		128
VV	78	112	140	76	106	118	140
EW	76	114	154	72	114	126	154
Mn (S.D.)	74.6 (6.19)	105.8 (6.18)	143 (12.78)	76.36 (8.98)	104.83 (7.21)	119.6 (5.18)	144 (11.94)

Subject	CS4 N75	CS4 CP80	CS4 P100	CS4CN105	CS4 P120	CS4 N145
RD	70	86	90		116	152
CD			102			144
BE	76		106		116	142
PF			100			134
MN	74		96			136
JN	74		106			126
NP	70	90	96		114	134
KR		86	98		120	150
TS	70		100		104	132
DT	74		98			128
VV	80	90	106		126	160
EW		86	114		122	168
Mn (S.D.)	73.5 (3.51)	87.6 (2.19)	101 (6.29)		116.7 (7.01)	142.2(13.09)
Subject	CS7 N75	CS7 CP80	CS7 P100	CS7CN105	CS7 P120	CS7 N145
RD	70	88	92		114	156
CD			112			152
BE	76	92	104			154
PF	74		98		122	130
MN	72		98			136
JN	78		112		138	180
NP	70	90	96			134
KR			114			152
TS	76		100			134
DT	74		96			126
VV	78	96	106		134	162
EW	72	88	94			126
Mn (S.D.)	74 (2.98)	90.8 (3.35)	101.83(7.6)		127 (11.01)	146.9(16.38)

A.3 Table of individual subject latencies (ms) for all the major components following full field 7° (FF7), full field 4° (FF4), central scotoma 4° (CS4) and central scotoma 7° (CS7). Key C = Contralateral and I = ipsilateral.

Subject	FF4 N75	FF4 P100	FF4 N145	FF7 N75	FF7 P100	FF7 LATEP	FF7 N145
RD	-0.59	3.39	-3.68	-1.08	4.13	3.58	-1.99
CD	-0.83	7.17	-7.15	-0.4	9.87		-9.72
BE		6.52	-2.26	-1.75	8.81	7.23	-4.52
PF		6.69	-0.57	-0.18	5.46		-2.94
MN	-3.79	8.83	-3.96		7.25		-9.31
JN	-3.07	1.98	-2.35	-1.89	2.5		-2.31
NP	-3.5	5.62	-2.05	0.65	7.12		-8.1
KR	-3.36	6.25	-4.5	2.06	9.71		-4.68
TS	-2.74	4.74	-1.19	-2.64	4.26	5.42	-2.74
DT	-1.65	5.89	-0.51	-4.27	6.8		-3.83
VV	-1.21	7.01	-5.87	-3.48	6	5.6	
EW	-2.44	9.5	-3.48	-0.42	8.41	8.44	-1.93
Mn (S.D.)	-2.33 (1.18)	6.13 (2.09)	-3.13 (2.05)	-1.79 (1.44)	6.53 (2.69)	6.05 (1.86)	-4.73 (2.94)

Subject	CS4 N75	CS4 CP80	CS4 P100	CS4CN105	CS4P120	CS4 N145
RD	-1.31	3.81	5.18		3.92	-3.31
CD			7.03			-4.38
BE	-5.63		8.36		3.14	-4.33
PF			6.4			-1.7
MN	-0.05		7.76			-4.21
JN	-0.64		4.96			-0.77
NP	-1.58	2.95	5.61		4.1	-2.74
KR		3.11				-4.83
TS	-0.97		7.9		6.4	-4.41
DT	-4.95		9.36			-4.78
VV	-0.73	3.77	4.16		3.94	-5
EW		5.66	8	2.06	8.07	-2.38
Mn (S.D.)	-1.97 (2.12)	3.86 (1.08)	6.79 (1.65)	2.06 (0)	4.93 (1.89)	-3.57 (1.38)
Subject	CS7 N75	CS7 CP80	CS7P100	CS7N105	CS7 P120	CS7 N145
RD	-1.65	2.65	4.44		3.52	-0.33
CD			4.43			-3.59
BE	-2.18	2.97	5.37			-4.24
PF	-3.52		5.16		2.95	-5.93
MN	-2.11		5.59			-3.2
JN	-2.45		2.11		2.5	-2.8
NP	-1.89	4.95	6.11			-3.62
KR			3.84			-4.52
TS	-2.31		3.34			-4.73
DT	-2.06		9.53			-4.14
VV	-0.26	4.99	4.67		4.25	-2.52
EW	-2.49	2.2	3.16			-3.74
Mn (S.D.)	-2.09 (0.81)	3.15 (1.98)	4.81 (1.87)		3.31 (.76)	-3.61 (1.38)

A.4 Table of individual subject amplitudes (μV) for all the major components following full field 7° (FF7), full field 4° (FF4), central scotoma 4° (CS4) and central scotoma 7° (CS7). Key C = Contralateral and I = ipsilateral.

VEP

FIELD/SUBJ	RA	CD	CN	AS
LHF70	96	98	102	96
FF70	98	104	98	92
RHF70	94	106	90	90
LHF34	98	104	96	90
FF34	98	102	100	92
RHF34	100	98	96	92
LHF22	96	112	102	94
FF22	98	112	106	94
RHF22	100	114	102	100

VEF

FIELD/SUBJ	RA	CD	CN	AS
LHF70	102	118	124	113
FF70	101	118	121	118
RHF70	99	120	120	115
LHF34	113	126	124	114
FF34	114	135	124	115
RHF34	116	153	126	115
LHF22	117	145	127	144
FF22	120	139	125	144
RHF22	118	120	131	134

A.5 Table of latencies of major peak in the VEP and VEMR for all subjects following left half field (LHF), full field (FF), and right half field (RHF) stimulation with 70', 34' and 22' checks.

Subject	LOHF N75	LOHF P100	LOHF P120	LOHF N145
RD	60	92	118	158
CD	82	106	118	150
BE	78	104		142
PF	76	102	126	132
MN	60	102		140
JN	76	104		132
NP	72	94		134
KR		110	116	152
TS	70	104		136
DT	76	98		130
VV	80	110		142
EW	78	112		144
Mn (S.D.)	73.45 (7.43)	103.17 (6.18)	119.5 (4.43)	141 (8.84)
Subject	UPHF N75	UPHF P100	UPHF P120	UPHF N145
RD	74	92	118	156
CD	74	104	136	168
BE	80	114	124	154
PF	82	106		130
MN	68	96	136	138
JN		90	138	158
NP		96	126	166
KR		116	132	150
TS	78	102	114	164
DT	74	102		156
VV	82	112	132	158
EW	76	92	120	150
Mn (S.D.)	76.44 (4.56)	101.83 (8.92)	1127.6 (8.42)	154 (11.05)

A.6 Table of individual subject latencies (ms) for all the major components following upper (UPHF) and lower half field (LOHF) stimulation.

Subject	LOHF N75	LOHF P100	LOHFP120	LOHF N145
RD	-2.2	4.64	2.05	-2.64
CD	-0.09	7.94	5.29	-12.44
BE	-2.46	7.83		-6.56
PF	-1.52	5.2	3.01	-3.46
MN	-4.07	13		-10.41
JN	-0.96	7.08		0.29
NP	-0.46	10.74		-7
KR		9.35	6.57	-5.35
TS	-2.17	8.09		-6.36
DT	-1.2	15.15		-5.37
VV	-4.83	7.69		-6.13
EW	-0.18	6.04		-2.2
Mn (S.D.)	-1.66 (1.73)	8.56 (3.09)	4.23 (2.07)	-5.46 (3.48)
Subject	UPHF N75	UPHF P100	UPHFP120	UPHF N145
RD	-0.2	5.1	4.06	-3.35
CD	-0.27	6.38	2.57	-3.58
BE	-3.88	4.05	3.82	-3.63
PF	-2.56	3.14		-2.38
MN	0.71	7.71	3.17	-0.31
JN		3.4	1.94	-1.21
NP		3.25	5.17	-3.43
KR		4.91	4.75	-2.38
TS	-4.33	4.2	3.56	-3.48
DT	-8.09	4.64		-1.33
VV	-3.58	4.37	1.9	-3.41
EW	-2.03	2.31	3.86	-2.94
Mn (S.D.)	-2.69 (2.69)	4.46 (1.48)	3.48 (1.09)	-2.62 (1.12)

A.7 Table of individual subject amplitudes (μV) for all the major components following upper (UPHF) and lower half field (LOHF) stimulation.

N75

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	74	72	66	82	82
AS				78	86
CD	78	78	72	76	
DM					
DS	76	74	70		
MD	74	76	76	86	64
RD	76	74	72		
TS	76	76	74	74	72
BE	76	74	76	78	78
MB	78			88	82
Mean	76	74.85	72.29	80.28	77.33
S Devn	1.51	1.95	3.55	5.22	8.07
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP	76	76	70		
AS	74	70	78	80	78
CD	74	72			
DM		76	70		
DS			62		60
MD		78	70	74	76
RD	70	68	72	74	
TS	82	86	84		
BE	74	74	76	80	80
MB	72	76	74		
Mean	74.57	75.11	72.89	77	73.5
S Devn	3.78	5.21	6.17	3.46	9.15

A.8 Table of individual subject latencies (ms) for N75 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

N75

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	-1.51	-1.22	1.94	-1.52	-0.52
AS				-2.22	-2.61
CD	-1.58	-0.47	-2.46	-2.86	
DM					
DS	-1.29	-0.44	-0.15		
MD	-3.01	-0.41	-2.67	-1.26	-1.45
RD	-0.28	-0.9	-0.86		
TS	-10.1	-3.69	-5.55	-1.33	-0.9
BE	-3.18	-3.88	-2.08	-1.5	-0.35
MB	-3.42			-0.82	-0.64
Mean	-3.05	-1.57	-2.24	-1.64	-1.08
S Devn	3.05	1.54	1.71	0.68	0.84
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP	-2.83	0.78	-1.04		
AS	-3.96	-1.41	-0.53	-0.18	-1.48
CD		-2.29			
DM		-1.79	-1.87		
DS			-1.35		-0.86
MD		-1.55	-1.39	-0.83	-1.36
RD	-2.49	-0.98	-0.89	-0.37	
TS	-0.82	-1.5	-0.77		
BE	-2.75	-3.44	-1.95	-0.72	-0.38
MB	-3.79	-2.3	-3.52		
Mean	-2.77	-1.61	-1.48	-0.53	-1.02
S Devn	1.23	1.14	0.9	0.3	0.5

A.9 Table of individual subject amplitudes (μV) for N75 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

P100

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	96	94	94	126	122
AS	102	108	92	106	122
CD	104	108	108	118	118
DM	94	94	92	104	94
DS	96	94	90	104	96
MD	98	96	96	124	126
RD	92	94	94	100	114
TS	102	102	96	96	112
BE	112	120	116	114	108
MB	106	104	108	118	116
Mean	100.2	101.4	98.6	111	112.8
S Devn	6.14	8.74	8.79	10.38	10.76
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP	96	92	88	94	96
AS	102	100	100	100	88
CD	98	98	96	102	108
DM	94	96	102	94	88
DS	98	98	96	98	96
MD	96	98	92	96	94
RD	92	94	94	94	128
TS	98	98	108	90	
BE	102	100	102	98	102
MB	102	100	102	120	96
Mean	97.8	97.4	98	98.6	99.56
S Devn	3.46	2.67	5.89	8.27	12.36

A.10 Table of individual subject latencies (ms) for P100 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

P100

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	3.25	3.57	4.09	4.5	3.23
AS	3.16	7.37	4.83	1.41	2.62
CD	5.22	6.97	5.04	5.61	3.61
DM	4.04	3.5	2.73	2.74	3
DS	2.78	3	2.67	3.08	2.87
MD	4.2	5.8	3.96	2.68	3.44
RD	5.1	3.78	4.12	3.95	3.14
TS	2.12	6.47	4.95	2.95	6.77
BE	4.58	5.17	3.05	4.46	3.51
MB	1.8	5.25	5.3	6.64	5.22
Mean	3.62	5.09	4.07	3.8	3.74
S Devn	1.19	1.57	0.98	1.55	1.28
Subject	LOHF	LOP1-10	LOP2-10	LOC2	LOC1
NP	7.11	5.94	4.97	4.65	2.48
AS	6.01	8.01	5.86	4.19	3.94
CD	7.35	5.07	4.26	2.97	3.85
DM	4.82	3.96	5.66	3.99	2.29
DS	7.23	5.52	4.41	5.22	2.27
MD	5.47	4.69	4.14	5.03	1.88
RD	6.57	6.32	5.54	3.18	2.92
TS	5.37	3.5	3.51	6.84	
BE	7.21	8.36	4.7	4.18	2.4
MB	10.32	8.1	5.56	3.73	2.53
Mean	6.75	5.95	4.86	4.39	2.73
S Devn	1.55	1.74	0.78	1.12	0.72

A.11 Table of individual subject amplitudes (μV) for P100 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

P120

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	126				
AS					
CD					
DM					
DS	116				
MD				120	110
RD	114	122	116	112	
TS	126		126	120	
BE	134				
MB					
Mean	123.2	122	121	117.33	110
S Devn	8.19	0	7.07	4.62	0
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP					
AS					
CD	118			134	
DM					
DS	142		140		120
MD					128
RD	116	126	118	116	
TS					
BE					
MB					
Mean	125.83	126	129	125	124
SDevn	14.47	0	15.56	12.72	5.66

A.12 Table of individual subject latencies (ms) for P120 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

P120

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	5.17				
AS					
CD					
DM					
DS	2.71				
MD				2.65	2.94
RD	3.98	2.83	3.23	3.47	
TS	3.51		4.95	5.12	
BE	4.54				
MB					
Mean	3.98	2.83	4.09	3.75	2.94
S Devn	0.93	0	1.22	1.26	0
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP					
AS					
CD	5.29			2.05	
DM					
DS	3.06		3.03		2.27
MD					2.83
RD	1.05	2.68	3.88	3.33	
TS					
BE					
MB					
Mean	3.13	2.68	3.46	2.69	2.55
S Devn	2.12	0	0.6	0.91	0.39

A.13 Table of individual subject amplitudes (μV) for P120 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

N145

Subject	UPHF	UPP1-10	UPP2-10	UPC2	UPC1
NP	162	138	120		
AS	180	166	152		150
CD	152	140			
DM	142	132	148		
DS	134	132	156	162	148
MD	138			172	170
RD	156	154	154		160
TS	166		158		156
BE	146	162	164	164	160
MB	188	154	154		
Mean	156.4	147.25	150.75	166	157.33
S Devn	17.81	13.44	13.26	5.29	7.97
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP	140	134	148	130	160
AS	150	152	156	140	148
CD	150	138	132	140	148
DM	128	128	156	128	134
DS	128	128	126	134	
MD	142	138	140	152	
RD	148	168	162	158	164
TS	130	132	134	128	
BE	138	162	148	136	128
MB	154	130	162		
Mean	140.8	141	146.4	138.44	147
S Devn	9.72	14.52	12.89	10.52	14.07

A. 14 Table of individual subject latencies (ms) for N145 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

N145

Subject	UPHF	UPP1-10	UPP2-10	UC2	UC1
NP	-3.17	-3.4	-3		
AS	-5.64	-4.65	-5.91		-1.78
CD	-1.91	-2.75			
DM	-3.49	-1.66	-2.43		
DS	-1.7	-1.42	-0.94	-2.7	-1.87
MD	-3.36			-2.85	-2.7
RD	-3.35	-1.35	-1.31		-3.45
TS	-1.42		-2.12		-2.44
BE	-4.26	-1.43	-2.73	-3.02	-2.75
MB	-1.04	-1.22	-2.87		
Mean	-2.95	-2.24	-2.66	-2.86	-2.49
S Devn	1.47	1.25	1.5	0.16	0.62
Subject	LOHF	LOPP1-10	LOPP2-10	LOC2	LOC1
NP	-7.38	10	6.05	-2.43	-3.45
AS	-8.36	5.34	-4.75	-3.96	-3.56
CD	-5.16	-7.26	-4.83	-3.72	-0.2
DM	-5.11	-2.82	-1.91	-4.33	-4.15
DS	-3.45	-2.41	-3.08	-0.89	
MD	-2.83	3.57	-3.28	-1.12	
RD	-3.17	-0.32	-2.96	-2.14	-2.06
TS	-10.04	-7.64	-9.44	-7.84	
BE	-3.28	-3.81	-3.77	-5.07	-4.9
MB	-6.82	-4.74	-7.62		
Mean	-5.26	-4.8	-4.77	-3.5	-3.05
S Devn	2.51	2.86	2.34	2.17	1.68

A.15 Table of individual subject amplitudes (μV) for N145 following upper half field (UPHF), upper peripheral 1-10° (UPP1-10) and 2-10° (UPP2-10) and lower half field (LOHF), peripheral 1-10° (LOPP1-10) and 2-10° (LOPP2-10) stimulation.

Subject	LLOQ N75	LLOQ CP80	LLOQ P100	LLOQ CN105	LLOQP120	LLOQN145
RD		90	94		122	156
CD			98			150
BE		80	98		136	148
PF			98			130
MN	76	76	102			134
JN	74		110		122	144
NP	72	88	98		106	132
KR		84	114		122	162
TS	74	74	98			130
DT		88	10			130
VV	84		96		110	142
EW	70	80	112	100	118	
Mn (S.D.)	75 (4.86)	82.5 (5.93)	101.5 (6.67)	100 (0)	119.43 (9.71)	141.6(11.38)

Subject	RLOQ N75	RLOQ CP80	RLOQ P100	RLOQP120	RLOQN145
RD	70	80	92	122	126
CD	80		102		148
BE	76		106		142
PF	72		100		132
MN			96		140
JN	62	84	108		164
NP	74	90	94		130
KR		86	110	114	156
TS	72		102		134
DT	74		94	114	146
VV	76		94	106	148
EW		82	90	120	
	72.89 (5.01)	84.4 (3.85)	99 (6.63)	115.2 (6.26)	142.4(11.55)

A.16 Table of individual subject latencies (ms) for all the major components following left (LLOQ) and right lower quadrant (RLOQ) stimulation.

Subject	LLOQ IN75	LLOQ CP80	LLOQ P100	LLOQN105	LLOQP120	LLOQN145
RD		2.74	4.89		3.08	-1.43
CD			6.18			-5.22
BE		4.2	5.49		2.74	-4.73
PF			5.2			-1.3
MN	-0.4	1.7	9.26			-6.8
JN	-0.5		2.89		2.86	-2.33
NP	-1.09	2.03	6.12		3.02	-3
KR		3	8.42		4.88	-5.62
TS	-5.04	2.57	5.06			-4.66
DT		9.47	9.01			-2.93
VV	-2.75		4.47		5.11	-4.7
EW	-0.35	2.86	6.94	-0.01	2.96	
Mn (S.D.)	-1.69 (1.88)	3.57 (2.49)	6.16 (1.94)	-0.01 (0)	3.52 (1.01)	-3.88 (1.79)
Subject	RLOQ IN75	RLOQ CP80	RLOQ P100	RLOQN105	RLOQP120	RLOQN145
RD	-0.22	2.03	3.96		1.59	-1.35
CD	-0.85		5.3			-7.48
BE	-1.01		5.8			-4.31
PF	-2.55		4.89			-1.54
MN	-1.39		6.19			-8.73
JN		2.4	2.57			-4.01
NP	1.34	4.83	6.67			-0.83
KR		4.5	8.64		4.36	-2.58
TS	-4.63		7.07			-6.17
DT	-1.55		7.69		3.57	-2.74
VV	-2.42		4.07		3.91	-8.17
EW		5.55	4.89		3.92	-0.27
Mn (S.D.)	-1.85 (1.37)	3.86 (1.56)	5.65 (1.72)		3.47 (1.09)	-4.02 (2.97)

A.17 Table of individual subject amplitudes (μV) for all the major components following left (LLOQ) and right lower quadrant (RLOQ) stimulation.

Subject	LUPQ IN75	LUPQCP80	LUPQ P100	LUPQ CN105	LUPQP120	LUPQN145
RD			92		116	
CD	72	100	96			146
BE	76		108		116	144
PF	80		102			130
MN	68		100		138	152
JN	90		114			144
NP	74	86	94	100	128	140
KR		90	110			120
TS	74		106	102	114	136
DT	74		100			154
VV		84	108		128	162
EW	72	84	100		140	158
Mn (S.D.)	75.56 (6.31)	88.8 (6.72)	102.5 (6.78)	101 (1.41)	125.71 (10.73)	144.2(12.41)
Subject	RUPQ IN75	RUPQ CP80	RUPQ P100	RUPQ CN105	RUPQP120	RUPQN145
RD			104		138	
CD	82		108		134	164
BE	76		108		124	166
PF	80	98	106			134
MN	76		98			140
JN	64	86	116		124	174
NP	66	74	102		122	164
KR	86	86	102		108	152
TS	74	96	110		124	138
DT	82	82	94	108		
VV	82	98	106			150
EW	70	86	94		120	
Mn (S.D.)	75.2 (6.68)	88.25 (8.51)	104 (6.49)	108 (0)	124.25 (9.03)	153.56 (14.20)

A.18 Table of individual subject latencies (ms) for all the major components following left (LUPQ) and right upper quadrant (RUPQ) stimulation.

Subject	LUPQ IN75	LUPO CP80	LUPQ P100	LUPQN105	LUPQP120	LUPQ N145
RD			1.78		3.51	
CD	-1.06		5.67			-1.21
BE	0.45	5.86	5.05		3.97	-2.6
PF	-1.1		2.64			-2.37
MN	-1.71		3.98		4.53	-1.43
JN	-1.9		3.42			-2.55
NP	-2.21	1.12	4.46	-0.27	1.54	-1.25
KR		7.4	6.61			-1.46
TS	-2.3		5.65	1.41	4.67	1.32
DT	-5.2		4.6			-1.79
VV		4.83	4.95		4.3	-1.35
EW	-1.1	3.79	4.5		4.33	-2.33
Mn (S.D.)	-2.07 (1.36)	4.6 (2.36)	4.44 (1.35)	-0.27 (0)	3.84 (1.08)	-1.79 (0.56)
Subject	RUPQ IN75	RUPQ CP8C	RUPQ P100	RUPQN105	LUPQP120	RUPQ N145
RD			4.52		4.65	
CD	-1.4		6.53		6.88	-2.6
BE	-2.98		5.17		4.99	-1.24
PF	0.52	4.29	4.63			-0.77
MN	-2.03		3.13			-2.55
JN	-1.1	3	2.43		2.32	-2.46
NP	-1.95	2.94	4.67		3	-1.57
KR		4.05	8.1		8.11	-3.57
TS	-4.83	-0.35	6.23		5.33	-1.46
DT	-1.47	1.77	1.27	-5.59		
VV	0.33	3.96	4.37			-5.35
EW	-2.46	3.16	2.93		2.9	
Mn (S.D.)	-2.28 (1.19)	3.31 (0.87)	4.49 (1.89)	-5.59(0)	4.77 (2.02)	-2.39 (1.40)

A.19 Table of individual subject amplitudes (μV) for all the major components following left (LUPQ) and right upper quadrant (RUPQ) stimulation.

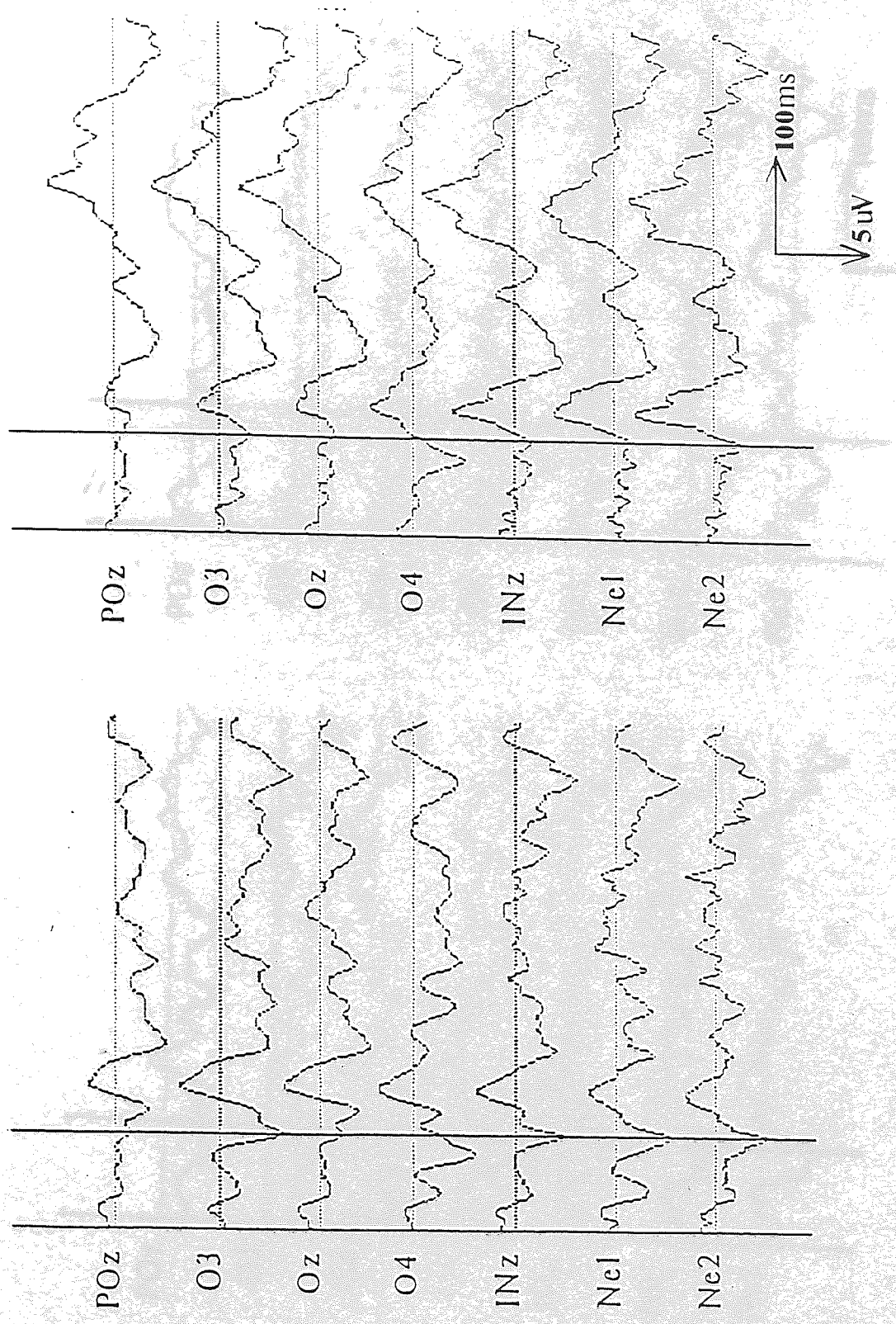

Fig.A.1 Waveform morphology following left upper quadrant (on the left) and left upper quadrant 1-10° stimulation. Subject V.V.

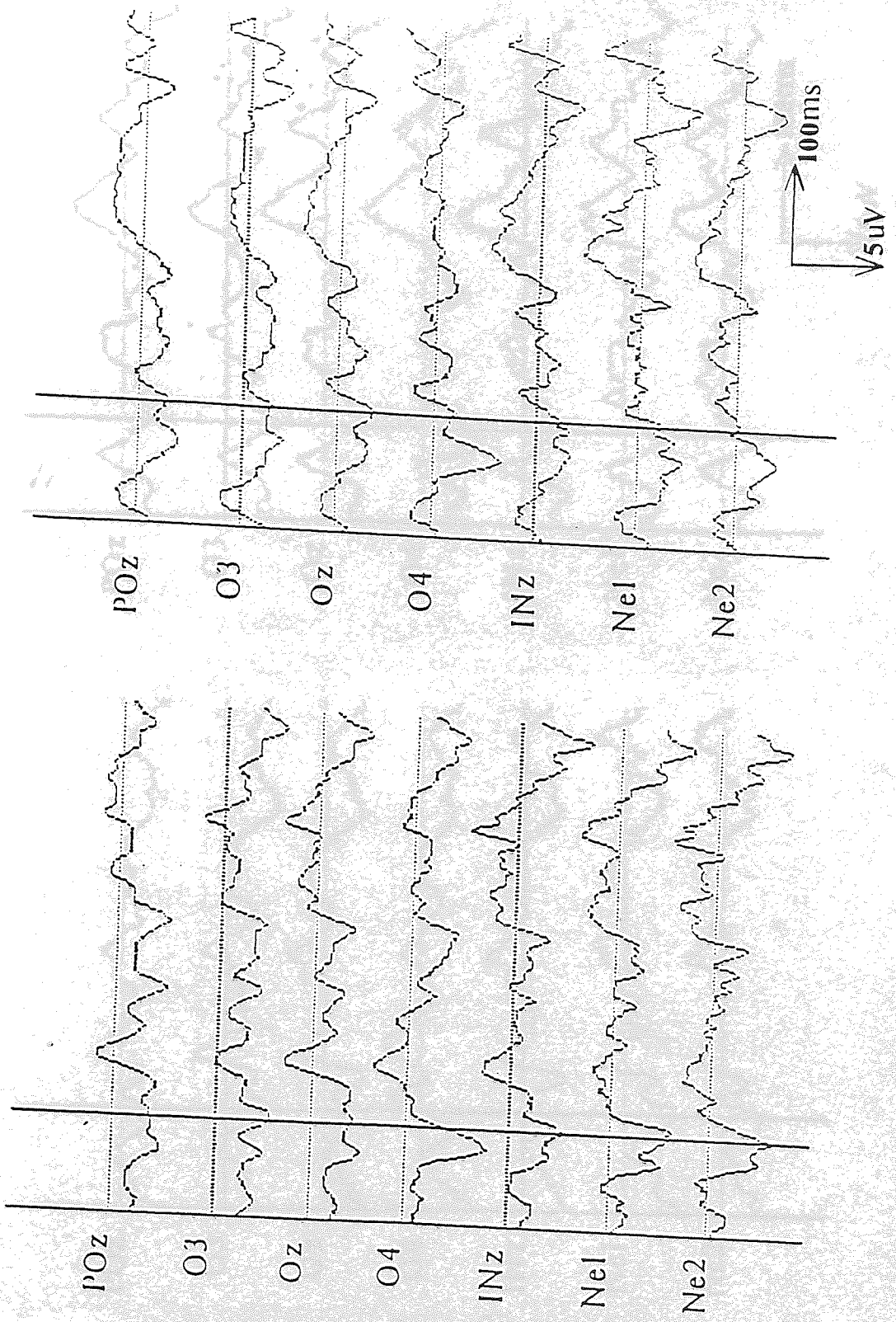

Fig.A.2 Waveform morphology following left upper quadrant 2-10° (on the left) and left upper quadrant 3-10° stimulation. Subject V.V.

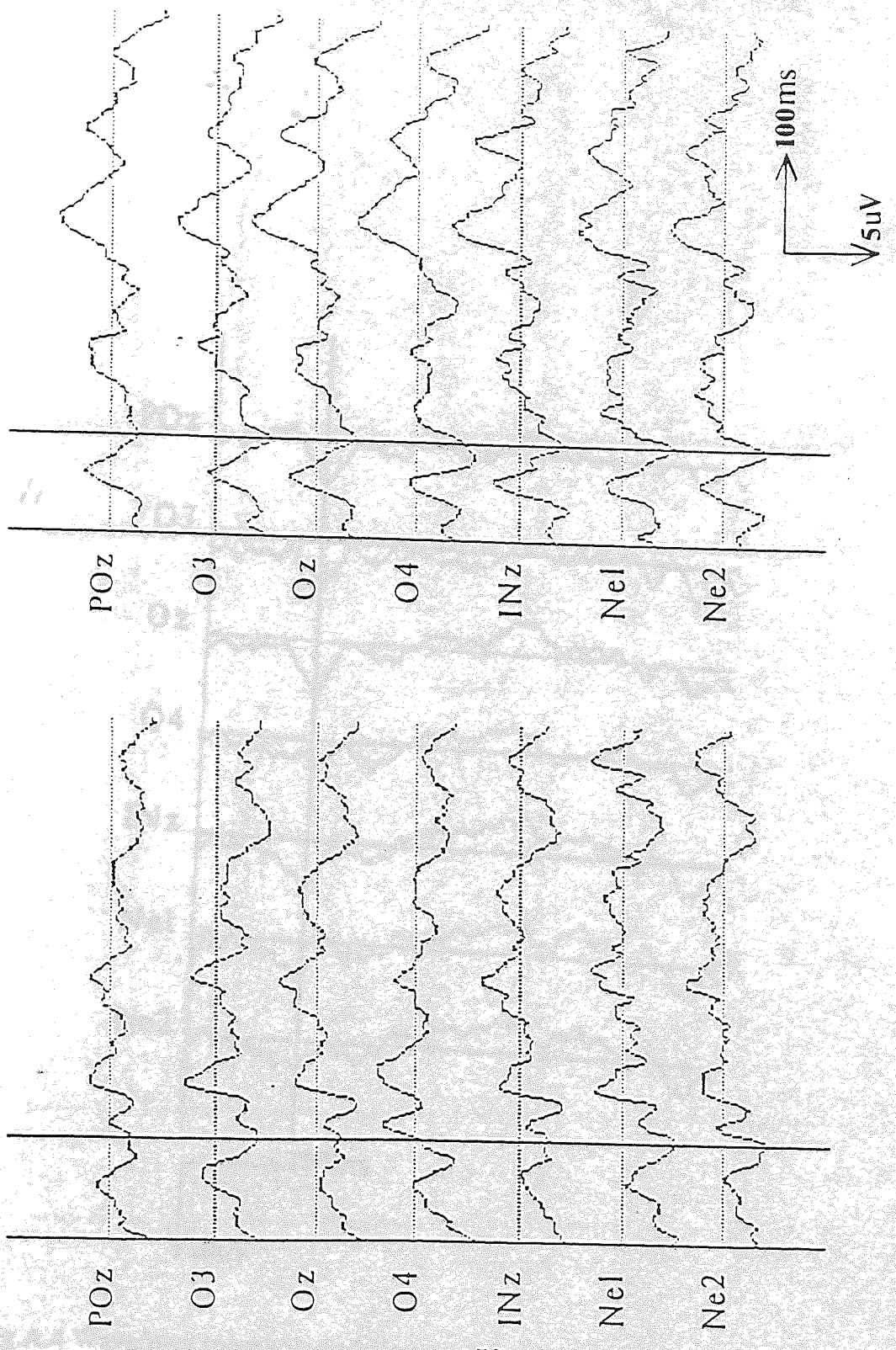

Fig.A.3 Waveform morphology following left upper quadrant 4-10° (on the left) and left upper quadrant 5-10° stimulation. Subject V.V.

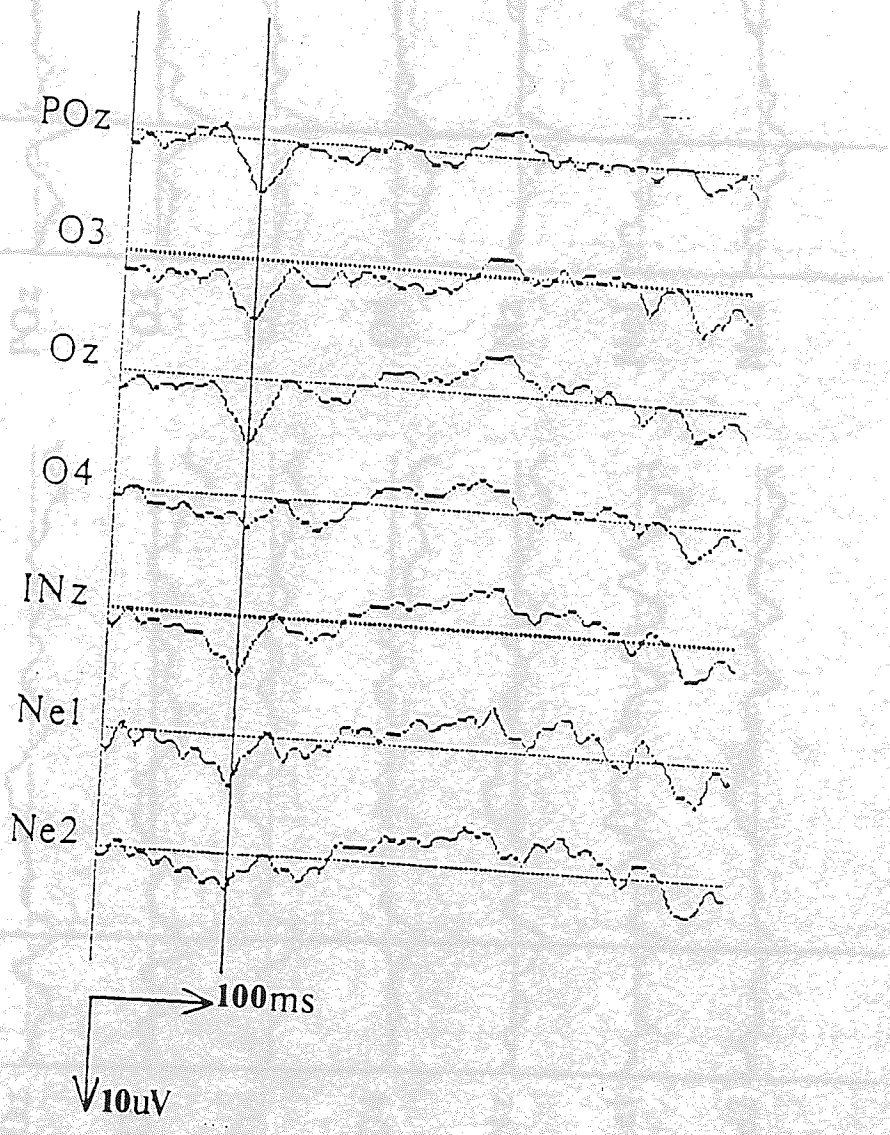

Fig.A.4 Waveform morphology following left upper quadrant stimulation. Subject G.B.

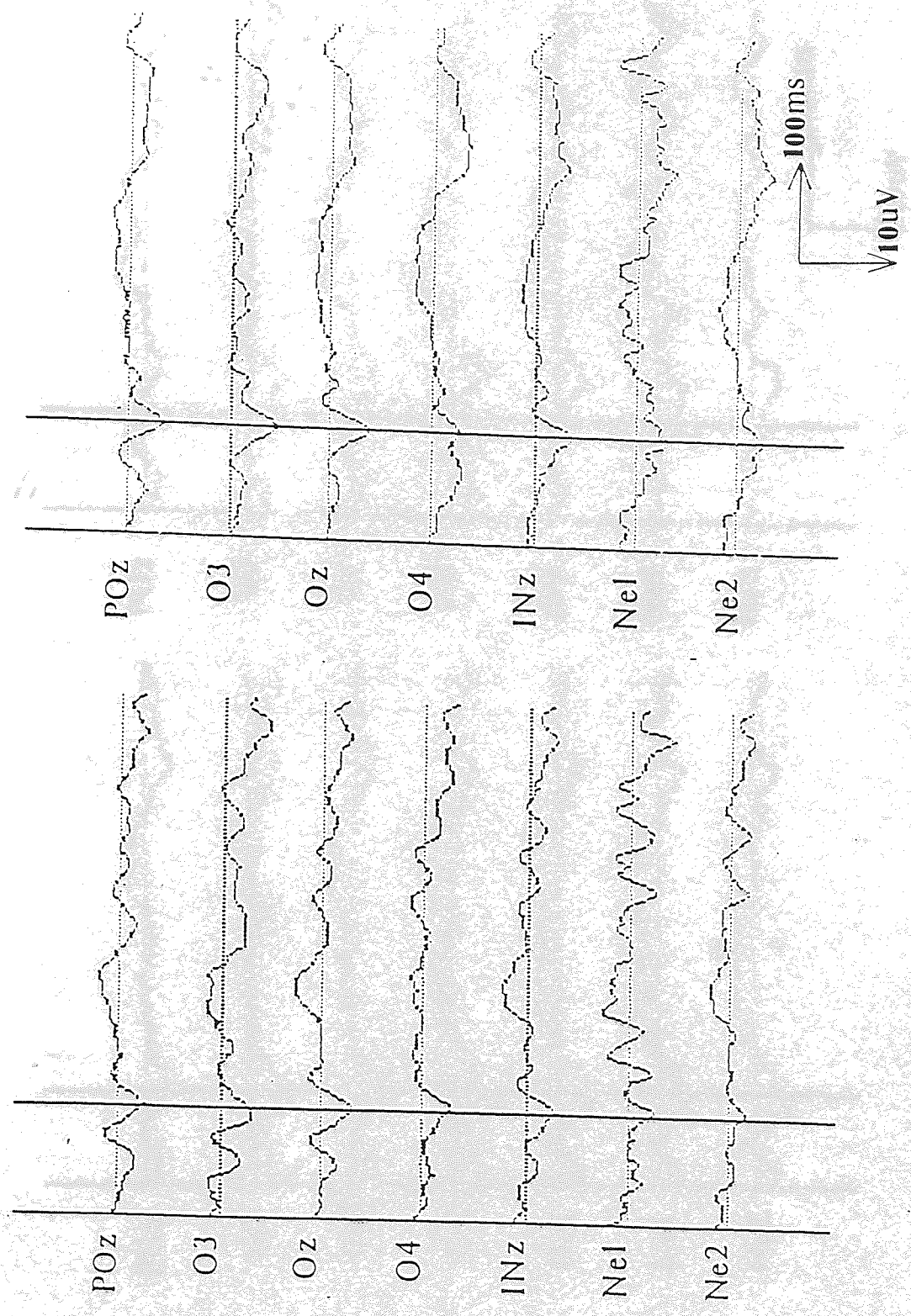

Fig.A.5 Waveform morphology following left upper quadrant 1-10° (on the left) and left upper quadrant 2-10° stimulation. Subject G.B.

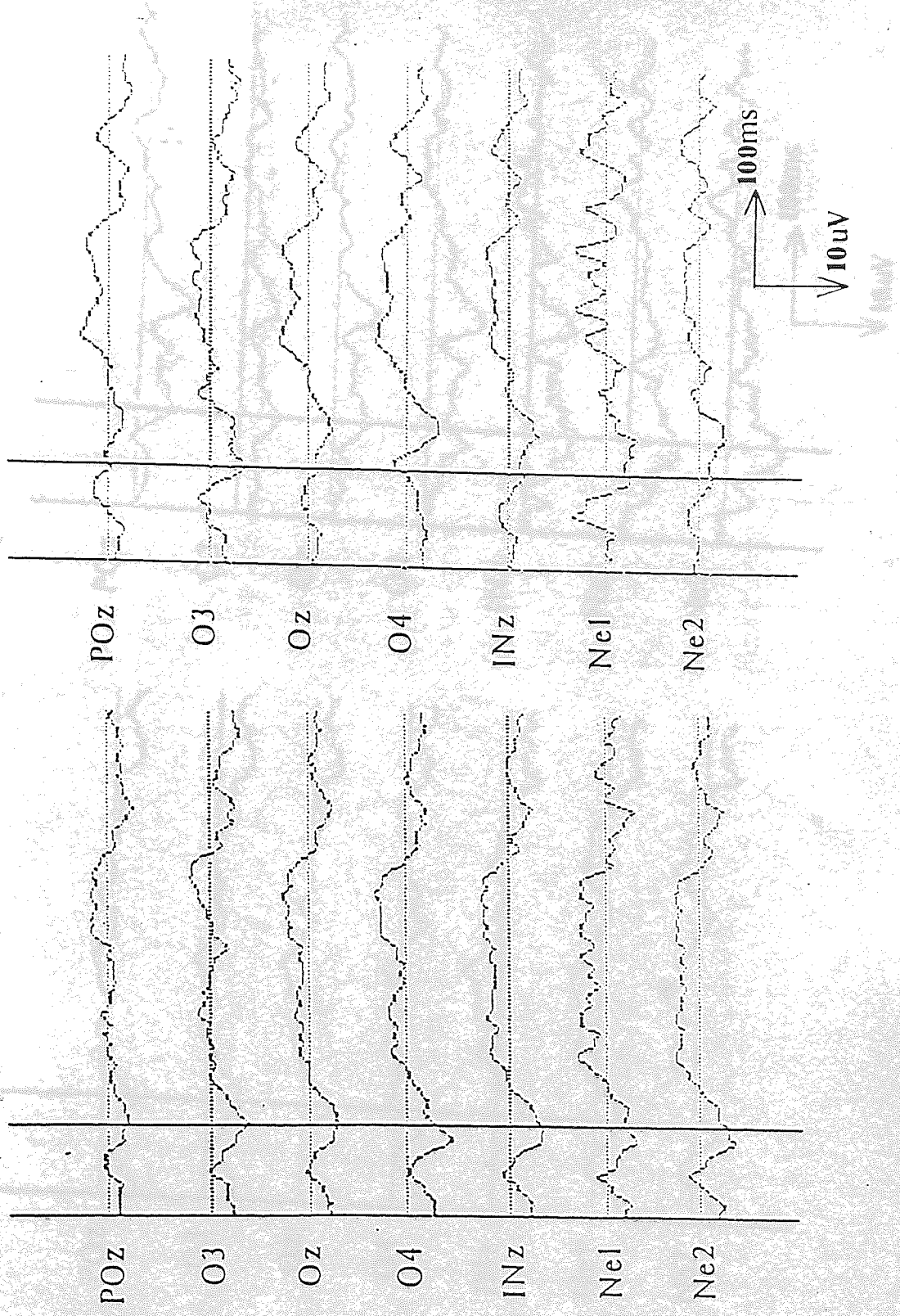

Fig.A.6 Waveform morphology following left upper quadrant 3-10° (on the left) and left upper quadrant 4-10° stimulation. Subject G.B.

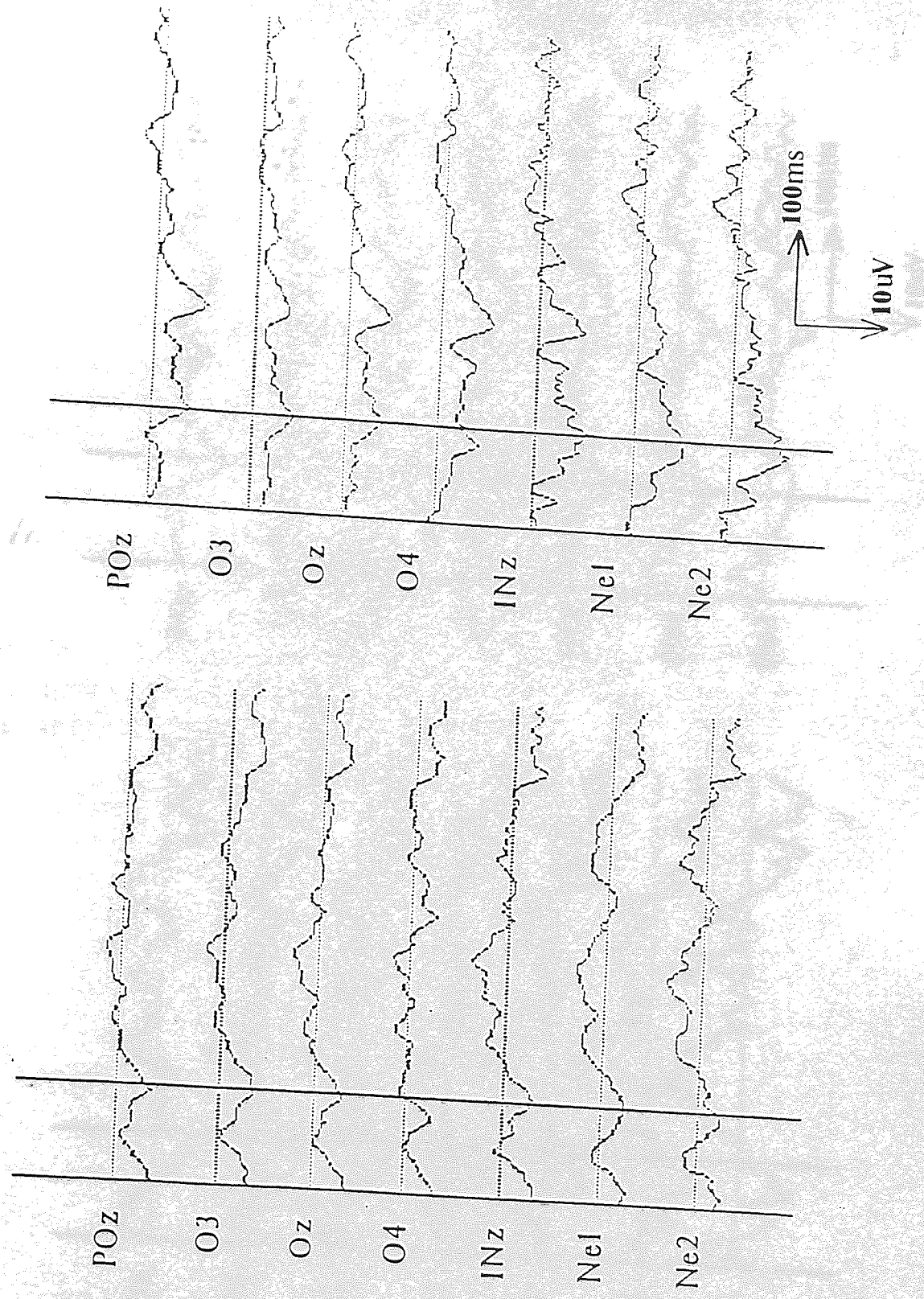

Fig.A.7 Waveform morphology following left upper quadrant (on the left) and left upper quadrant 2-10° stimulation. Subject E.W.

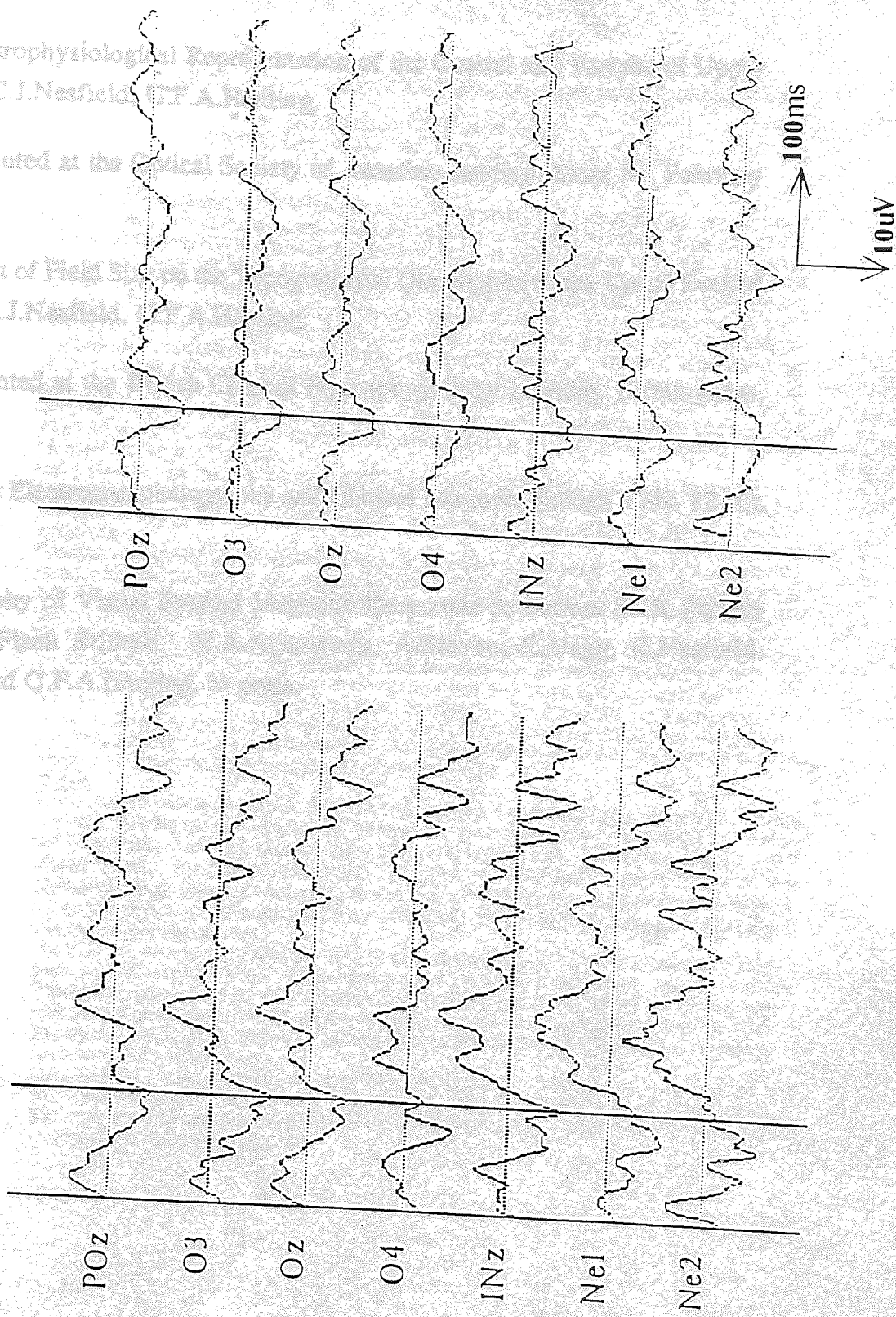

Fig.A.8 Waveform morphology following left upper quadrant 3-10° (on the left) and left upper quadrant 4-10° stimulation. Subject E.W.

List of Supporting Publications and Refereed Conference Presentations:

1. The Electrophysiological Representation of the Central and Peripheral Upper Half Field.C.J.Nesfield, G.F.A.Harding.

Poster presented at the Optical Society of America meeting. Santa Fe, February 1991.

2. The Effect of Field Size on the Topographical Distribution of the Visual Evoked Potential. C.J.Nesfield, G.F.A.Harding.

Paper presented at the British Clinical Neurophysiology meeting. Birmingham, June 1991.

Published in Electroencephalography and Clinical Neurophysiology 1992. 82 (1); 12P.

3. Topography of Visual Evoked Magnetic Responses to Pattern Shift, Pattern Onset and Flash Stimuli. R.A.Armstrong, A.Slaven, C.Degg, C.Nesfield, B.Janday and G.F.A.Harding. in press.

Electrophysiological Representation of the Central and Peripheral Upper Half Field.

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Aston University

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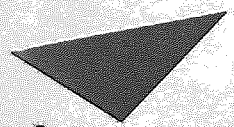
The Effect of Field Size on the Topographic Distribution of the Visual Evoked Potential (VEP).

C.J.Nesfield and G.F.A.Harding (Aston University, Birmingham)



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Topography of visual evoked magnetic responses to pattern shift, pattern onset and flash stimuli

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