Report PERS 419 1987

The Diagnostic Role of Some Electrophysiological Procedures in Dementia

B.R. Mallinson

001.3072068 HSRC
NIPR PERS 419

Nasionale Instituut vir Personeelnavorsing — NIPN
National Institute for Personnel Research — NIPR

RGN·HSRC

BIBLIOTEEK
LIBRARY

RGN

RAAD VIR
GEESTESWETENSKAPLIKE
NAVORSING

HSRC

HUMAN
SCIENCES RESEARCH
COUNCIL

RGN·HSRC

071728

RGN·HSRC

SENTRUM VIR BIBLIOTEEK- EN INLIGTINGSDIENSTE

CENTRE FOR LIBRARY AND INFORMATION SERVICES

VERVALDATUM/DATE DUE

1179*1085

0001541101

001.3072068 HSRC NIPR PERS 419

154110

The Diagnostic Role of Some Electrophysiological Procedures in Dementia

PERS 419

The Diagnostic Role of Some Electrophysiological Procedures in Dementia

B.R. Mallinson

Pretoria
Human Sciences Research Council
1987

RGN BIBLIOTEEK LIBRARY HSRC
1987 -07- 03
STANDKODE
001-3072068 HSRC
NIPR PERS 419
BESTELNOMMER
R/0
REGISTERNOMMER
071728

B.R. Mallinson, M.Sc., Chief Researcher

National Institute for Personnel Research
Executive Director: Dr G.K. Nelson

ISBN 0 7969 0491 X

© Human Sciences Research Council, 1987

Printed and published by HSRC
134 Pretorius Street

ACKNOWLEDGEMENTS

Mr B.D. Murdoch, Head, Division of Neuropsychology, NIPR

Dr G.K. Nelson, Executive Director, NIPR

CONTENTS

		Page
1.	INDEX OF TABLES	vii
2.	INDEX OF FIGURES	x
3.	EKSERP	
	ABSTRACT	xii
4.	INTRODUCTION	1
5.	SUBJECTS	7
6.	PROCEDURE	
	EEG	10
	FFT	10
	PRVEP	12
7.	RESULTS	
	EEG	13
	FFT	20
	PRVEP	55
8.	DISCUSSION	68
9.	REFERENCES	83

INDEX OF TABLES

Table		Page
1	Diseases with accompanying Dementia	3
2	Age and sex distribution for Alzheimer's and Multi-infarct groups	9
3	Age distribution of Alzheimer's and Multi-infarct subjects	9
4	C T scan atrophy ratings for Alzheimer's and Multi-infarct groups	11
5	Distribution of subjects according to C T scan atrophy rating	11
6	Distribution of subjects according to EEG abnormality rating	15
7	Lateralization of EEG abnormalities in Alzheimer's and Multi-infarct groups	15
8	Distribution of subjects (both groups) according to dominant EEG frequency	21
9	Group dominant EEG frequency according to age	21
10	Spectral Analysis: Alzheimer's and Multi-infarct groups (absolute scores)	26
11	Spectral Analysis: Alzheimer's and Multi-infarct groups (proportional scores)	29
12	Spectral Analysis: Left/Right hemisphere comparisons for Alzheimer's group (absolute scores)	32

Index of Tables continued:

Table		Page
13	Spectral Analysis: Left/Right hemisphere comparisons for Multi-infarct group (absolute scores)	33
14	Spectral Analysis: Left/Right hemisphere comparisons for Alzheimer's group (proportional scores)	34
15	Spectral Analysis: Left/Right hemisphere comparisons for Multi-infarct group (proportional scores)	35
16	Spectral Analysis: Eyes Open/Closed comparisons for Alzheimer's group (absolute scores)	36
17	Spectral Analysis: Eyes Open/Closed comparisons for Alzheimer's group (proportional scores)	39
18	Spectral Analysis: Eyes Open/Closed comparisons for Multi-infarct group (absolute scores)	41
19	Spectral Analysis: Eyes Open/Closed comparisons for Multi-infarct group (proportional scores)	42
20	EEG reactivity ratios for left and right hemisphere for Alzheimer's and Multi-infarct groups	43
21	Spectral Analysis for all subjects for left hemisphere, eyes closed, according to age (absolute scores)	44
22	Spectral Analysis for all subjects for left hemisphere, eyes closed, according to age (proportional scores)	45
23	EEG reactivity ratios for total group according to age	49

Index of Tables continued:

Table		Page
24	Spectral Analysis for all subjects for left hemisphere, eyes closed, according to EEG abnormality rating (absolute scores)	50
25	Spectral Analysis for all subjects for left hemisphere, eyes closed, according to EEG abnormality rating (proportional scores)	51
26	EEG reactivity ratios according to abnormality category	54
27	PRVEP abnormality for Alzheimer's and Multi-infarct groups	60
28	PRVEP abnormality and age for Alzheimer's and Multi-infarct groups	60
29	PRVEP abnormality and CT scan atrophy rating for Alzheimer's and Multi-infarct groups	61
30	PRVEP abnormality and EEG abnormality rating for Alzheimer's and Multi-infarct groups	61
31	P100 latencies for Alzheimer's, Multi-infarct and a control group	62
32	P100 amplitude for the Alzheimer's and Multi-infarct groups	63
33	PRVEP latency and age for all subjects compared with a control group	64
34	P100 amplitude and age for all subjects	65
35	P100 Latency and EEG abnormality rating for all subjects compared with a control group	66
36	P100 amplitude and EEG abnormality rating for all subjects	67

INDEX OF FIGURES

Figure		Page
1	EEG showing normal activity from a 70-year-old female with Alzheimer's disease	16
2	EEG showing generalised slowing of the dominant frequency to 7 Hz from a 74-year-old female with Alzheimer's disease	18
3	EEG showing generalized slowing of the dominant frequency and 2 to 3 Hz activity in the left temporal area from a 76-year-old male with Alzheimer's disease	19
4	Distribution of subjects in Alzheimer's disease and Multi-infarct groups according to frequency band (7 to 11 Hz)	22
5	Mean alpha frequency for each group according to age	23
6	Proportional power for the present group compared to results in the literature	25
7	Spectral Analysis: Alzheimer's and Multi-infarct groups (absolute scores)	28
8	Spectral Analysis: Alzheimer's and Multi-infarct groups (proportional scores)	31
9	Spectral Analysis: Eyes open/eyes closed (absolute scores)	37
10	Spectral Analysis: Eyes open/eyes closed (proportional scores)	40
11	Spectral Analysis: Frequency band and age for combined groups (absolute scores)	46

Index of figures continued:

Figure		Page
12	Spectral Analysis: Frequency band and age for all subjects (proportional scores)	47
13	Spectral Analysis: Frequency band and EEG abnormality rating for all subjects (absolute scores)	52
14	Spectral Analysis: Frequency band and EEG abnormality for all subjects (proportional scores)	53
15	A normal PRVEP from a 78-year-old female with Alzheimer's disease	57
16	Bilaterally delayed PRVEP from a 74-year-old male with Alzheimer's disease	58
17	Left-sided delay in PRVEP from a 82-year-old female with Alzheimer's disease	59

EKSERP

Elektroënkefalogramme (EEGs) en patroon-omgekeerde visuele ontlokte potensiale (POVOPs) is van 42 verouderde proefpersone met demensie (gemiddelde ouderdom 77,0 jaar, SA 7,1) opgeneem. Die proefpersone is in groepe ingedeel volgens ouderdom en diagnose (Alzheimer se siekte (AS) en Veelvoudige Infark-demensie). Die EEGs is volgens 'n standaard kliniese benadering en ook 'n objektiewe Vinnige Fourier Transformasie (VFT) ontleed.

Die standaard kliniese EEG was sensitief ten opsigte van brein disfunksie, en 69% van die proefpersone het abnormale EEGs gehad. Vertraging van die dominante frekwensie (alfa-ritme) en fokale stadige aktiwiteit in die linker temporale gebied was die algemeenste abnormaliteite. Die alfa-ritme het ook met toenemende ouderdom stadiger geword. Die VFT was relatief onsensitief behalwe vir 'n meting van reaksie op oë toemaak.

POVOPs was abnormaal by 29% van die proefpersone wat moontlik met periferale visuele disfunksie verband hou. Resultate word met dié in die literatuur vergelyk. Die etiologie van AS word bespreek.

ABSTRACT

Electroencephalograms (EEGs) and pattern reversal visual evoked potentials (PRVEPs) were recorded from 42 elderly subjects with dementia (mean age 77,0 years SD 7,1). Subjects were grouped according to age and diagnosis (Alzheimer's disease (AD) or Multi-infarct dementia). The EEGs were analysed using a standard clinical approach as well as an objective Fast Fourier Transformation (FFT).

The standard clinical EEG proved sensitive to brain dysfunction and 69% of the subjects had abnormal EEGs. The most common abnormalities were slowing of the dominant frequency (alpha rhythm) and focal slow activity in the left temporal area. The alpha rhythm slowed with increasing age. The FFT was relatively insensitive apart from a measure of reactivity to eye closure.

PRVEPs were abnormal in 29% of the subjects, perhaps related to peripheral eye dysfunction. Results are compared with those in the literature. The etiology of AD is discussed.

INTRODUCTION

It is widely believed that over the years medical science has lengthened the life span of man. Adams and Victor (1977), however, point out that since biblical times when man's lifespan was regarded as "three score years and ten" human life has not lengthened greatly. What has increased, chiefly as a result of reduced infant mortality and the elimination of fatal infectious diseases, is the number of people reaching old age. This increase, together with a decreased fertility rate in recent years, has resulted in a dramatic increase in the percentage of old people in the population of developed countries. In the USA in 1900, 4% of the population were over 65 years of age. In 1977 this figure had risen to 10% and the projected figures for the year 2030 are 17% to 23% (Besdine, 1980). In the RSA, according to the 1980 census, 7,8% of the white population was over 65 years of age. Although the proportion of elderly individuals in the black population is much lower (3,09%), there is a large number of elderly black people in the RSA due to the much greater size of this population, (Mostert, 1982). The Centre of Applied Social Science at the University of Natal, Durban, indicates that by the year 2020 there will be 4 241 700 black people over the age of 65 in the RSA. In the same year there will be 1 304 790 white people in this age group (Tollman, 1985).

Not only is the demography of the elderly changing, but also their lifestyle and aspirations. A pamphlet published by the National Institutes of Health in the USA (NIH Publication no.

81-85, 1980) points out that in the early part of this century very few of the elderly lived alone (10%). By 1970, however, a quarter of the population over 65 years old were living independently. In 1900, work was a lifelong affair whereas in 1980 most workers retired after 65 years of age and work had become a phase of life with the majority of people expecting to live for many years after retirement.

The educational level of the elderly population has also changed. In the USA in 1960, 10% of the elderly had had four years of high school education. By 1980 the figure had risen to 20%. A similar increase (from 3,7% to 7,6%) was seen in the number of old people with a college education (NIH Publication no. 81-85, 1980).

These factors have resulted in an increase in society's concern for the wellbeing and quality of life of the aged. The writer of the book of Ecclesiastes says, "and therefore if a man lives many years, let him enjoy all of them". One of the most prevalent health problems of the elderly and one which markedly affects their quality of life, is dementia. Dementia may be defined as, " (the) loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning. The deficit is multifaceted and involves memory, judgement, abstract thought and a variety of other higher cortical functions " (DSM III, 1980).

There are many causes of dementia (Table 1), but in the elderly the most common are Alzheimer's disease (AD) and

Table 1

Diseases with accompanying Dementia

- Alzheimer's disease
- Pick's disease
- Extrapyramidal syndromes with dementia
  - Parkinson's disease
  - Huntington's disease
  - Wilson's disease
  - Progressive supranuclear palsy
  - Spinocerebellar degenerations
  - Miscellaneous extrapyramidal syndromes
- Multi-infarct dementia
- Viral and other infectious dementias
  - Creutzfeldt-Jacob disease
  - Syphilis
  - Chronic meningitis
  - Miscellaneous CNS infections
- Toxic and metabolic dementias
- Hydrocephalic dementias
- Traumatic dementias
- Neoplastic dementias
- Myelin disease with dementia
- Dementias associated with psychiatric disorders
  - Depression
  - Schizophrenia
  - Miscellaneous psychiatric disorders

(from Cummings, 1985)

Multi-infarct Dementia (MID), with the former being the most prevalent.

The cause of AD is unknown but it is associated with diffuse cerebral atrophy. At autopsy, numerous plaques can be seen throughout the cortex and neurofibrillary tangles are seen in the neurons (Adams and Victor, 1977). Some of these changes, however, are also present in the brains of non-demented aged people and AD was therefore regarded as an exaggeration of normal ageing. This has recently been refuted by several authors (Berg, 1985; Kachaturian, 1985) and the issue is still being hotly debated. There is some evidence of genetic susceptibility, as first-degree relatives are four times more likely to develop the disease than members of the general population (DSM III, 1980). Cummings (1985) claims that in 20% of cases the disease is inherited as an autosomal dominant condition. Those with Down's syndrome also appear to be predisposed to the illness (Kachaturian, 1985). Interestingly, a recent study by Weinreb (1985) has shown that the fingerprints of patients suffering from AD have similar dermatoglyphic patterns to those in Down's syndrome. He states that the implication of this finding is unclear, but that further dermatoglyphic and genetically based studies in AD are clearly indicated.

AD usually is insidious in onset and most often begins after the age of 50. The disease advances through three stages in a relatively orderly and consistent way. In the first stage,

memory, cognition and visuo-spatial skills are compromised, but speech articulation and other motor functions remain normal. This means that the patient may have difficulty generating word lists and there is a paucity of ideas in speech. EEG and CT scans are unremarkable (Cummings 1985) and diagnosis of the condition at this stage is extremely difficult.

The second stage sees a continued deterioration of intellectual functions. Comprehension and memory for recent and remote events are impaired. Cognitive skills, including calculation and abstraction, are severly impaired. Visuo-spatial and cognitive deterioration produce disorientation and the patient may lose his way. The EEG shows an increase in the amount of theta activity and the CT scan may begin to show signs of diffuse cortical atrophy (Cummings, 1985).

In the final stage, all intellectual functions are severely impaired. Verbal output disappears or is reduced to echolalia or palilalia. Sphincter control is lost and the patient's limbs assume a rigid, flexed position. The EEG contains excessive delta activity, and the CT scan shows diffuse cerebral atrophy and ventricular dilatation (Cummings, 1985).

The diagnosis of the disease during the first stage is obviously difficult. Most epidemiological studies, therefore, only estimate the prevalence of the more severe stages. DSM III (1980) quotes the prevalence as being between 2% and 4% of the population and increasing with age, particularly after 75 years. Schoenberg et al. (1985) found 1% of the population over

40 years of age of Copia County in the USA to be suffering from severe dementia. This figure increased to 2,2% for the population older than 65 years and to 7% for 80 years and older. The authors found a higher incidence in blacks and females. This may be because they measured prevalence, which is a function of both incidence and survival. The increased prevalence amongst females may be attributed to their increased survival rate. Schoenberg et al. (1985), however, say this reasoning cannot explain the race differences as they doubt that blacks with severe dementia have a better survival rate than whites of the same age and sex.

Treves et al. (1986) carried out a nation-wide epidemiological study of presenile dementia of the Alzheimer type in Israel. They limited their study to individuals aged between 40 and 60 years and found that for every 100 000 of the population 2,4 new cases arose each year. Unfortunately prevalence figures as a percentage of the population are not given. An interesting finding was that the incidence of the disease in European-American-born Jews living in Israel was higher than that of African-Asian-born Jews. Although the incidence of the disease for females in this study was higher than for the males this difference was not statistically significant.

The other main cause of dementia in the aged is MID . This is sometimes known as arteriosclerotic dementia and is the result of cerebrovascular disease. MID can usually be distinguished from AD by its sudden onset and step-wise progression as the compounding effect of recurrent strokes impairs the intellect.

The cture may be a composite of the two (Adams and Victor, 1977). The prevalence of MID is not given in the DSM III manual but it is stated that it is apparently much less common than AD (DSM III, 1980).

In recent years there has been an increased interest in the effects of ageing and particularly in senile dementia. If AD represents an exaggerated form of ageing then it was hoped that by studying this disease new insights into normal ageing would be gained. Berg (1985) reviewed this research and concluded that the evidence is conflicting and that the question of whether AD represents exaggerated ageing cannot be answered until much more is known about the causes and mechanisms of both normal ageing and AD.

A few years ago, our laboratory was approached by a drug company to record clinical EEGs in demented elderly subjects entering a drug trial. Permission was obtained to record additional electrophysiological data for our own purposes. The results obtained are described in this report. Clinical EEGs, Fourier Transforms of the EEG (FFT) and Pattern Reversal Visual Evoked Potentials (PRVEPs) were recorded.

METHOD

Subjects

Forty two residents (33 females, 9 males) of a local geriatric home were subjects. They were part of a larger group which had

been enrolled for the drug study. Their ages ranged from 65 to 91 years (mean 77,0 SD 7,1). All had been diagnosed as mildly to moderately demented, either AD or MID, or a combination of the two. The diagnosis was made on the basis of "their medical history and examination, neurological examination, EEG recording and Computed Tomography (CT)" (Levinson et al., 1985).

Subjects were selected for the present study if they had not been excluded from the drug study for any reason and if all three tests (EEG, FFT and PRVEP) had been performed.

In the present study the subjects were subdivided, on the basis of the computed tomography scan results, into a group with evidence of previous infarcts (MID group) and those with no evidence of infarcts. Details of these groups are given in Table 2.

Twenty-six percent of of all subjects showed evidence of previous infarcts. The groups were sub-divided into age categories according to decades, and the proportion of subjects with AD and MID in each decade was similar. For both disease groups, there were fewer subjects in the 60 to 69 age group and no significant differences between the number of subjects in the 70 to 79 and 80+ age groups (Table 3).

Although subjects in the MID group all showed signs of previous infarcts, there was no difference between this group and the AD group in terms of the degree of cerebral atrophy shown on the

Table 2

Age and sex distribution for Alzheimer's and Multi-infarct groups

	Alzheimer's		Multi-infarct		Total	
	M	SD	M	SD	M	SD
Age (yrs)	77,7	(7,35)	75,5	(6,71)	77,0	(7,1)
N	31		11		42	
Males/Females	8/23		1/10		9/33	

The difference in age between the Alzheimer's and Multi-infarct groups was not statistically significant (t=0,87 df=40).

Table 3

Age distribution of Alzheimer's and Multi-infarct subjects

	Age (yrs)			
	60-69	70-79	80+	Total
Alzheimer's	6(75%)	13(76,5%)	12(71%)	31(74%)
Multi-infarct	2(25%)	4(23,5%)	5(29%)	11(26%)
Total	8	17	17	42(100%)

CT scan, as rated by an experienced radiologist (Table 4). CT abnormality ratings showed that 90% of the multi-infarct group were categorized as abnormal or worse, whereas only 52% of the AD group fell into this category (Table 5).

Procedure

EEG recording and analysis

Routine clinical EEGs were recorded according to a standardised procedure. Twenty-two electrodes were positioned according to the 10-20 system (Jasper, 1958). Electrode impedances less than 5 Kohms were routinely obtained by cleaning the scalp with alcohol and the application of electrode jelly. The electroencephalograph was an OTE Galileo E18b with 16 channels. Each recording included comprehensive coverage of the scalp by means of 5 preselected electrode montages. Photic stimulation was employed as an activation procedure. Hyperventilation was not used because of the health hazard associated with its use in aged subjects. Each recording session, including activation, lasted approximately 20 to 25 minutes.

EEG analysis was carried out by an experienced electroencephalographer. The EEG activity was described and the record rated for overall abnormality on a four-point-scale. Focal abnormalities and mean alpha frequency were noted.

Fast Fourier Transforms

Between the second and third recording montage of the EEG,

Table 4

C T scan atrophy ratings for Alzheimer's and Multi-infarct groups

	Alzheimer's	Multi-infarct	Total
Mean Atrophy Rating	2,38	2,8	2,49
Standard Deviation	0,78	0,63	0,76
N	29	10	39

The difference in mean atrophy rating between groups was not statistically significant (t=1,53 df=37)

Table 5

Distribution of subjects according to C T scan atrophy rating

	Alzheimer's	Multi-infarct	Total group
Normal/Mildly Abnormal	3 (10%)	1 (10%)	4 (10%)
Moderately Abnormal	11 (38%)	0 (0%)	11 (28%)
Abnormal +	15 (52%)	9 (90%)	24 (62%)
Total	29 (100%)	10 (100%)	39 (100%)

simultaneous 2-channel FFTs of the EEG activity between electrodes P3 and O1 (left hemisphere) and P4 and O2 (right hemisphere) were made. FFTs were performed on-line by an OTE 1244 Berg Fourier Analyser. The frequency bands employed were: 0,5 to 1,5 Hz; 1,6 to 3,9 Hz; 4,0 to 5,9 Hz; 6,0 to 7,9 Hz; 8,0 to 12,9 Hz; 13,0 to 15,9 Hz and total energy for hemisphere. FFTs were carried out for 2 periods of 60 seconds (with the eyes closed and eyes open) and the values expressed as $\mu V^2/Hz$. An artefact reject facility and time constant of 0,3 were routinely used. Statistical calaculations were made on the energy values obtained after correction against an FFT of a 50μV square wave calibration signal using the same parameters (Sciarretta and Erculani, 1978). Energy values for each band were obtained and expressed as percentages of the total energy.

Pattern Reversal Visual Evoked Potentials

Immediately after the FFT, PRVEPs were recorded from silver-chlorided silver disc electrodes placed at Fpz and Oz (Jasper 1958) and on the mastoid bones bilaterally. Electrode impedances of below 2 Kohms were routinely obtained by cleaning the electrode sites with alcohol. Electrodes were attached and impedances reduced by means of Beckman adhesive paste.

PRVEPs were recorded from Oz referred to the commoned mastoids so that a positivity at Oz produced an upward deflection of the graph. Electrode Fpz served as ground. EEG activity was

amplified by means of an OTE 2305 EEG preamplifier and averaged on an OTE 1239 Neuroaverager. An artefact reject facility rejected trials with excessive EMG or EOG artefact. Hard copy of the PRVEP was obtained by means of a Moseley X-Y plotter.

Pattern reversal chessboard stimulation generated on a Nicolet Nic-1005 Visual Stimulator was used. The subject was seated one metre from the TV screen which subtended a visual angle of 14 degrees. Individual checks subtended a visual angle of 55' and reversed at a rate of 0,94 reversals per second. Room illumination and screen luminance were kept constant during testing. Trials employing binocular and left and right monocular input were recorded and replicated.

The major positive peak of the PRVEP (P100) was identified by extrapolating the positive- and negative-going slopes of the most prominant peak in the region of 100msec and drawing in a perpendicular at the point of intersection. P100 was taken at the point where this line crossed the evoked potential. Latency and amplitude (peak to trough) measurements were made. P100 latency was compared with values obtained from an asymptomatic, young adult, control group recorded on the same system (Murdoch 1981). Values in excess of the norm group mean plus 3 standard deviations were regarded as abnormal.

RESULTS

1) EEG

The EEG results are summarised in Tables 6 to 8. Thirteen

subjects (31%) had normal or mildly abnormal EEGs, 18 (43%) had moderately abnormal EEGs, and 11 (26%) had abnormal or severely abnormal EEGs (Table 6). There were differences between the AD and MID groups. Fifty-two percent of the AD group's EEGs were moderately abnormal, whereas only 18% of the MID group fell into this category. However, 55% of the MID group were abnormal or worse with only 16% of the AD group in this category (Table 6). The small number of subjects in each group precluded calculation of the statistical significance of these findings.

Samples of EEG recordings, all from subjects in the AD group, are seen in Figures 1 to 3. Figure 1, recorded from a 70-year-old female, shows a normal symmetrical alpha rhythm at 9 Hz. No generalised or focal features are seen. Only 4 (9,5%) of the 42 subjects had EEGs of this nature, equally divided between the AD and MID groups. The EEG sample in Figure 2, recorded from a 74-year-old female, shows generalised slowing of the dominant frequency to 7 Hz. No focal abnormalities were identified. Again, only 4 subjects showed this pattern, all in the AD group. Figure 3, a record from a 76-year-old male, also shows generalised slowing of the dominant frequency and in addition 2 to 3 Hz activity in the left hemisphere. Fifty percent of the subjects had EEGs similar to this and they were equally divided between the two groups. The remaining subjects (31%) showed a dominant frequency in the alpha range but with focal slowing.

The most frequent EEG abnormality was generalised slowing of the dominant frequency. In addition to this, focal

Table 6

Distribution of subjects according to EEG abnormality rating

	Alzheimer's	Multi-infarct	Total group
Normal/Mildly Abnormal	10 (32%)	3 (27%)	13 (31%)
Moderately Abnormal	16 (52%)	2 (18%)	18 (43%)
Abnormal +	5 (16%)	6 (55%)	11 (26%)
Total	31 (100%)	11(100%)	42 (100%)

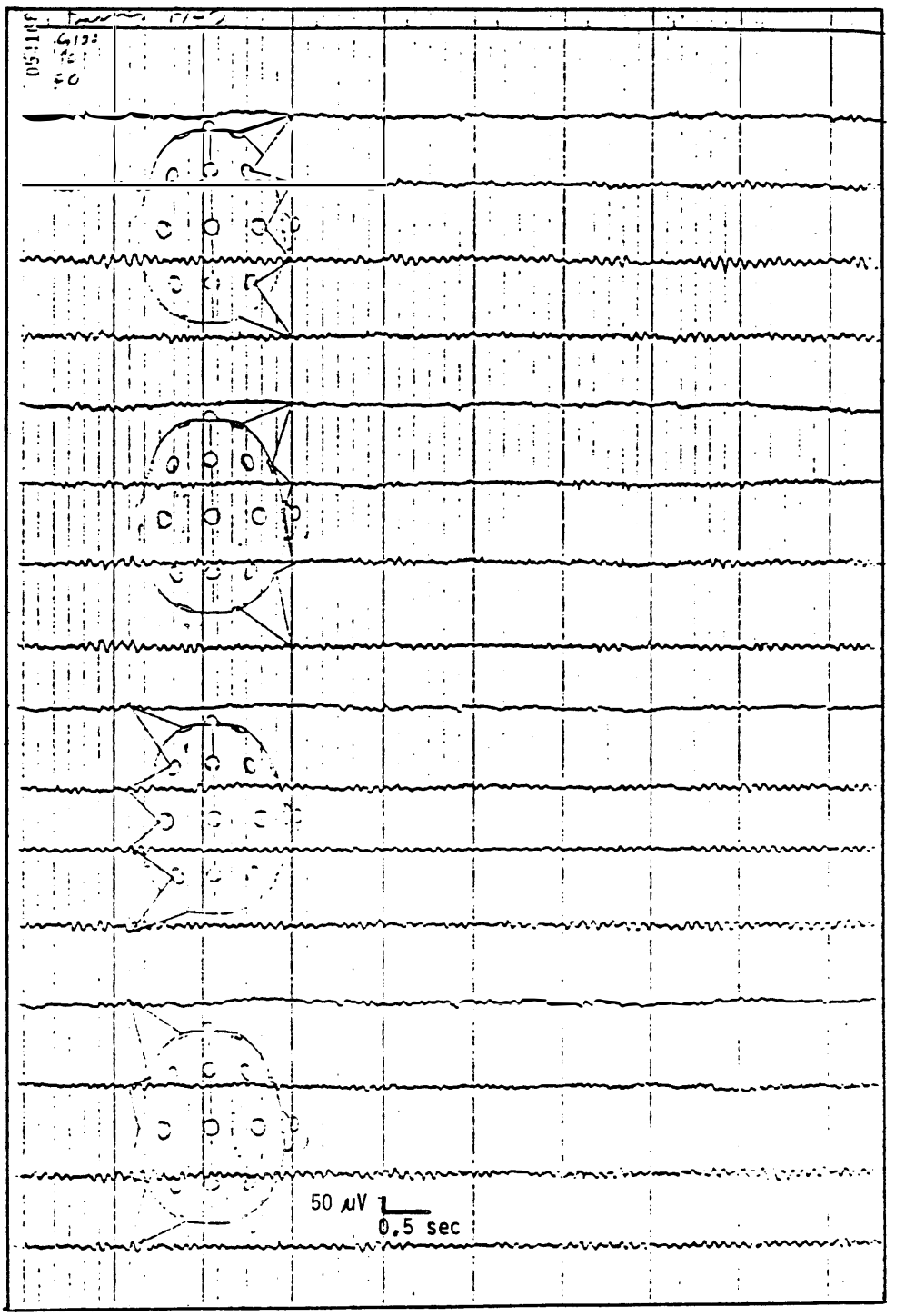
Table 7

Lateralization of EEG abnormalities in Alzheimer's and Multi-infarct groups

EEG Abnormality Lateralization	Alzheimer's	Multi-infarct	Total group
Left Hemisphere	14 (45%)	7 (64%)	21 (50%)
Right Hemisphere	6 (19%)	2 (18%)	8 (19%)
Bilateral	5 (16%)	0	5 (12%)
No lateralization	4 (13%)	0	4 (9,5%)
Normal	2 (7%)	2 (18%)	4 (9,5%)

Figure 1

EEG showing normal activity from a 70-year-old female with Alzheimer's disease



abnormalities were present in 69% of subjects. Fifty percent of the subjects showed focal abnormalities confined to the left hemisphere, but only 19% showed focal abnormalities unilaterally on the right. There was no difference in the occurence of unilateral left-hemisphere dysfunctions in the AD and MID groups. However, all the MID subjects had a lateralised dysfunction (in addition to generalised slowing), but only 16,1% of the AD group had bilateral localisation, and 12,9% showed no localisation (Table 7).

Table 8 and Figure 4 show the distribution of subjects according to EEG frequency bands between 7 and 11 Hz. The modal frequency was 8 Hz. Twenty one percent of the subjects had a dominant frequency in the 7 to 8 Hz range, 52,4% had an alpha frequency of 8 Hz, and only 9,5% of the subjects had an alpha frequency of 10 Hz and higher.

The mean values of the dominant EEG frequency for each group at each decade are seen in Table 9. The value for the total group (N=42) was 8,7 Hz. Although the groups were too small to test statistical significance, there appeared to be very little difference between the AD and MID groups with regard to alpha frequency at any age, although alpha frequency showed a tendency to decrease with each decade for the total group and the AD group (Figure 5). This trend was however not seen in the MID group. The group size may have been too small for this trend to emerge. Frequency measures based on normal clinical analysis of the EEG ("eye-balling") are also not very precise.

Figure 2

EEG showing generalised slowing of the dominant frequency to 7 Hz from a 74-year-old female with Alzheimer's disease

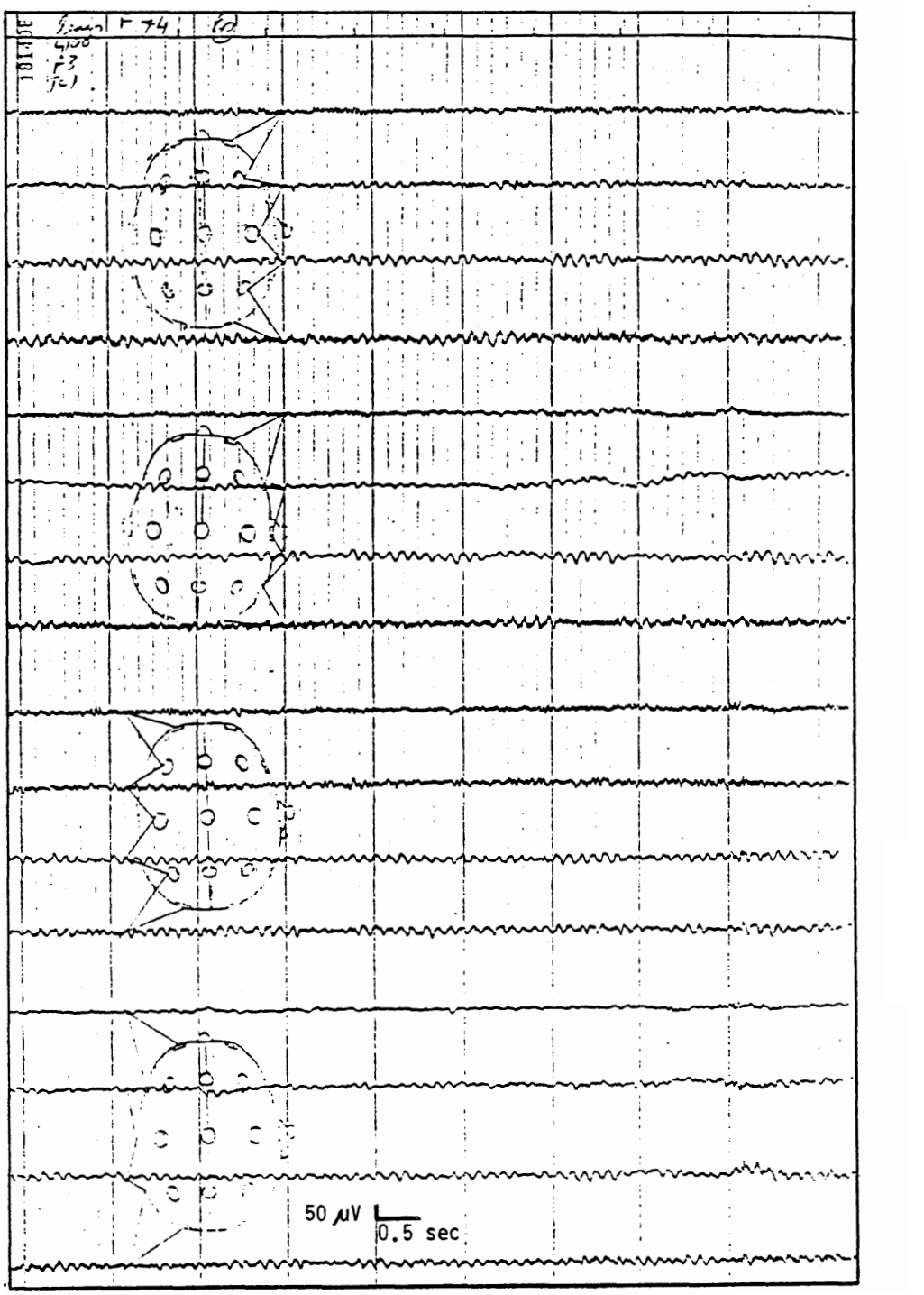
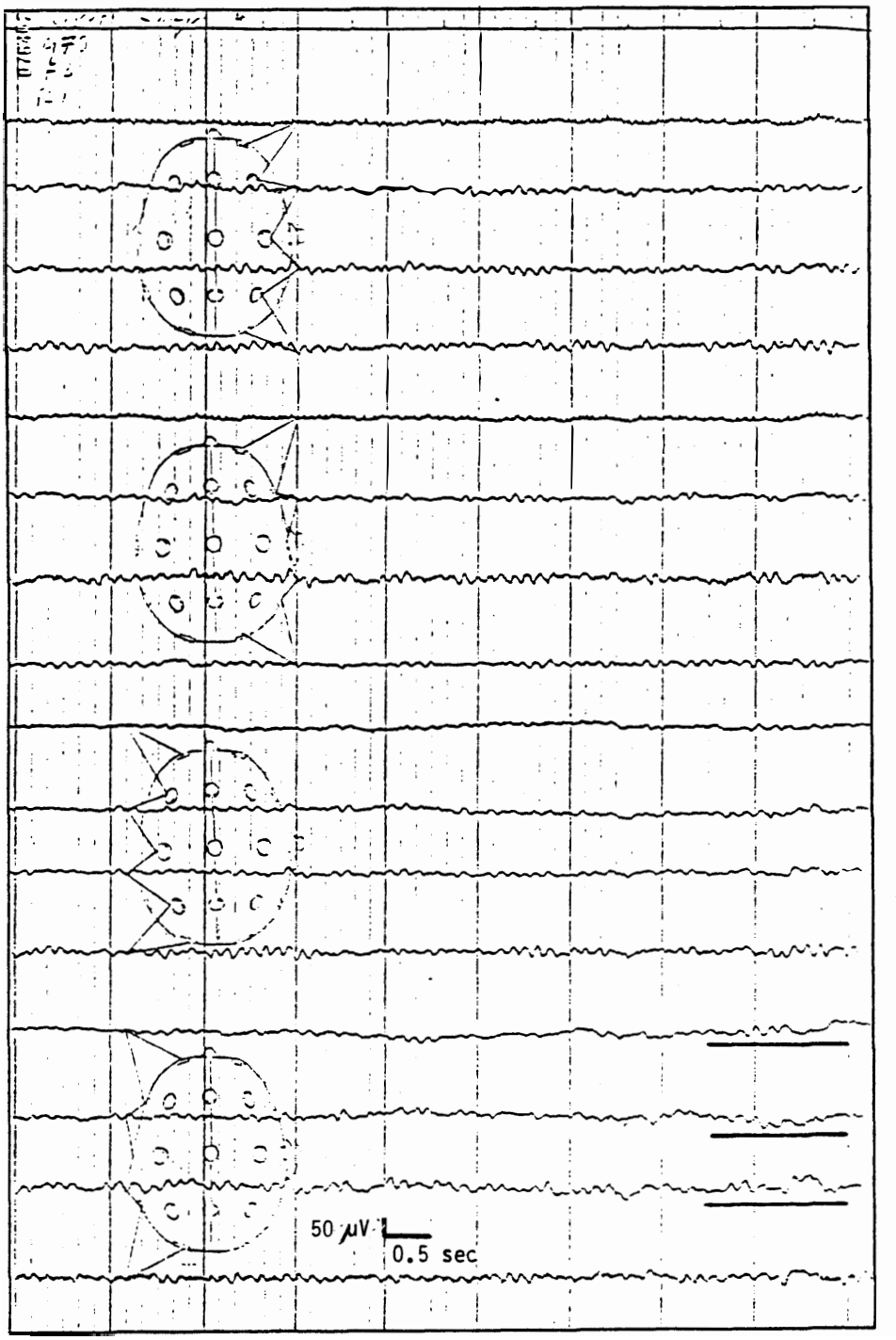


Figure 3

EEG showing generalized slowing of the dominant frequency and 2 to 3 Hz activity in the left temporal area from a 76-year-old male with Alzheimer's disease



2)FFT

FFT results are given in Tables 10 to 26 and Figures 6 to 14. Figures 6 to 10 show that the alpha band contained most of the power in the spectral array. Alpha power varied between 23,9 and 30% of total power depending on the group and the recording condition. The low delta band contributed 17,5 to 25,4% of the total power, and the high delta, the high theta and the beta bands each contributed between 13,2 and 17,3% to the total power. The percentage power in the delta, theta and alpha bands for our subjects is compared with results from previous studies in Figure 6. Our results were similar to these previous studies in the delta and theta bands, but our subjects appeared to have less alpha activity.

There were no significant differences between the AD and MID groups with respect to any of the frequency bands analysed by the FFT. This was true for both the absolute values (Table 10, Figure 7), and for each frequency band expressed as a proportion of the total power (Table 11, Figure 8) both for eyes open and eyes closed for both hemispheres.

Hemispheric differences were examined separately for the two groups. Neither group showed significant differences between the hemispheres, either for the eyes open or the eyes closed recording conditions, for any of the frequency bands, either in terms of absolute (Tables 12 and 13) or in terms of proportional (Tables 14 and 15) values.

Table 8

Distribution of subjects (both groups) according to dominant EEG frequency

Frequency (Hz)	N	%
7-8	9	21,4
8	22	52,4
9	7	16,7
10	1	2,4
11	3	7,1
Total	42	100,0

Table 9

Group dominant EEG frequency according to age

	Alzheimer's			Multi-infarct			Total group		
Age (yrs)	N	M	SD	N	M	SD	N	M	SD
60 to 69	(6)	9,2	1,3	(2)	8,6	1,1	(8)	9,1	1,2
70 to 79	(14)	8,5	1,1	(4)	8,9	1,9	(18)	8,6	1,3
above 80	(11)	8,4	0,6	(5)	8,7	0,5	(16)	8,5	0,6
Total group	(31)	8,6	1,0	(11)	8,8	1,2	(42)	8,7	1,0

Figure 4

Distribution of subjects in Alzheimer's disease and Multi-infarct groups according to frequency band (7 to 11 Hz)

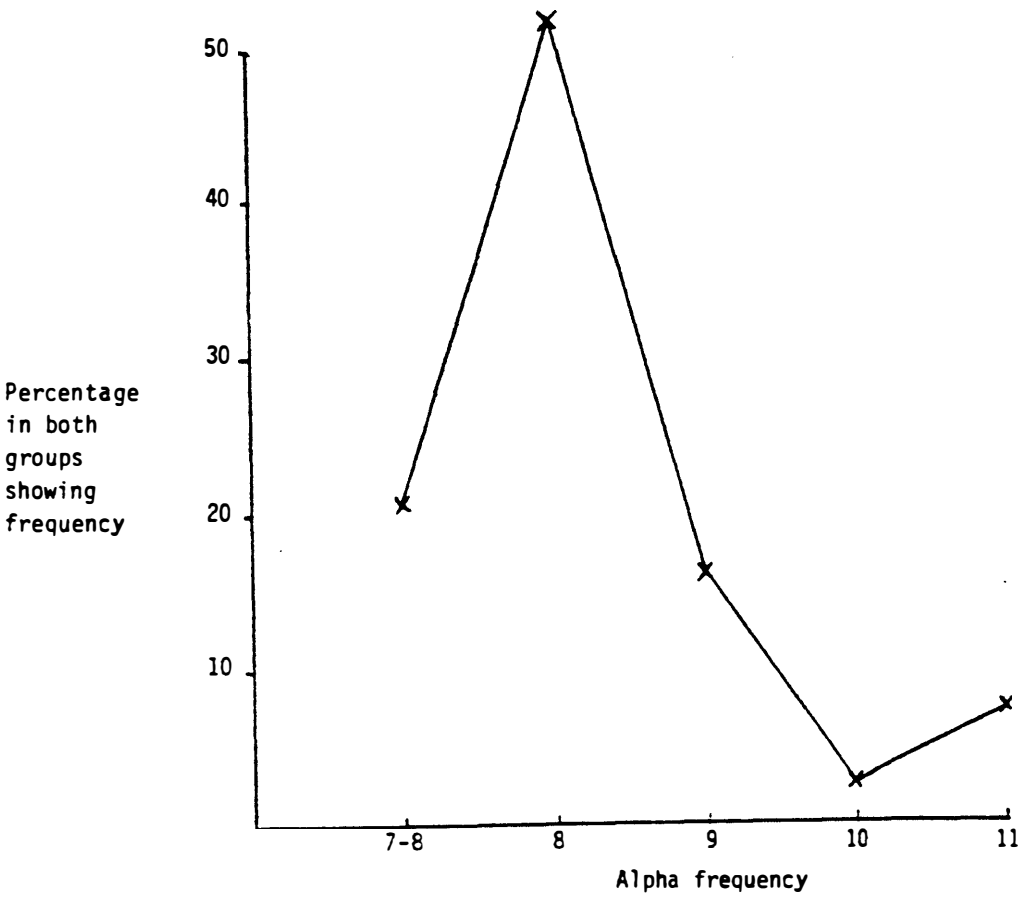


Figure 5

Mean alpha frequency for each group according to age

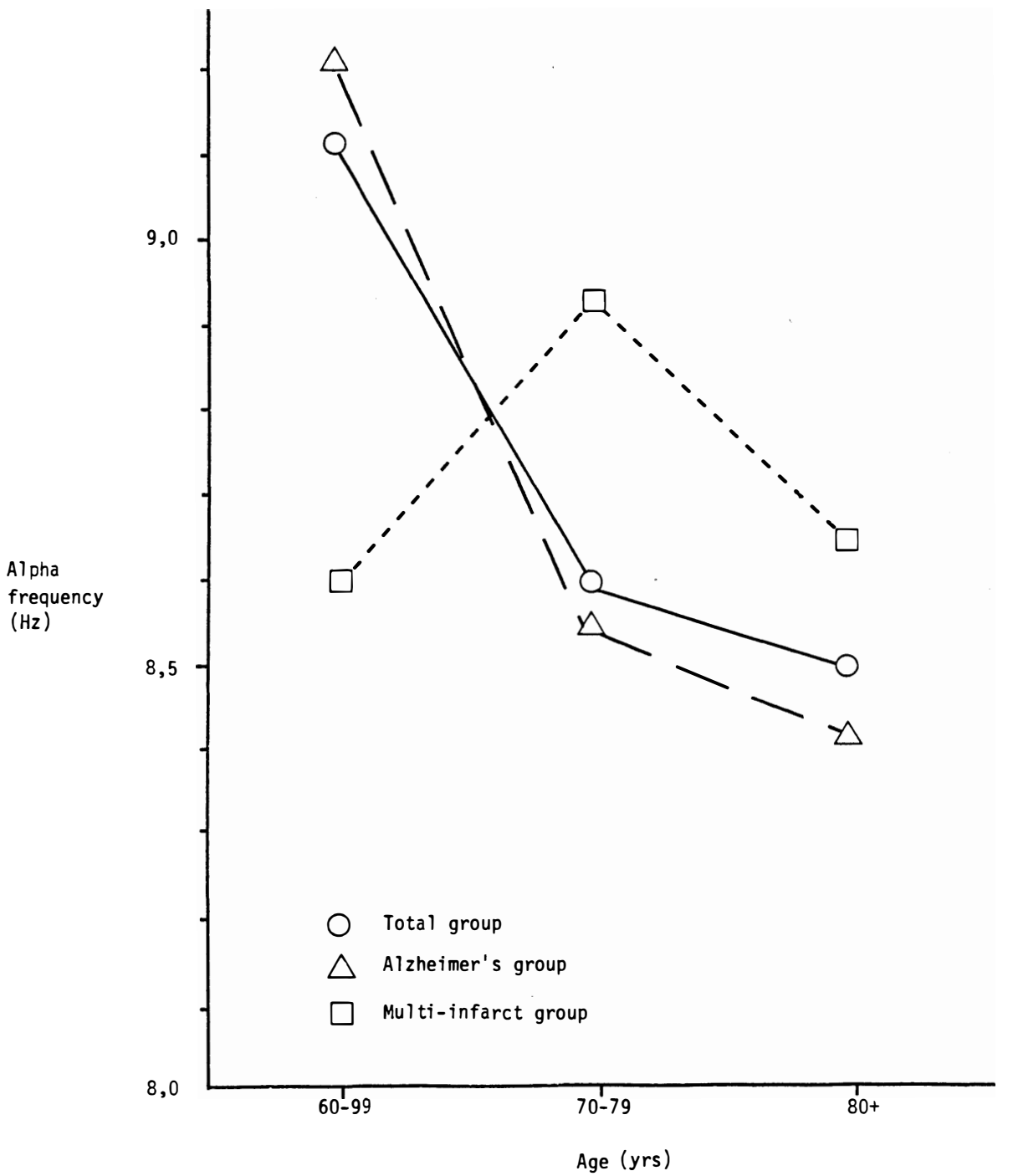


Table 16 and Figure 9 compare the spectral energy seen with eyes open and eyes closed for the AD group. Significant increases in absolute power were seen on eye closure in total power, as well as in the high theta and alpha bands for the left hemisphere, and in the high theta and alpha bands for the right hemisphere. In terms of percentage power, both low and high delta activity decreased significantly while alpha increased significantly in both hemispheres. The increase in percentage alpha was greater in the right hemisphere than the left (Table 17, Figure 10).

The MID group failed to show any FFT differences with eyes open and closed for any frequency band in either hemisphere, for both the absolute value (Table 18, Figure 9) and in terms of percentage power (Table 19, Figure 10).

Alpha reactivity to eye closure may be expressed as a percentage of the power in the alpha band for eyes open in relation to the power for eyes closed. The lower the percentage the more reactive the alpha. Table 20 shows alpha ratios for the two groups for left and right hemispheres. The MID group showed very little reactivity, and there was no hemispheric difference. The AD group again showed the greater reactivity, especially in the right hemisphere, although the difference between hemispheres was not statisticaly significant.

The relationship between the FFT measures and age was also investigated. The combined AD and MID groups were divided according to decades (60 to 69 years, 70 to 79 years and 80

Figure 6

Proportional power for the present group compared to results in the literature

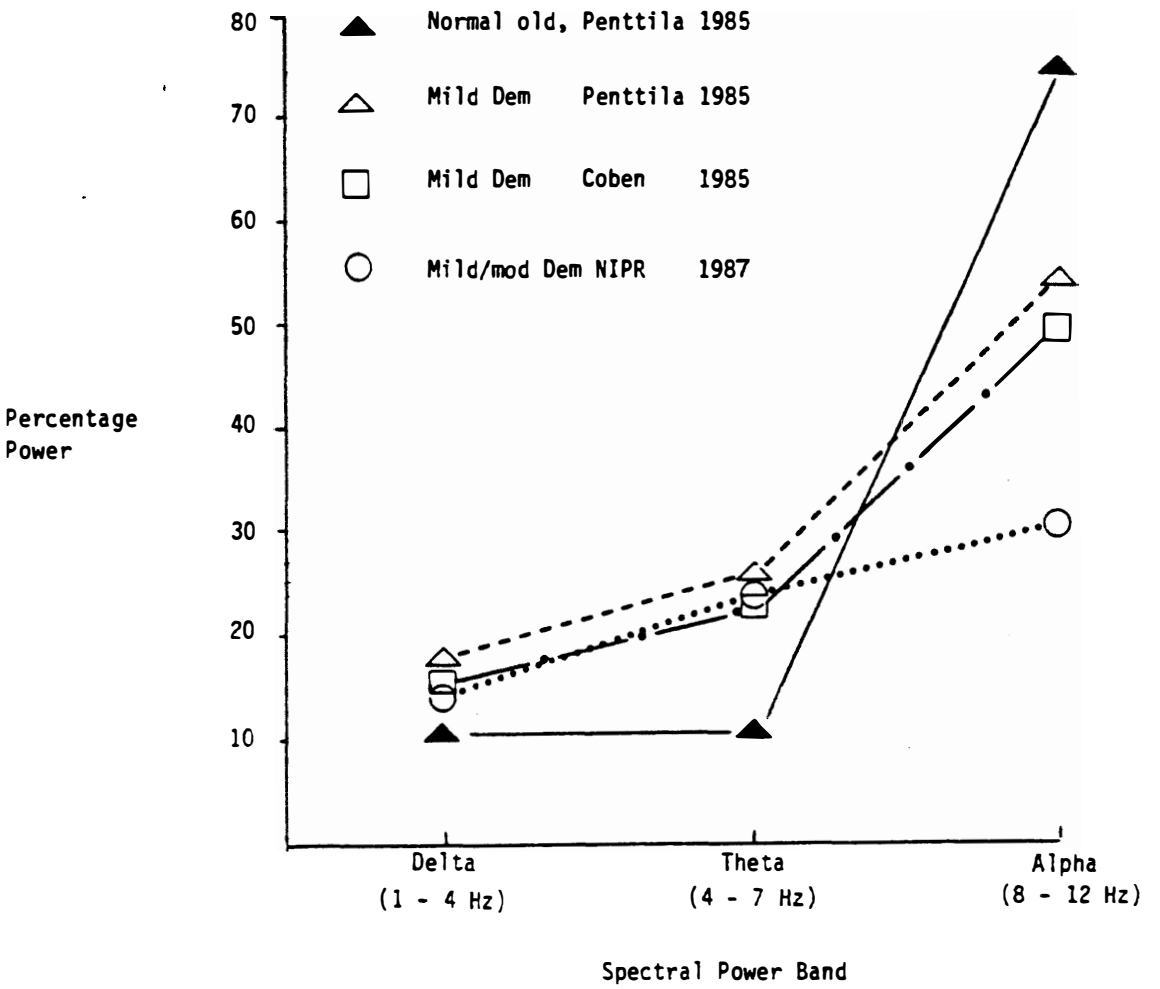


Table 10

Spectral Analysis: Alzheimer's and Multi-infarct groups (absolute scores)

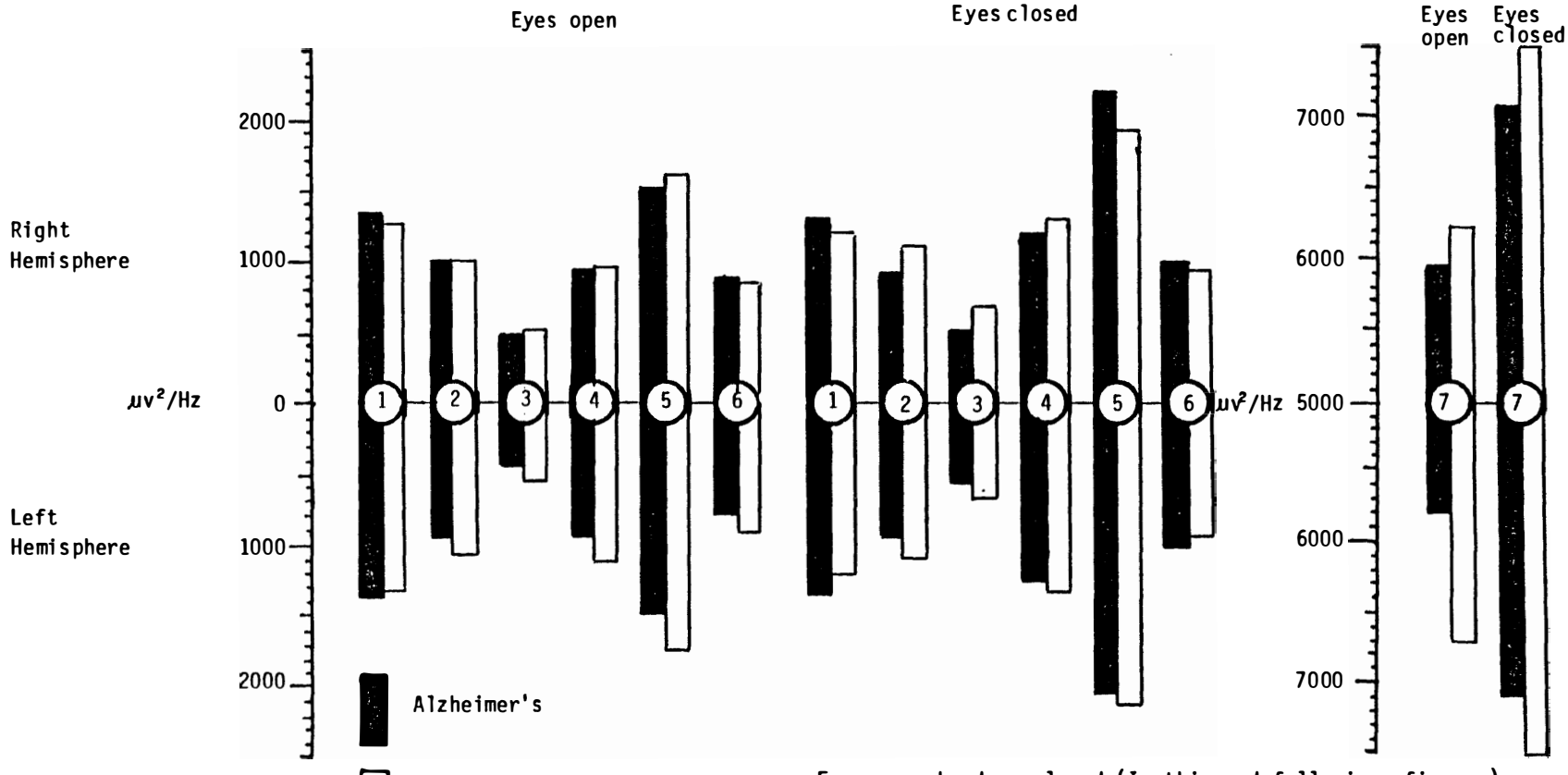
Eyes Open/ Closed	Hemisphere Right/ Left	Spectral frequency band (Hz)	Alzheimer's/ Multi-infarct	N	Mean Power (uV/Hz)	Standard deviation	t	p
O	R	Total	AD	30	5957	2844		
			MID	11	6205	2915	-0,25	
O	R	0,5-1,5	AD	31	1364	441		
			MID	11	1287	440	0,5	
O	R	1,6-3,9	AD	31	986	548		
			MID	11	985	675	0,01	
O	R	4,0-5,9	AD	31	519	385		
			MID	11	539	378	-0,15	
O	R	6,0-7,9	AD	31	940	634		
			MID	11	954	741	-0,06	
O	R	8,0-12,9	AD	31	1533	977		
			MID	11	1591	914	-0,17	
O	R	13,0-15,9	AD	31	886	584		
			MID	11	844	451	0,22	
O	L	Total	AD	30	5802	2529		
			MID	11	6738	3174	-0,98	
O	L	0,5-1,5	AD	31	1387	448		
			MID	11	1360	513	0,17	
O	L	1,6-3,9	AD	31	945	535		
			MID	11	1068	601	-0,64	
O	L	4,0-5,9	AD	31	489	357		
			MID	11	570	337	0,66	
O	L	6,0-7,9	AD	31	961	649		
			MID	11	1106	763	-0,61	
O	L	8,0-12,9	AD	31	1494	926		
			MID	11	1733	989	-0,72	
O	L	13,0-15,9	AD	31	829	595		
			MID	11	924	454	-0,48	
C	R	Total	AD	31	7071	3231		
			MID	10	7481	3110	-0,35	
C	R	0,5-1,5	AD	31	1308	472		
			MID	10	1219	499	0,51	
C	R	1,6-3,9	AD	31	942	545		
			MID	10	1108	751	-0,76	
C	R	4,0-5,9	AD	31	537	362		
			MID	10	695	486	-1,1	
C	R	6,0-7,9	AD	31	1229	710		
			MID	11	1296	793	-0,26	
C	R	8,0-12,9	AD	31	2177	1250		
			MID	11	1956	1030	0,53	
C	R	13,0-15,9	AD	31	1034	767		
			MID	11	957	576	0,3	

Table 10 continued:

Eyes Open/ Closed	Hemisphere Right/ Left	Spectral frequency band (Hz)	Alzheimer's/ Multi-infarct	N	Mean Power (uV/Hz)	Standard deviation	t	p
C	L	Total	AD	31	7120	3345		
			MID	10	7588	3115	-0,4	
C	L	0,5-1,5	AD	31	1354	532		
			MID	10	1180	514	0,91	
C	L	1,6-3,9	AD	31	959	613		
			MID	10	1085	701	-0,55	
C	L	4,0-5,9	AD	31	550	389		
			MID	10	658	455	-0,73	
C	L	6,0-7,9	AD	31	1269	766		
			MID	11	1339	733	-0,26	
C	L	8,0-12,9	AD	31	2070	1213		
			MID	11	2112	1014	-0,1	
C	L	13,0-15,9	AD	31	1024	791		
			MID	11	962	576	0,24	

Figure 7

Spectral Analysis: Alzheimer's and Multi-infarct groups
(Absolute scores)



No significant differences between groups

Frequency bands analysed (In this and following figures)

1 = 0,5 - 1,5 Hz
2 = 1,6 - 3,9 Hz
3 = 4,0 - 5,9 Hz
4 = 6,0 - 7,9 Hz
5 = 8,0 - 12,9 Hz
6 = 13,0 - 15,9 Hz
7 = Total energy in hemisphere

Table 11

Spectral Analysis: Alzheimer's and Multi-infarct groups (proportional scores)

Eyes Open/ Closed	Hemisphere Right/ Left	Spectral frequency band (Hz)	Alzheimer's/ Multi-infarct	N	% of Total Power	Standard deviation	t	p
O	R	0,5-1,5	AD	30	24,9	9,4		
			MID	10	24,0	10,6	0,25	ns
O	R	1,6-3,9	AD	30	15,9	3,9		
			MID	10	14,9	5,8	0,62	ns
O	R	4,0-5,9	AD	30	7,6	2,6		
			MID	10	7,8	2,8	-0,21	ns
O	R	6,0-7,9	AD	30	14,8	8,4		
			MID	10	13,5	6,5	0,45	ns
O	R	8,0-12,9	AD	30	24,1	7,9		
			MID	10	25,8	8,9	-0,57	ns
O	R	13,0-15,9	AD	30	14,1	6,2		
			MID	10	14,7	7,6	-0,25	ns
O	L	0,5-1,5	AD	30	25,4	8,2		
			MID	10	22,9	8,8	0,82	ns
O	L	1,6-3,9	AD	30	15,3	4,1		
			MID	10	14,6	2,6	0,51	ns
O	L	4,0-5,9	AD	30	7,4	2,3		
			MID	10	7,6	2,1	-0,24	ns
O	L	6,0-7,9	AD	30	15,4	7,4		
			MID	10	14,7	6,6	0,27	ns
O	L	8,0-12,9	AD	30	23,9	8,0		
			MID	10	26,2	5,3	-0,85	ns
O	L	13,0-15,9	AD	30	13,1	6,3		
			MID	10	15,4	6,5	-0,99	ns
C	R	0,5-1,5	AD	30	20,6	7,7		
			MID	10	18,8	9,7	0,6	ns
C	R	1,6-3,9	AD	30	13,5	4,7		
			MID	10	13,8	5,8	-0,17	ns
C	R	4,0-5,9	AD	30	7,1	2,9		
			MID	10	8,3	3,5	-1,08	ns
C	R	6,0-7,9	AD	30	16,7	7,6		
			MID	10	16,8	6,1	-0,04	ns
C	R	8,0-12,9	AD	30	29,8	10,0		
			MID	10	27,9	9,9	0,52	ns
C	R	13,0-15,9	AD	30	13,6	6,5		
			MID	10	14,3	8,2	-0,28	ns

Table 11 continued:

Eyes Open/ Closed	Hemisphere Right/ Left	Spectral frequency band (Hz)	Alzheimer's/ Multi-infarct	N	% of Total Power	Standard deviation	t	p
C	L	0,5-1,5	AD	30	21,0	7,2		
			MID	10	17,5	7,8	1,31	
C	L	1,6-3,9	AD	30	13,3	4,9		
			MID	10	13,3	4,8	0,0	
C	L	4,0-5,9	AD	30	7,1	2,9		
			MID	10	7,8	3,4	-0,64	
C	L	6,0-7,9	AD	30	17,3	7,4		
			MID	10	16,8	5,6	0,2	
C	L	8,0-12,9	AD	30	28,5	9,7		
			MID	10	30,0	9,3	-0,43	
C	L	13,0-15,9	AD	30	13,2	6,4		
			MID	10	15,1	7,3	-0,79	

Figure 8

Spectral Analysis: Alzheimer's and Multi-infarct groups
(Proportional scores)

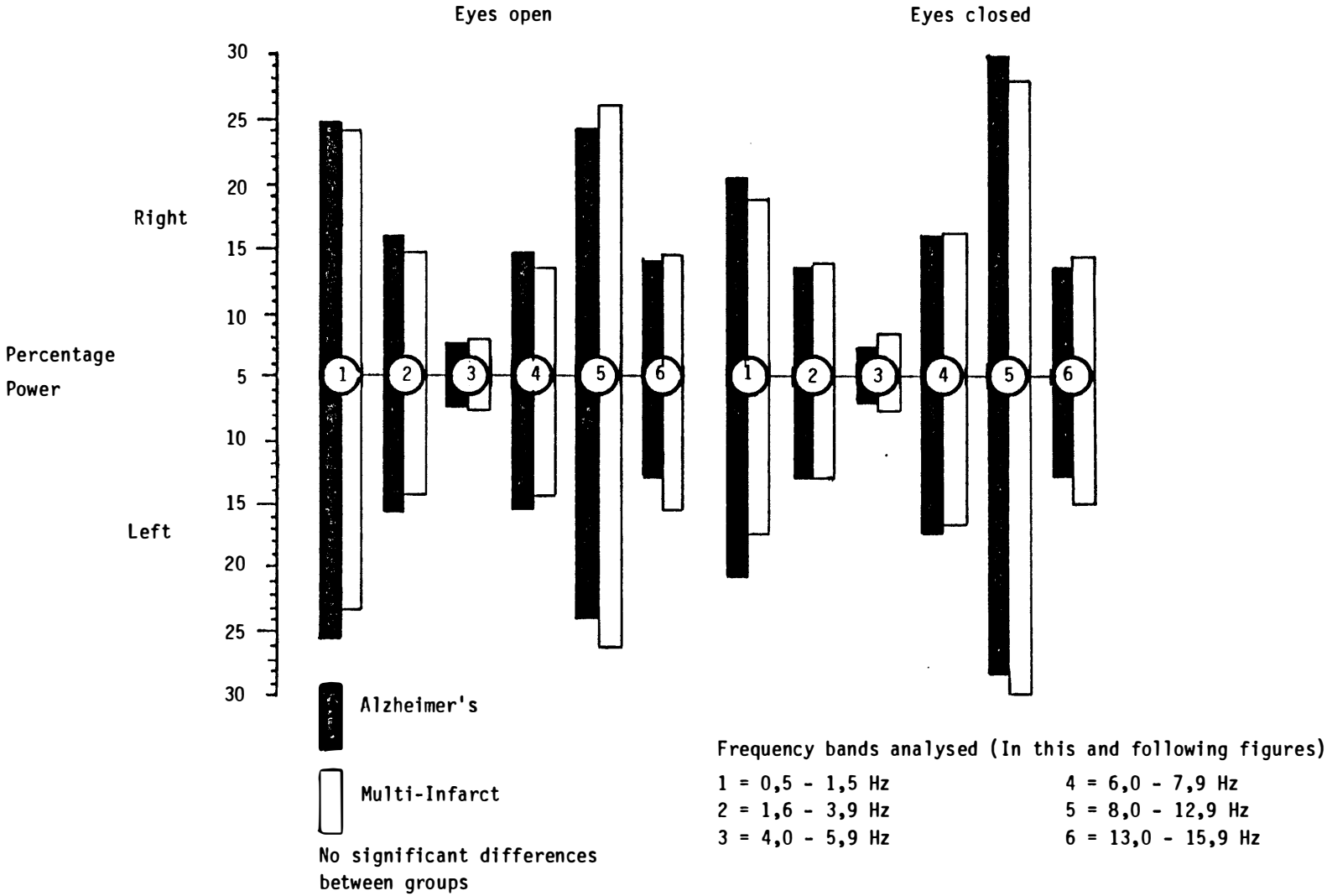


Table 12

Spectral Analysis: Left/Right hemisphere comparisons for Alzheimer's group (absolute scores)

Eyes Open/ Closed	Spectral frequency band (Hz)	Hemisphere Left / Right	N	Mean Power (μV^2/Hz)	Standard deviation	t	p
O	Total	L	30	5802	2529		
		R	30	5957	2844	-0,22	ns
O	0,5-1,5	L	31	1387	448		
		R	31	1364	441	0,2	ns
O	1,6-3,9	L	31	945	535		
		R	31	986	548	-0,3	ns
O	4,0-5,9	L	31	489	357		
		R	31	519	385	-0,32	ns
O	6,0-7,9	L	31	961	649		
		R	31	940	634	0,13	ns
O	8,0-12,9	L	31	1494	926		
		R	31	1533	977	-0,16	ns
O	13,0-15,9	L	31	829	595		
		R	31	886	584	-0,38	ns
C	Total	L	31	7120	3345		
		R	31	7071	3231	0,06	ns
C	0,5-1,5	L	31	1354	532		
		R	31	1308	472	0,36	ns
C	1,6-3,9	L	31	959	613		
		R	31	942	545	0,12	ns
C	4,0-5,9	L	31	550	389		
		R	31	537	362	0,14	ns
C	6,0-7,9	L	31	1269	766		
		R	31	1229	710	0,21	ns
C	8,0-12,9	L	31	2070	1213		
		R	31	2177	1250	-0,34	ns
C	13,0-15,9	L	31	1024	791		
		R	31	1034	767	-0,05	ns

Table 13

Spectral Analysis: Left/Right hemisphere comparisons for Multi-infarct group (absolute scores)

Eyes Open/ Closed	Spectral frequency band (Hz)	Hemisphere Left / Right	N	Mean Power (uV/Hz)	Standard deviation	t	p
O	Total	L	11	6738	3174		
		R	11	6205	2915	0,41	ns
O	0,5-1,5	L	11	1360	513		
		R	11	1287	440	0,36	ns
O	1,6-3,9	L	11	1068	601		
		R	11	985	675	0,31	ns
O	4,0-5,9	L	11	570	337		
		R	11	539	378	0,2	ns
O	6,0-7,9	L	11	1106	763		
		R	11	954	741	0,47	ns
O	8,0-12,9	L	11	1733	989		
		R	11	1591	914	0,35	ns
O	13,0-15,9	L	11	924	454		
		R	11	844	451	0,41	ns
C	Total	L	10	7588	3115		
		R	10	7481	3110	0,08	ns
C	0,5-1,5	L	10	1180	514		
		R	10	1219	499	-0,17	ns
C	1,6-3,9	L	10	1085	701		
		R	10	1108	751	-0,07	ns
C	4,0-5,9	L	10	658	455		
		R	10	695	486	-0,18	ns
C	6,0-7,9	L	11	1339	733		
		R	11	1296	793	0,13	ns
C	8,0-12,9	L	11	2112	1014		
		R	11	1956	1030	0,36	ns
C	13,0-15,9	L	11	962	449		
		R	11	957	576	0,02	ns

Table 14

Spectral Analysis: Left/Right hemisphere comparisons for Alzheimer's group (proportional scores)

Eyes Open/ Closed	Spectral frequency band (Hz)	Hemisphere Left / Right	N	% of Total Power	Standard deviation	t	p
O	0,5-1,5	L	30	25,4	8,2		
		R	30	24,9	9,4	0,22	ns
O	1,6-3,9	L	30	15,3	4,1		
		R	30	15,9	3,9	-0,58	ns
O	4,0-5,9	L	30	7,4	2,3		
		R	30	7,6	2,6	-0,32	ns
O	6,0-7,9	L	30	15,4	7,4		
		R	30	14,8	8,4	0,29	ns
O	8,0-12,9	L	30	23,9	8,0		
		R	30	24,1	7,9	-0,1	ns
O	13,0-15,9	L	30	13,1	6,3		
		R	30	14,1	6,2	-0,62	ns
C	0,5-1,5	L	30	21,0	7,2		
		R	30	20,6	7,7	0,21	ns
C	1,6-3,9	L	30	13,3	4,9		
		R	30	13,5	4,7	-0,16	ns
C	4,0-5,9	L	30	7,1	2,9		
		R	30	7,1	2,9	0,0	ns
C	6,0-7,9	L	30	17,3	7,4		
		R	30	16,7	7,6	0,31	ns
C	8,0-12,9	L	30	28,5	9,7		
		R	30	29,8	10,0	-0,51	ns
C	13,0-15,9	L	30	13,2	6,4		
		R	30	13,6	6,5	-0,24	ns

Table 15

Spectral Analysis: Left/Right hemisphere comparisons for Multi-infarct group (proportional scores)

Eyes Open/ Closed	Spectral frequency band (Hz)	Hemisphere Left / Right	N	% Total Power	Standard deviation	t	p
O	0,5-1,5	L	10	22,9	8,8		
		R	10	24,0	10,6	-0,25	ns
O	1,6-3,9	L	10	14,6	2,6		
		R	10	14,9	5,8	-0,15	ns
O	4,0-5,9	L	10	7,6	2,1		
		R	10	7,8	2,8	-0,18	ns
O	6,0-7,9	L	10	14,7	6,6		
		R	10	13,5	6,5	0,41	ns
O	8,0-12,9	L	10	26,2	5,3		
		R	10	25,8	8,9	0,12	ns
O	13,0-15,9	L	10	15,4	6,5		
		R	10	14,7	7,6	0,22	ns
C	0,5-1,5	L	10	17,5	7,8		
		R	10	18,8	9,7	-0,33	ns
C	1,6-3,9	L	10	13,3	4,8		
		R	10	13,8	5,8	-0,21	ns
C	4,0-5,9	L	10	7,8	3,4		
		R	10	8,3	3,5	-0,32	ns
C	6,0-7,9	L	10	16,8	5,6		
		R	10	16,8	6,1	0,0	ns
C	8,0-12,9	L	10	30,0	9,3		
		R	10	27,9	9,9	0,49	ns
C	13,0-15,9	L	10	15,1	7,3		
		R	10	14,3	8,2	0,23	ns

Table 16

Spectral Analysis: Eyes Open/Closed comparisons for Alzheimer's group (absolute scores)

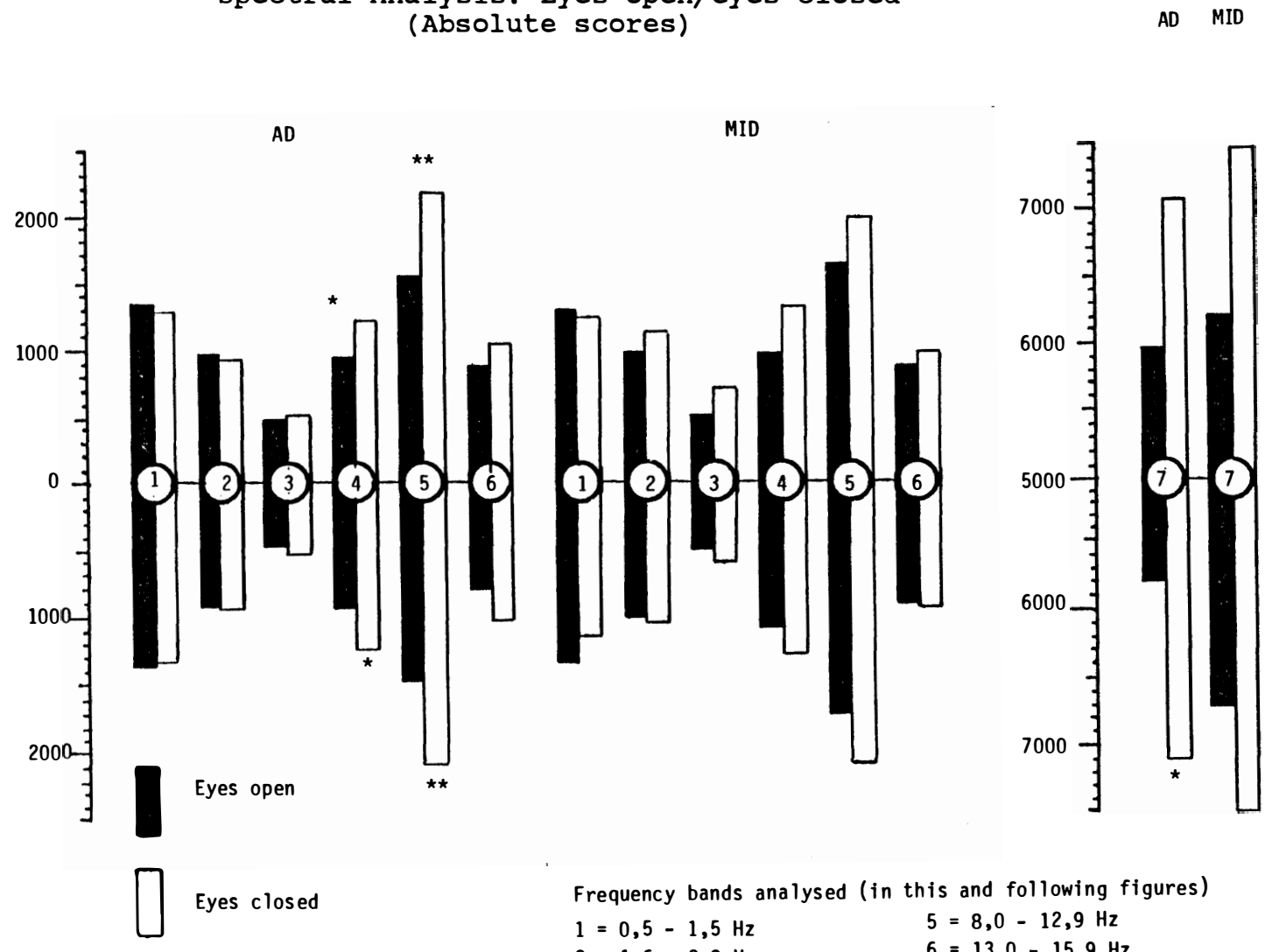
Hemisphere	Spectral frequency band (Hz)	Eyes Open/ Closed	N	Mean Power (uV/Hz)	Standard deviation	t	p
R	Total	O	30	5957	2844		
		C	31	7071	3231	-1,43	ns
R	0,5-1,5	O	31	1364	441		
		C	31	1308	472	0,48	ns
R	1,6-3,9	O	31	986	548		
		C	31	942	545	0,32	ns
R	4,0-5,9	O	31	519	385		
		C	31	537	362	-0,19	ns
R	6,0-7,9	O	31	940	634		
		C	31	1229	710	-1,69	*
R	8,0-12,9	O	31	1533	977		
		C	31	2177	1250	-2,26	**
R	13,0-15,9	O	31	886	584		
		C	31	1034	767	-0,85	ns
L	Total	O	30	5802	2529		
		C	31	7120	3345	-1,73	*
L	0,5-1,5	O	31	1387	448		
		C	31	1354	532	0,26	ns
L	1,6-3,9	O	31	945	535		
		C	31	959	613	-0,1	ns
L	4,0-5,9	O	31	489	357		
		C	31	550	389	-0,64	ns
L	6,0-7,9	O	31	961	649		
		C	31	1269	766	-1,71	*
L	8,0-12,9	O	31	1494	926		
		C	31	2070	1213	-2,1	**
L	13,0-15,9	O	31	829	595		
		C	31	1024	791	-1,1	ns

Significance levels (this and subsequent tables)

* = p < 0,05
** = p < 0,025
*** = p < 0,01

Figure 9

Spectral Analysis: Eyes open/eyes closed
(Absolute scores)



Significance Levels
* = p<0,05
** = p<0,025
*** = p<0.01

Frequency bands analysed (in this and following figures)
1 = 0,5 - 1,5 Hz
2 = 1,6 - 3,9 Hz
3 = 4,0 - 5,9 Hz
4 = 6,0 - 7,9 Hz
5 = 8,0 - 12,9 Hz
6 = 13,0 - 15,9 Hz
7 = Total energy in hemisphere

years and over). There was no difference between hemispheres, and therefore, for the purpose of this analysis, only the results from the left hemisphere for the eyes closed condition were used. Results appear in Tables 21 and 22 and in Figures 11 and 12. There were no significant differences in FFT power between the three age groups for any of the frequency bands recorded. The proportion of low delta activity however decreased with age, although this was only statistically significant when the oldest and youngest groups were compared. There was also an increase in the proportion of high theta activity with age. Once more, this was only significant when the oldest and youngest groups were compared

The relationship between age and reactivity to eye closure for the combined groups can be seen in Table 22. In this table the activity with eyes open is expressed as a percentage of the activity with eyes closed. Alpha activity showed the greatest reactivity, although reactivity in the high theta band was very similar. No significant difference was seen between the three age groups, although there was a trend towards lower reactivity with increased age.

The fact that there were so few significant differences between the age groups was surprising. It was therefore decided to examine the data for differences between groups formed on the basis of the clinical EEG abnormality rating. Subjects were divided into those with normal to mildly abnormal EEGs (group 1), those whose EEGs were rated as moderately abnormal (group2) and those with ratings of abnormal to grossly abnormal (group

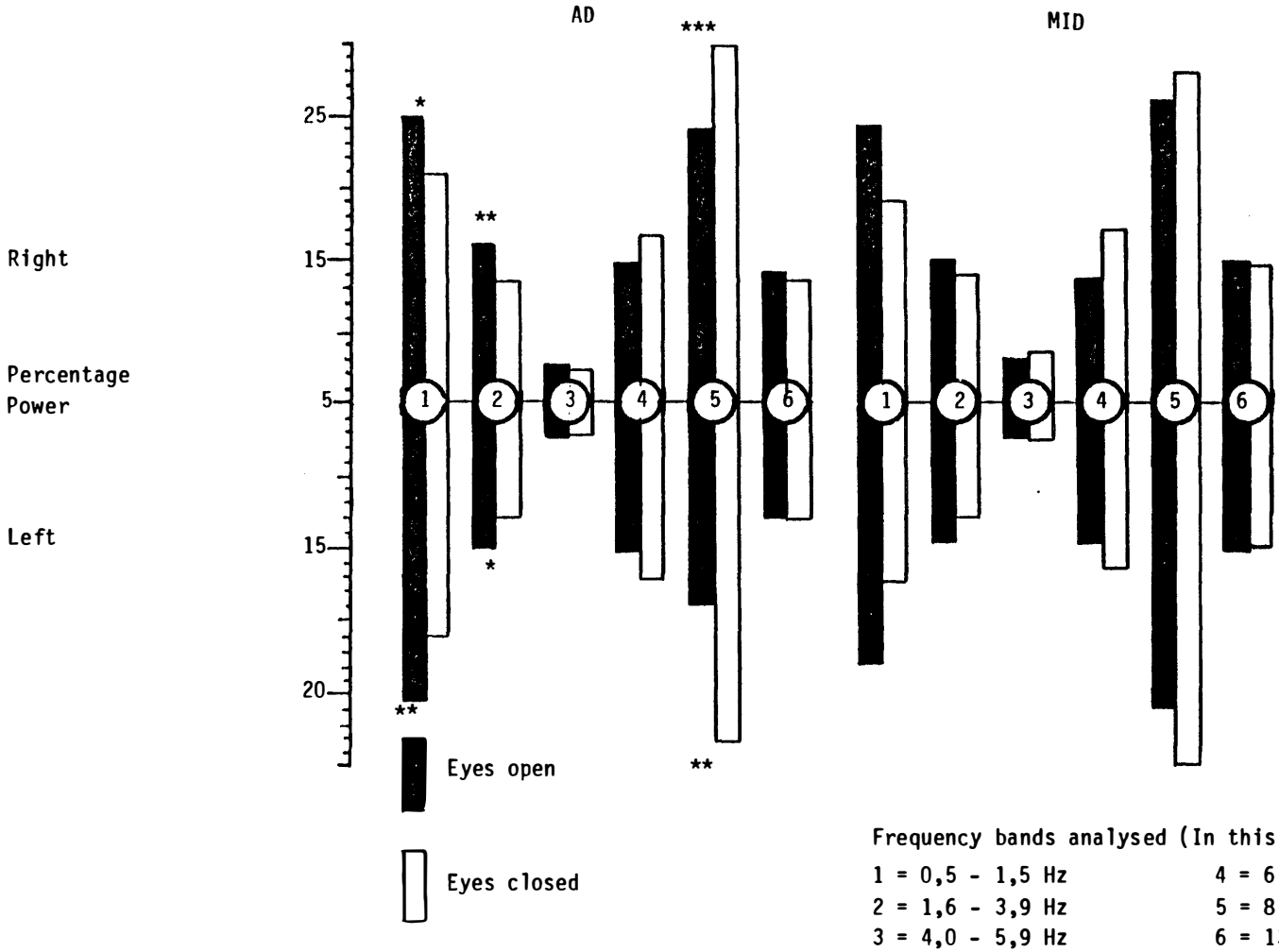
Table 17

Spectral Analysis: Eyes Open/Closed comparisons for Alzheimer's group (proportional scores)

Hemisphere	Spectral frequency band (Hz)	Eyes Open/ Closed	N	% of Total Power	Standard deviation	t	p
R	0,5-1,5	O	30	24,9	9,4		
		C	30	20,6	7,7	1,94	*
R	1,6-3,9	O	30	15,9	3,9		
		C	30	13,5	4,7	2,15	**
R	4,0-5,9	O	30	7,6	2,6		
		C	30	7,1	2,9	0,7	ns
R	6,0-7,9	O	30	14,8	8,4		
		C	30	16,7	7,6	-0,92	ns
R	8,0-12,9	O	30	24,1	7,9		
		C	30	29,8	10,0	-2,45	**
R	13,0-15,9	O	30	14,1	6,2		
		C	30	13,6	6,5	0,31	ns
L	0,5-1,5	O	30	25,4	8,2		
		C	30	21,0	7,2	2,21	**
L	1,6-3,9	O	30	15,3	4,1		
		C	30	13,3	4,9	1,72	*
L	4,0-5,9	O	30	7,4	2,3		
		C	30	7,1	2,9	0,44	ns
L	6,0-7,9	O	30	15,4	7,4		
		C	30	17,3	7,4	-0,99	ns
L	8,0-12,9	O	30	23,9	8,0		
		C	30	28,5	9,7	-2,00	**
L	13,0-15,9	O	30	13,1	6,3		
		C	30	13,2	6,4	-0,06	ns

Figure 10

Spectral Analysis: Eyes open/eyes closed
(Proportional scores)



Frequency bands analysed (In this and following figures)

1 = 0,5 - 1,5 Hz	4 = 6,0 - 7,9 Hz
2 = 1,6 - 3,9 Hz	5 = 8,0 - 12,9 Hz
3 = 4,0 - 5,9 Hz	6 = 13,0 - 15,9 Hz

Table 18

Spectral Analysis: Eyes Open/Closed comparisons for Multi-infarct group (absolute scores)

Hemisphere	Spectral frequency band (Hz)	Eyes Open/ Closed	N	Mean Power (uV/Hz)	Standard deviation	t	p
R	Total	O	11	6205	2915		
		C	10	7481	3110	-0,97	ns
R	0,5-1,5	O	11	1287	440		
		C	10	1219	499	0,33	ns
R	1,6-3,9	O	11	985	675		
		C	10	1108	751	-0,4	ns
R	4,0-5,9	O	11	539	378		
		C	10	695	486	-0,83	ns
R	6,0-7,9	O	11	954	741		
		C	11	1296	793	-1,05	ns
R	8,0-12,9	O	11	1591	914		
		C	11	1956	1030	-0,88	ns
R	13,0-15,9	O	11	844	451		
		C	11	957	576	-0,51	ns
L	Total	O	11	6738	3174		
		C	10	7588	3115	-0,62	ns
L	0,5-1,5	O	11	1360	513		
		C	10	1180	514	0,8	ns
L	1,6-3,9	O	11	1068	601		
		C	10	1085	701	-0,06	ns
L	4,0-5,9	O	11	570	337		
		C	10	658	455	-0,51	ns
L	6,0-7,9	O	11	1106	763		
		C	11	1339	733	-0,73	ns
L	8,0-12,9	O	11	1733	989		
		C	11	2112	1014	-0,79	ns
L	13,0-15,9	O	11	924	454		
		C	11	962	449	-0,2	ns

Table 19

Spectral Analysis: Eyes Open/Closed comparisons for Multi-infarct group (proportional scores)

Hemisphere	Spectral frequency band (Hz)	Eyes Open/ Closed	N	% of Total Power	Standard deviation	t	p
R	0,5-1,5	O	10	24,0	10,6		
		C	10	18,8	9,7	1,14	ns
R	1,6-3,9	O	10	14,9	5,8		
		C	10	13,8	5,8	0,42	ns
R	4,0-5,9	O	10	7,8	2,8		
		C	10	8,3	3,5	-0,35	ns
R	6,0-7,9	O	10	13,5	6,5		
		C	10	16,8	6,1	-1,17	ns
R	8,0-12,9	O	10	25,8	8,9		
		C	10	27,9	9,9	-0,5	ns
R	13,0-15,9	O	10	14,7	7,6		
		C	10	14,3	8,2	0,11	ns
L	0,5-1,5	O	10	22,9	8,8		
		C	10	17,5	7,8	1,45	ns
L	1,6-3,9	O	10	14,6	2,6		
		C	10	13,3	4,8	0,75	ns
L	4,0-5,9	O	10	7,6	2,1		
		C	10	7,8	3,4	-0,16	ns
L	6,0-7,9	O	10	14,7	6,6		
		C	10	16,8	5,6	-0,77	ns
L	8,0-12,9	O	10	26,2	5,3		
		C	10	30,0	9,3	-1,1	ns
L	13,0-15,9	O	10	15,4	6,5		
		C	10	15,1	7,3	0,1	ns

Table 20

EEG reactivity ratios* for left and right hemisphere for Alzheimer's and Multi-infarct groups

	Alzheimer's	Multi-infarct	t score
Left Hemisphere			
Mean	75,05	89,5	-1,52
SD	21,82	38,76	
N	31	11	ns
Right Hemisphere			
Mean	74,51	87,23	-1,25
SD	25,63	37,11	
N	31	11	ns
t score	0,09 ns	0,14 ns	

* (in this and following figures) Reactivity ratio= absolute power for eyes open recording divided by the absolute power for eyes closed recording expressed as a percentage.

Table 21

Spectral Analysis for all subjectsfor left hemisphere, eyes closed, according to age (absolute scores)

Frequency Band (Hz)	Age Group (yrs)	Mean (uV /Hz)	SD	N	t - scores 1x2	1x3	2x3
Total	1:60-69	6827	3028	8	-0,19	-0,42	-0,55
	2:70-79	7094	3494	17			
	3:80+	7585	3282	16	ns	ns	ns
0,5-1,5	1:60-69	1505	498	8	0,84	0,11	1,55
	2:70-79	1275	692	17			
	3:80+	1254	298	16	ns	ns	ns
1,6-3,9	1:60-69	1053	677	8	0,34	-0,15	0,22
	2:70-79	958	641	17			
	3:80+	991	632	16	ns	ns	ns
4,0-5,9	1:60-69	523	373	8	-0,44	0,21	-0,32
	2:70-79	605	461	17			
	3:80+	574	372	16	ns	ns	ns
6,0-7,9	1:60-69	997	705	8	-1,07	0,17	-1,21
	2:70-79	1378	883	17			
	3:80+	1333	622	16	ns	ns	ns
8,0-12,9	1:60-69	1864	880	8	-0,24	-0,73	-0,87
	2:70-79	1980	1219	17			
	3:80+	2285	1224	16	ns	ns	ns
13,0-15,9	1:60-69	1001	426	8	0,18	-0,5	-0,27
	2:70-79	944	830	17			
	3:80+	1076	722	16	ns	ns	ns

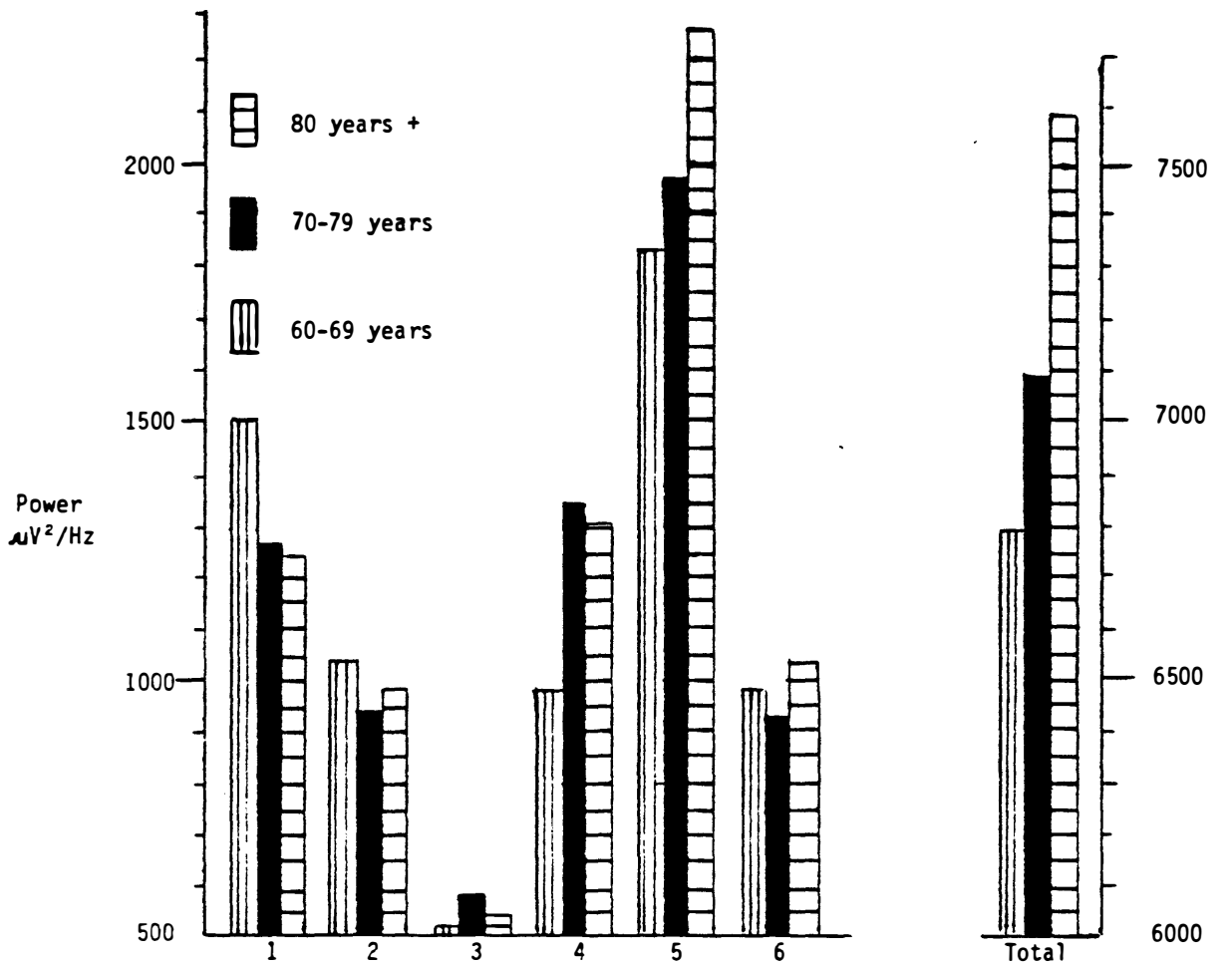
Table 22

Spectral Analysis for all subjects for left hemisphere, eyes closed, according to age (proportional scores)

Frequency Band (Hz)	Age Group	Percent Power	SD	N	t - scores 1x2	1x3	2x3
0,5-1,5	1:60-69	23,6	6,1	8	1,05	1,79	0,52
	2:70-79	20,0	8,6	16			
	3:80+	18,6	6,6	16	ns	*	ns
1,6-3,9	1:60-69	14,5	4,8	8	0,38	1,14	1,14
	2:70-79	13,6	5,7	16			
	3:80+	12,4	4,0	16	ns	ns	ns
4,0-5,9	1:60-69	6,9	2,6	8	-0,56	-0,17	0,53
	2:70-79	7,7	3,6	16			
	3:80+	7,1	2,7	16	ns	ns	ns
6,0-7,9	1:60-69	13,2	4,6	8	-1,5	-2,29	0,08
	2:70-79	18,3	8,9	16			
	3:80+	18,1	5,1	16	ns	**	ns
8,0-12,9	1:60-69	28,1	5,7	8	0,08	-0,73	-0,76
	2:70-79	27,7	12,1	16			
	3:80+	30,5	8,3	16	ns	ns	ns
13,0-15,9	1:60-69	15,5	4,8	8	0,91	0,68	-0,36
	2:70-79	12,8	7,6	16			
	3:80+	13,7	6,6	16	ns	ns	ns

Figure 11

Spectral Analysis: Frequency band and age for combined groups (absolute scores)



Frequency bands analysed

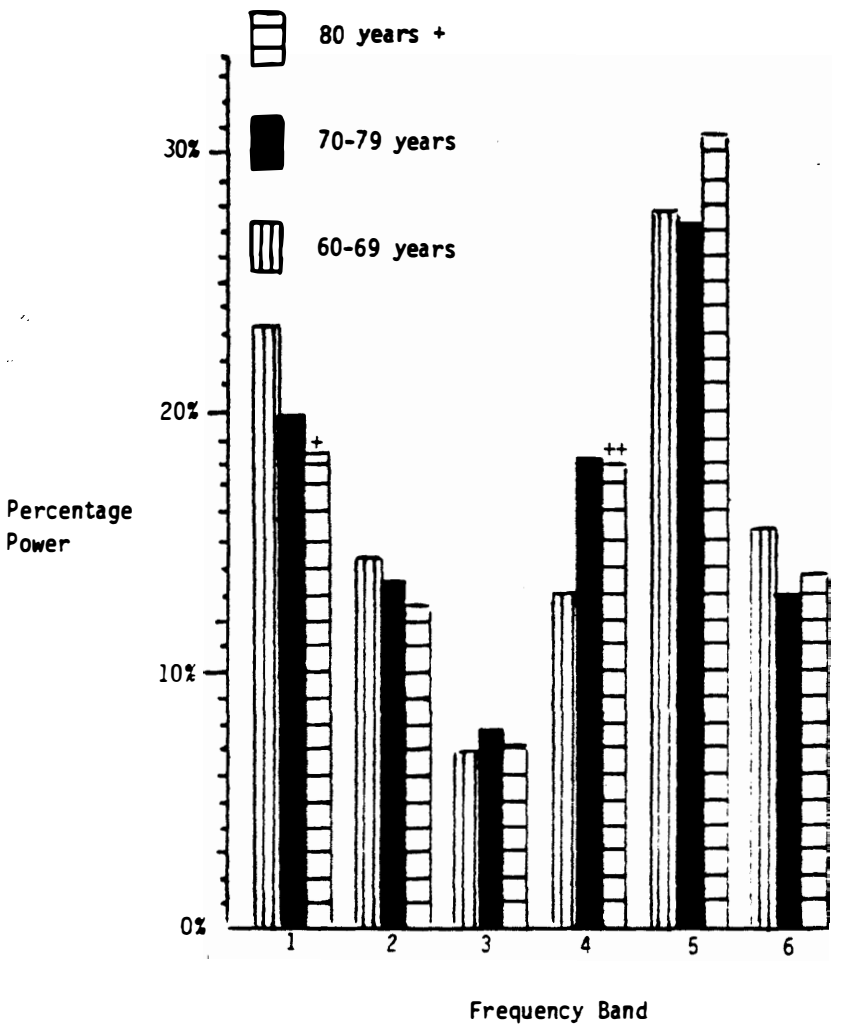
1 = 0,5 - 1,5 Hz	4 = 6,0 - 7,9 Hz
2 = 1,6 - 3,9 Hz	5 = 8,0 - 12.9 Hz
3 = 4,0 - 5,9 Hz	6 = 13,0 - 15,9 Hz

Figure 12

Spectral Analysis: Frequency band and age for all subjects (proportional scores)

+ 80-year-olds differ significantly from 60 - 69 year olds, $p<0.05$

++ 80-year-olds differ significantly from 60 -69 year olds, $p<0,025$



3). Once again, only the data from the left hemisphere eyes closed condition were examined. Results can be seen in Tables 24 and 25 and Figures 13 and 14.

FFT power increased for all frequency bands from group 1 to group 2, with the exception of the alpha band where power decreased from group 1 to 2. Power increased from group 2 to group 3 for all frequencies with the exception of the low delta band, where again, the power in this band showed a decrease. None of these trends was statistically significant (Table 24, Figure 13).

The proportional energy in each frequency band for each abnormality category is shown in Table 25 and Figure 14. Percent power in the low delta and alpha bands showed a decrease with increased EEG abnormality rating, while the percent power in the high delta and low theta bands showed an increase with abnormality rating. High theta activity increased from groups 1 to 2 and 1 to 3 but decreased from 2 to 3. Beta activity decreased from group 1 to group 2 but increased from 1 to 3 and from 2 to 3. Some of these changes were very small and were probably chance events. The only significant change was for the low theta band between groups 1 and 3 where a significant increase in power was seen with increased abnormality.

Table 26 shows that all the frequency bands measured became less reactive to eye closure with increased EEG abnormality. The alpha band was the most reactive for abnormality categories

Table 23

EEG reactivity ratios for total group according to age

Frequency Band (Hz)	Age (years) 60-69	70-79	80+
0,5-1,5	84,17	77,05	88,99
1,6-3,9	102,46	102,12	110,13
4,0-5,9	91,64	93,11	107,87
6,0-7,9	81,84	84,45	93,38
8,0-12,9	73,99	77,65	78,85
13,0-15,9	78,32	82,95	89,03

Table 24

Spectral Analysis for all subjects for left hemisphere, eyes closed, according to EEG abnormality rating (absolute scores)

Frequency Band	Abnorm Rating	Mean Power (uV/Hz)	SD	N	t - scores 1x2	1x3	2x3
Total	1:norm	6849	3389	13	-0,17	-0,89	-0,77
	2:mod	7057	3420	18			
	3:gros	8053	2961	10	ns	ns	ns
0,5-1,5	1:norm	1266	496	13	-0,38	-0,24	0,17
	2:mod	1347	651	18			
	3:gros	1309	318	10	ns	ns	ns
1,6-3,9	1:norm	914	687	13	-0,03	-1,09	-1,23
	2:mod	920	600	18			
	3:gros	1214	614	10	ns	ns	ns
4,0-5,9	1:norm	459	359	13	-0,9	-1,56	-0,67
	2:mod	592	438	18			
	3:gros	703	388	10	ns	ns	ns
6,0-7,9	1:norm	1126	750	13	-0,79	-0,83	-0,11
	2:mod	1348	780	18			
	3:gros	1380	737	11	ns	ns	ns
8,0-12,9	1:norm	2232	1255	13	0,51	0,42	-0,07
	2:mod	2003	1205	18			
	3:gros	2033	1022	11	ns	ns	ns
13,0-15,9	1:norm	953	677	13	-0,07	-0,71	-0,55
	2:mod	973	850	18			
	3:gros	1131	531	11	ns	ns	ns

RAAD VIR GEESTESWETENSKAPLIKE NAVORSING
HUMAN SCIENCES RESEARCH COUNCIL

Table 25

Spectral Analysis for all subjects for left hemisphere, eyes closed, according to EEG abnormality rating (proportional scores)

Frequency Band (Hz)	Abnormal Rating	Percent Power	SD	N	t - scores 1x2	1x3	2x3
0,5-1,5	1:norm	21,7	8,5	13	0,41	1,32	1,05
	2:mod	20,5	7,6	17			
	3:gros	17,6	5,6	10	ns	ns	ns
1,6-3,9	1:norm	12,5	4,5	13	-0,28	-1,18	-0,89
	2:mod	13,0	5,2	17			
	3:gros	14,8	4,8	10	ns	ns	ns
4,0-5,9	1:norm	6,1	2,4	13	-1,5	-2,01	-0,31
	2:mod	7,8	3,5	17			
	3:gros	8,2	2,6	10	ns	**	ns
6,0-7,9	1:norm	14,9	6,1	13	-1,56	-0,85	0,72
	2:mod	19,1	8,1	17			
	3:gros	17,0	5,5	10	ns	ns	ns
8,0-12,9	1:norm	32,1	9,9	13	1,07	1,67	0,57
	2:mod	28,1	10,3	17			
	3:gros	26,0	6,8	10	ns	ns	ns
13,0-15,9	1:norm	14,2	6,7	13	0,9	-0,45	-1,29
	2:mod	12,2	5,5	17			
	3:gros	15,6	8,2	10	ns	ns	ns

071728

Figure 13

Spectral Analysis: Frequency band and EEG abnormality rating for all subjects (absolute scores)

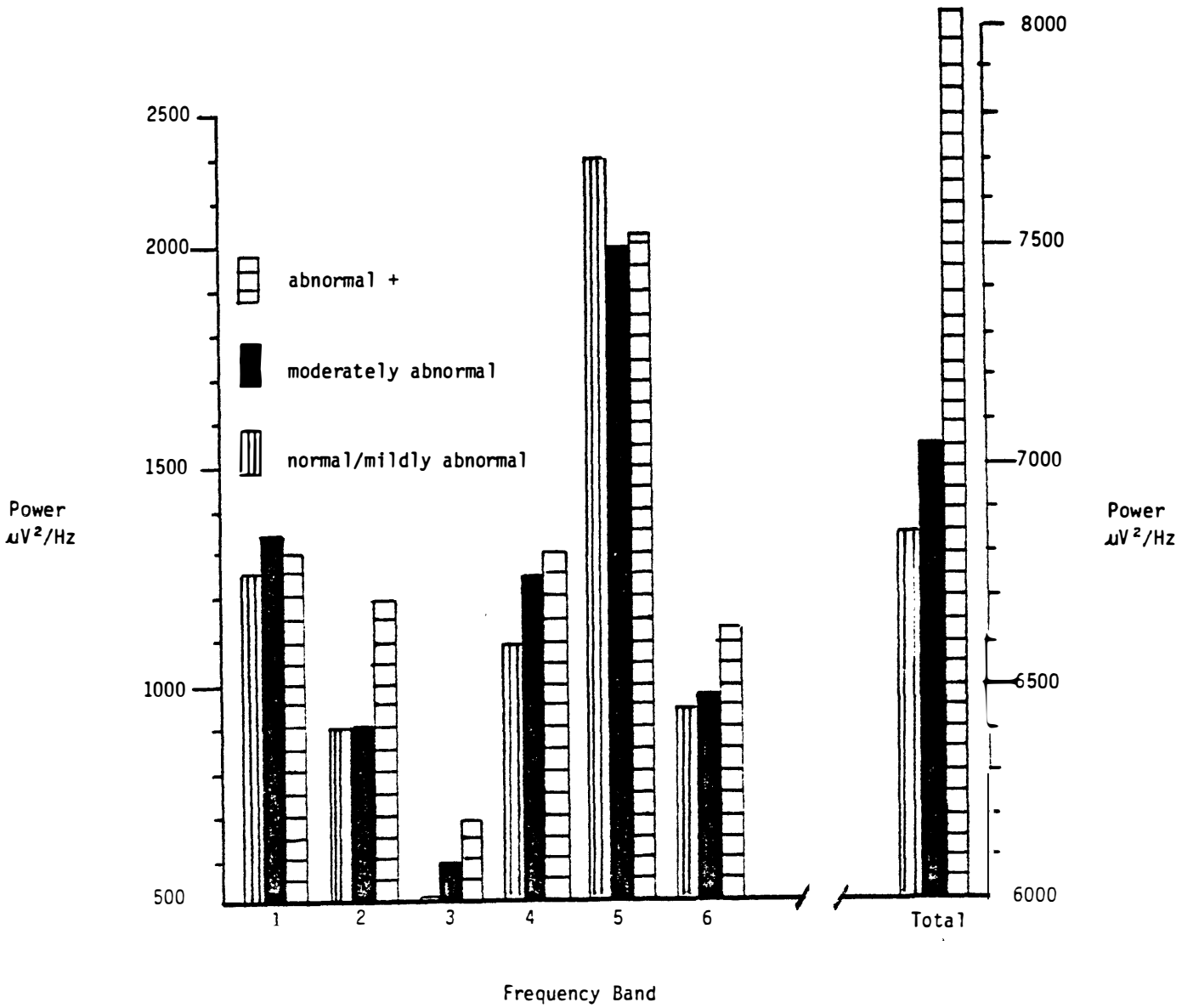


Figure 14

Spectral Analysis: Frequency band and EEG abnormality for all subjects (proportional scores)

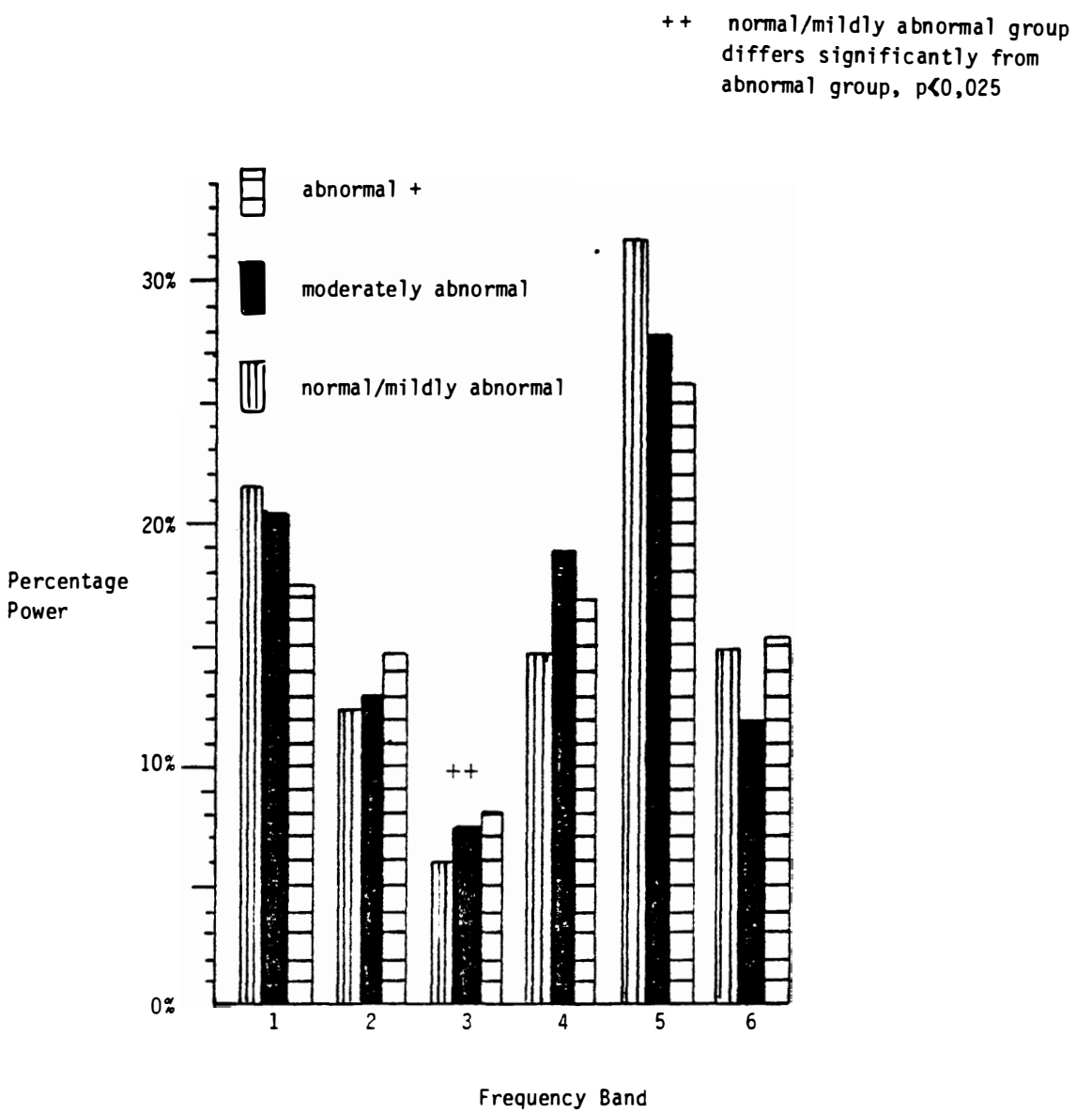


Table 26

EEG reactivity ratios according to abnormality category

Frequency (Hz)	EEG Abnormality Rating 1:normal mildly abn	2:moderately abnormal	3:grossly abnormal
Total	78,32	77,94	95,87
0,5-1,5	100,95	104,75	110,77
1,6-3,9	90,70	102,50	99,67
4,0-5,9	86,49	84,14	93,31
6,0-7,9	69,12	72,75	93,55
8,0-12,9	63,31	70,00	97,39
13,0-15,9	76,31	80,90	98,32

1 and 2, but for category 3, the high theta band showed the most reactivity. There was, however, very little difference seen in reactivity between the various frequency bands in those subjects falling into abnormality category 3 and all were close to 100 indicating little response to eye closure.

3) PRVEP

Examples of PRVEP recordings appear in Figures 15 to 17. Results were examined in two ways. Firstly, the individual records were rated as either normal or abnormal according to standard clinical criteria and groups compared for abnormality. Secondly, the mean values for the latencies and amplitudes of P100 were compared for the groups.

The PRVEP in Figure 15 is within normal limits and shows a replicable response for all three recording conditions. Figure 16 shows a record that is abnormally delayed bilaterally, and Figure 17 shows a record with a unilateral delay and poor replicability.

Twelve (29%) of the 42 PRVEPs were abnormal according to the usual clinical criteria. Six (14% of the total) had bilateral abnormalities, 4 (10%) left-sided abnormalities and only 2 (5%) had right-sided abnormalities. There was no difference between the AD and MID groups in the number of bilateral or unilateral abnormalities (Table 27).

Table 28 shows the PRVEP results according to age groups. Three

of the 8 subjects in their seventh decade were abnormal, two of these were in the MID group. Only one subject out of the 17 who were in their eighth decade was abnormal, but eight out of 17 in their ninth or tenth decades were abnormal.

The PRVEP results showed some relationship to the CT scan abnormality. Forty-one percent of the subjects with grossly abnormal CT scans had abnormal PRVEPs. This reduced to 37% for the subjects with moderately abnormal scans, and those with mildly abnormal or normal scans showed no PRVEP abnormalities (Table 29).

A similar tendency was seen if PRVEP results were compared with the clinical EEG results (Table 30). There was, however, no difference in the number of abnormal PRVEPs for those with normal/mildly abnormal EEGs compared with those who had moderately abnormal EEGs. Forty-five percent of those with abnormal EEGs had abnormal PRVEPs.

Table 31 gives the mean P100 latency, and Table 32 the mean amplitude for the AD and MID groups. There were no significant latency or amplitude differences between the two groups. Tables 33 and 34 give the P100 latencies and amplitudes according to age, and Tables 35 and 36 the P100 latencies and amplitudes according to EEG abnormality. Neither age nor EEG abnormality appeared to influence latency or amplitude measures, although there was a tendency for the P100 latency to be slightly prolonged in the the group of subjects with the most abnormal EEGs. This group, however, was very small.

Figure 15

A normal PRVEP from a 78-year-old female with Alzheimer's disease

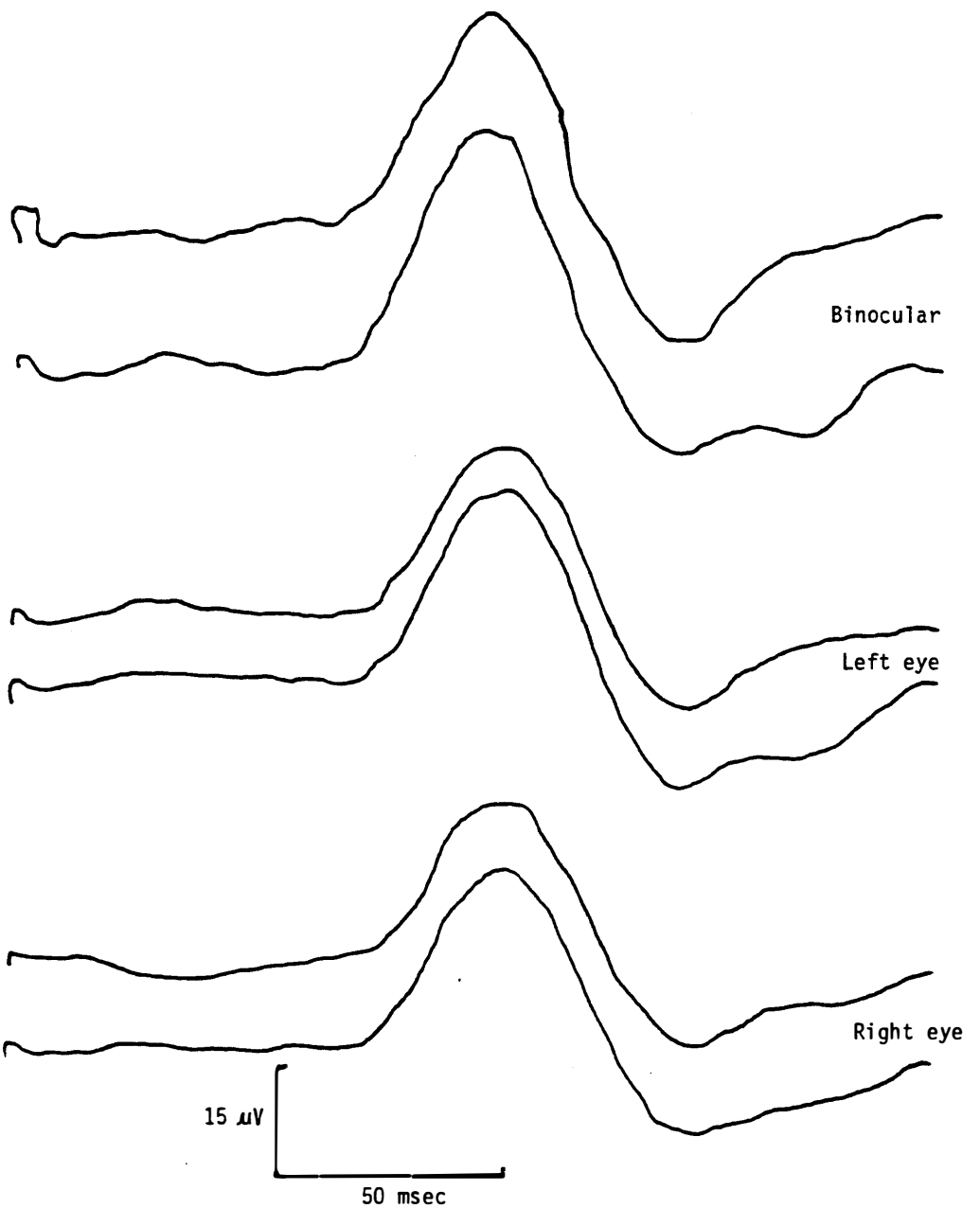


Figure 16

Bilaterally delayed PRVEP from a 74-year-old male with Alzheimer's disease

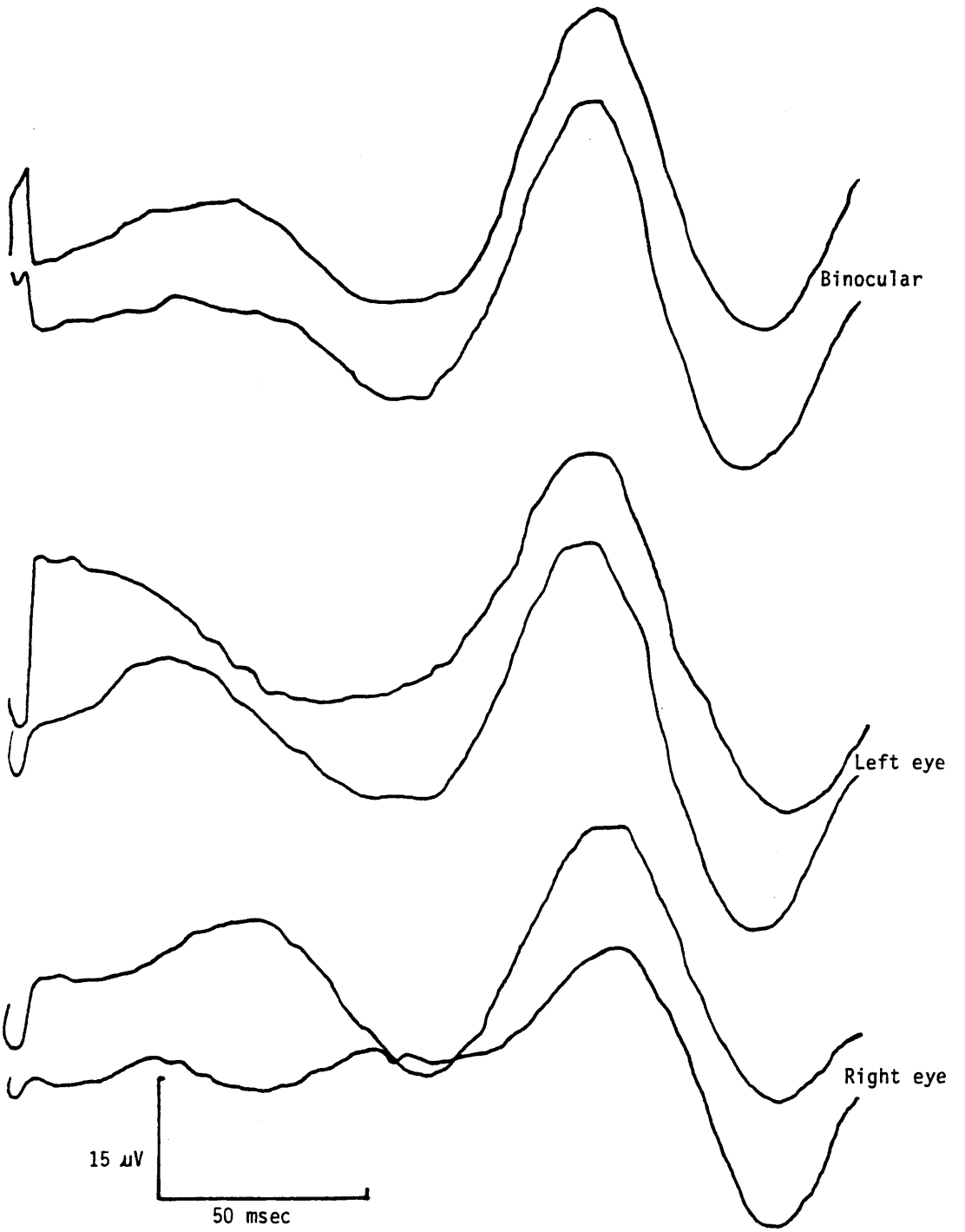


Figure 17

Left-sided delay in PRVEP form a 82-year-old female with Alzheimer's disease

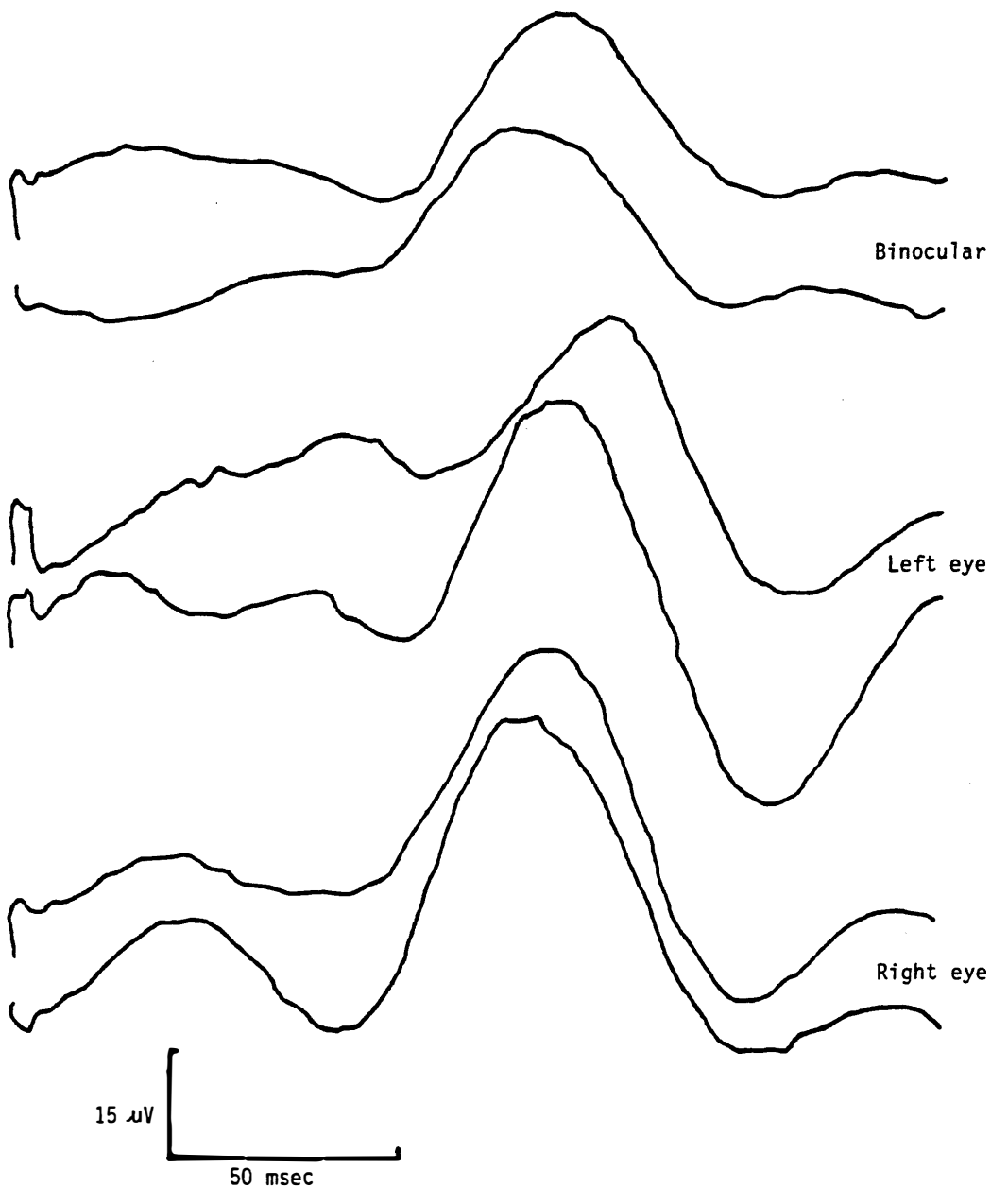


Table 27

PRVEP abnormality for Alzheimer's and Multi-infarct groups

	Alzheimer's	Multi-Infarct	Total
Normal	23 (74%)	7 (64%)	30 (71%)
Binocular Abnormality	4 (13%)	2 (18%)	6 (14%)
Left-sided Abnormality	3 (10%)	1 (9%)	4 (10%)
Right-sided Abnormality	1 (3%)	1 (9%)	2 (5%)

Table 28

PRVEP abnormality and age for Alzheimer's and Multi-infarct groups

	60-69	70-79	80+	Total
Normal	5	16	9	30 (71%)
Binocular Abnormality	2	0	4	6 (14%)
Left-sided Abnormality	0	0	4	4 (10%)
Right-sided Abnormality	1	1	0	2 (5%)
Total Abnormal	3 (37,5%)	1 (5,9%)	8 (47,1%)	12 (29%)

Table 29

PRVEP abnormality and CT scan atrophy rating for Alzheimer's and Multi-infarct groups

	Atrophy rating		
	Normal/Mild	Moderate	Marked
Normal PRVEP	4 (100%)	8 (63%)	17 (59%)
Abnormal PRVEP	0	3 (37%)	7 (41%)
Total	4	11	24

Table 30

PRVEP abnormality and EEG abnormality rating for Alzheimer's and Multi-infarct groups

	EEG abnormality rating		
	Normal/Mild	Moderate	Abnormal +
Normal PRVEP	10 (77%)	14 (78%)	6 (55%)
Abnormal PRVEP	3 (23%)	4 (22%)	5 (45%)

Table 31

P100 latencies for Alzheimer's, Multi-infarct and a control group

	Alzheimer's	Multi-Infarct	Total	Young Control group (Murdoch 1981)
Binocular				
Mean Latency (msec)	112,6	108,8	111,6	105,7
SD	10,3	9,8	10,2	9,5
N	27	9	36	20
t score	2,346	0,805	2,124	
df	45	27	54	
p	<0,025	NS	<0,025	
Left Eye				
Mean Latency (msec)	115,3	111,6	114,4	104,8
SD	10,3	11,8	10,6	8,1
N	25	8	33	20
t score	3,727	1,759	3,368	
df	43	26	51	
p	<0,001	<0,05	<0,001	
Right Eye				
Mean Latency (msec)	115,3	114,1	115,0	104,7
SD	9,3	11,1	9,6	9,5
N	25	8	33	20
t score	3,763	2,257	3,801	
df	43	26	51	
p	<0,001	<0,025	<0,001	

(all comparisons with young control group values)

Table 32

P100 amplitude for the Alzheimer's and Multi-infarct groups

	Altzheimer's	Multi-Infarct	Total
Binocular			
Mean Amplitude (µV)	8,31	3,82	7,19
SD	6,83	2,62	6,33
N	27	9	36
Left Eye			
Mean Amplitude (µV)	8,13	6,62	7,78
SD	7,54	8,04	7,49
N	24	8	32
Right Eye			
Mean Amplitude (µV)	9,60	10,46	9,81
SD	11,53	10,19	11,07
N	25	8	33

Table 33

PRVEP Latency and age for all subjects compared with a control group

	Age group			
	60-69	70-79	80+	Young Control group (Murdoch 1981)
Binocular Mean Latency (msec)	114,4	107,4	114,4	105,7
SD	11,8	7,8	10,6	9,5
N	7	14	15	20
t score	1,96	0,55	2,55	
df	25	32	33	
p	<0,05	NS	<0,01	
Left Eye Mean Latency (msec)	114,7	119,1	115,8	104,8
SD	12,7	8,8	10,2	8,1
N	7	14	12	20
t score	2,4	4,89	3,37	
df	25	32	30	
p	<0,025	<0,001	<0,001	
Right Eye Mean Latency (msec)	118,0	112,7	116,0	104,7
SD	12,6	8,5	9,0	9,5
N	7	14	12	20
t score	2,93	2,52	3,32	
df	25	32	30	
p	<0,001	<0,01	<0,001	

(all comparisons with young control group)

Table 34

P100 Amplitude and age for all subjects

	60-69	70-79	80+
Binocular			
Mean Amplitude (μV)	6,21	7,97	6,91
SD	4,84	6,94	6,49
N	7	14	15
Left Eye			
Mean Amplitude (μV)	6,67	8,09	8,03
SD	3,99	9,03	7,61
N	7	14	12
Right Eye			
Mean Amplitude (μV)	5,33	12,90	8,12
SD	2,39	15,40	6,89
N	7	14	12

Table 35

P100 Latency and EEG Abnormality rating for all subjects compared with a control group

	EEG Abnormality Rating			
	Normal/Mild	Moderate	Abnormal	Control (Murdoch 1981)
Binocular				
Mean Latency (msec)	111,1	110,7	120,5	105,7
SD	11,2	9,5	8,2	9,5
N	13	20	3	20
t score	1,49	1,66	2,55	
df	31	38	21	
p	NS	NS	<0,01	
Left Eye				
Mean Latency (msec)	113,5	113,9	124,1	104,8
SD	11,9	10,0	9,9	8,1
N	11	20	2	20
t score	2,42	3,16	3,17	
df	29	38	20	
p	<0,025	<0,001	<0,001	
Right Eye				
Mean Latency (msec)	115,3	114,2	121,3	104,7
SD	10,2	9,7	4,4	9,5
N	12	19	2	20
t score	2,97	3,09	2,40	
df	30	37	20	
p	<0,001	<0,001	<0,05	

(all comparisons with young control group)

Table 36

P100 Amplitude and EEG Abnormality rating for all subjects

	EEG Abnormality rating		
	Normal/Mild	Moderate	Abnormal
Binocular			
Mean Amplitude (μV)	5,42	8,74	4,51
SD	4,16	7,42	3,28
N	13	20	3
Left Eye			
Mean Amplitude (μV)	4,42	9,51	7,25
SD	2,79	8,82	6,48
N	10	20	2
Right Eye			
Mean Amplitude (μV)	4,60	13,74	3,76
SD	2,68	13,23	0,13
N	12	19	2

DISCUSSION

The results show that in patients with senile dementia the EEG is a more sensitive index of CNS dysfunction than is the PRVEP. Sixty nine percent of our subjects had abnormal EEGs while only 29% had abnormal PRVEPs. These results accord with those of Visser et al. (1985). They found that 65% of demented subjects had abnormal EEGs, but only 24% had abnormal PRVEPs. They concluded that the EEG detected abnormalities in dementia more often than the VEP did.

Comparison of our results with those previously published is made difficult as not all authors give details of the degree of dementia of their subjects and EEG abnormality varies with the severity of dementia. In older studies, demented subjects are included in groups with senile psychosis or organic brain syndrome. Our results generally, however, appear to agree with those previously reported. For example, in 1954 Mundy-Castle et al. recorded EEGs from 104 patients (mean age 73,4 years SD 9,1) with senile psychosis. Fifty-four percent of this group had abnormal EEGs compared to only 24% of a normal control group. The percentage of abnormal EEGs increased and alpha index decreased significantly with increased dementia.

Roubicek (1977) found 42,4% of his sample of 159 old persons (mean age 72,5, range 46-92) to have abnormal EEGs. They were described as having "mild psychic disability". In comparison, Gereby (1980) found 89% of his subjects with severe dementia had abnormal EEGs. Our subjects had mild to moderate dementia

and our findings, therefore, are in keeping with these results. Unfortunately data were not available to allow a comparison of the degree of dementia and EEG abnormality in individuals in our sample.

Specific EEG abnormalities in our sample consisted of slowing of the dominant occipital rhythm together with focal slow wave abnormalities maximally in the left fronto-temporal area. This pattern has previously been described (Mundy-Castle, 1962; Obrist and Henry, 1958; Markand, 1986; Celesia, 1986). These authors, however, reported that the dominant frequency of their demented patients was lower than 8 Hz, whereas only 21% of our subjects had a dominant frequency less than 8 Hz, with the majority (52,4%) having a frequency in the 8 Hz band and 26,2% with frequencies of 9 Hz and greater (Table 8). The mean frequency of our sample was 8,7 Hz. Again, this probably reflects the relatively mild state of dementia in our subjects. Otomo (1966) reports very similar results. He investigated the alpha rhythm in three groups of elderly subjects (60 years and older). The normal subjects had a mean alpha frequency of 9,47 Hz while the "neurological subjects" had a mean frequency of 8,65 Hz. The modal frequency for his neurological group was also 8 Hz and approximately 10% had frequencies of less than 8 Hz.

The high incidence of left fronto-temporal focal abnormalities found in our subjects has also previously been reported. Authors are however divided as to the significance of this

feature. Obrist and Henry (1958) recorded EEGs from 45 patients with "clear cut evidence of brain syndrome" and compared them with EEGs from 45 elderly subjects with functional illnesses. They found a high incidence of diffuse slow activity (delta and theta) in the organic group and predominantly left fronto-temporal focal abnormalities in both the organic and functional groups. They concluded that diffuse slow wave activity was the most reliable index of mental deterioration and that focal slow waves were not related to psychological deterioration.

Busse (1973) reported that 30 to 40% of healthy elderly individuals showed focal EEG abnormalities maximally in the temporal area, and that the incidence of temporal lobe abnormalities appeared to increase with increasing age. Six percent of the normal population between the ages of 20 and 40 years showed these abnormalities increasing to 20% for the population between 40 and 60 years old. Busse (1973) also claimed that there was no firm evidence correlating these disturbances with psychological functioning or mental status.

Bassi and Sbrascini (1983), however, stated that the EEG parameters used in assessing young adult EEGs were still valid in patients over the age of 80 years. They found that EEG abnormality, using these criteria, correlated well with patient history and scores on psychometric tests. They found focal slow wave activity to be associated with marked memory dysfunction.

These conflicting findings may reflect differences in the meaning of normality in the aged.

regarded as "normal" in an elderly person would not be so in young adults. These include slow and cautious motor activity and changes in intellectual ability. There is a need to establish a normative scale of physical and psychological abilities in the aged to help determine the degree of dysfunction in this group (Hubbard et al. 1976). Such a scale is not available at present and some of the changes seen in the EEGs of the elderly described as "normal" or "healthy" may reflect certain pathological processes accompanying old age, rather than an association with ageing in the CNS itself. These processes may include arteriosclerosis, mild metabolic deficiencies or drug toxicity.

Research in this field is further complicated by the fact that samples of old people are often biased. It is difficult to gather a sample of 80 year-olds from the general population. Samples are usually collected at clinics or from old age homes and may be biased towards less healthy individuals. Individuals who have reached an advanced age may represent the "survivors" in the population and research performed on them may not reflect the norm. Hubbard et al. (1976) recorded EEGs from 10 centenarians described as healthy and living in the community. Seven were regarded as clinically normal while three had evidence of CNS dysfunction. The average posterior background frequency of the 7 clinically normal individuals was 8,62 Hz. Only three of these subjects showed focal temporal signs. These EEGs appear to be more "normal" than those reported by other authors often using younger subjects (for example, Roubicek

(1977)

iate

various types of dementia (Celesia 1986; Markand 1986). In our study, no statistically significant differences were found between the AD and MID groups. This was probably due to the small size of the MID group. as trends compatible with the above authors' findings emerged. Further, all but one of our MID subjects showed signs of cortical atrophy (Table 5) and they could be regarded as being of mixed diagnosis (AD and MID). This is reflected in the EEG abnormalities. Fifty-two percent of the AD group had moderately abnormal EEGs compared with only 18% of the MID group. However, only 16% of the AD group were rated as abnormal or worse while 55% of the MID group were in this category (Table 6), probably reflecting the greater abnormality associated with the combined diseases. Further evidence for this is in Table 7. Sixty-four percent of the AD group, but 82% of the MID group had focal EEG abnormalities. Markand (1986) states "the presence of focal EEG findings ..... strongly suggests the possibility of MID rather than AD" (p 13).

Although EEG findings in dementia are non-specific, both Markand (1986) and Celesia (1986) claim the EEG is useful in the differential diagnosis of this condition. For example, in Pick's disease the EEG is usually normal in the early stages, whereas in this stage of AD, slow wave activity is usually seen. Patients with Huntington's disease usually have very low amplitude or "flat" records, while patients with Creutzfeldt-

Jacob disease show a distinctive EEG pattern of periodic lateralised epileptiform discharges (PLEDS).

The real value of the EEG in dementia, however, is to assist in identifying those forms that are reversible or responsive to treatment. Elderly patients are often cognitively impaired as a result of psychiatric disorders. In such cases the EEG is usually normal and treatment for the psychiatric disorder often reverses the dementia. Markand (1986) says that chronic encephalopathies related to toxic or metabolic states closely simulate the degenerative dementias. Correct diagnosis of these disorders is important as they are potentially reversible.

Although the EEG can be used to assist in the differential diagnosis of dementia it has several drawbacks. It is relatively time-consuming and analysis of the record requires skill and experience. A procedure permitting quick and objective analysis would be a considerable advantage. Frequency analysis of the EEG record by means of a Fast Fourier Transform may fulfill this need. The FFT provides an objective quantification of the EEG and allows specification of the total power in the EEG, the power in pre-selected frequency bands, the mean frequency in each band and also the peak frequency of the EEG. The FFT requires only a brief period of recording and may be obtained from a limited array of electrodes. It also relies less on the skill and experience of the electroencephalographer than the clinical EEG procedure. These factors led to the use of the FFT in the present

research.

We did not have access to a normal aged control group and hence, normative FFT values were not available. The value of the FFT in the diagnosis of dementia in our subjects could, therefore, not be considered. However, the literature provides several studies which have addressed this issue. Visser et al. (1985) found that 57% of their subjects with dementia showed power in the delta band in excess of 45% of the total EEG power, whereas only 10% of a normal group and 10% of a group with behavioural disorders did so. A reduced peak frequency on the FFT was almost as powerful in discriminating between demented patients and the other groups. They stated that spectral analysis of the EEG revealed EEG abnormalities as often as standard visual inspection.

Giaquinto and Nolfe (1986) compared normal middle-aged subjects with normal elderly and demented elederly subjects on several FFT measures. There were very few differences between their young and old normals, but the demented group had significant increases in the percentage delta and theta power, and a decrease in the percentage alpha power. Coben et al. (1985) reported a longitudinal study comparing patients with AD and healthy controls on three tests conducted over 2,5 years. The percentages of alpha, beta, theta and delta power in the FFT were examined. The percentage theta power distinguished between all four stages of dementia (control, mild, moderate and severe). Penttila et al. (1985) also found that percentage power in the theta band distinguished patients suffering from

mild AD from normal controls. They indicated that slowing of the dominant occipital rhythm and an increase of diffuse, irregular slow waves, typically regarded as EEG signs of AD, are only characteristic of an advanced stage of the disease. An increase in theta power is the most reliable and sensitive indicator of mild AD.

FFT analysis of the EEGs we recorded yielded similar results to those seen in previous studies. Figure 6 shows the percentage power in three frequency bands of our subjects compared with those of Penttila et al. (1985) and Coben et al. (1985) for mildly demented subjects. Percent power in the delta and theta bands is similar in the three groups, but the power in the alpha band is reduced in our group. This may reflect a slight difference in degree of dementia as our subjects were described as having mild to moderate dementia. It may also be due to a lower level of arousal or alertness in our group.

The previous studies cited do not present data on the absolute power in each frequency band. This practice has been criticised by Fein et al. (1986) who point out that percentage power only gives information on spectral shape and that information on the absolute level of EEG activity in each band is lost. Also, this approach introduces an interdependence between frequency bands. For example, a significant increase in the proportion of delta power may result either from a real increase in delta activity or from a decrease in power in the other frequency bands. The conclusion that an increase in relative delta power

indicates an increase in absolute energy in the delta band is, therefore, unwarranted.

Fein et al. (1986) indicate that use of percentage power in preference to absolute power is based on the belief that absolute power spectra are unreliable. Gasser et al. (1985), however, have shown that the test-retest reliability of absolute power spectra over a period of 10 months is acceptable. They also showed that FFT analysis of 20 seconds of EEG activity is as reliable as analysis of 40 or 60 seconds. Fein et al. (1984) showed that test-retest reliability can extend over a period of 1 to 3 years. Clearly, whenever possible, both percentage power and absolute power values of FFT should be reported.

Murdoch (1985), using the same equipment and recording procedures as were used in the present study, reported absolute spectral values in a study of epileptic patients. His subjects were aged 23 to 60 years (mean 40,9; SD 10,2). All had been diagnosed as epileptic and were receiving anti-convulsant medication. The absolute total power in his sample for the left hemisphere eyes closed condition was 46% higher than the total absolute power seen in our sample for this condition. Interestingly, the proportional values showed very little difference between the two groups. Both groups showed a decrease in the alpha band and an increase in the theta band compared with normal values seen in the literature. The emergence of this pattern in the two groups is probably for different reasons. In our group it could be related to the

presence of AD, and in the epileptic group to reduced level of arousal due to medication (Murdoch 1985). Further research would be needed to determine the significance of the differences in total absolute power in these two groups.

Our FFT results in our study were similar to the clinical EEG results and revealed very few differences between the AD and MID groups, and no interhemispheric differences were seen. However, a significant increase in absolute power in the alpha and high theta bands, in response to eye closure, was seen in the AD group (Table 16) but not in the MID group (Table 17). These differences were even more striking if the proportional values were examined (Tables 17 and 19). This was not reflected in alpha reactivity but this may have been because the alpha activity had been replaced by theta in the AD group.

Age changes in the FFT results were anomalous. The power in the delta band reduced with increasing age, and power in the alpha band increased with age. Although not statistically significant for the absolute values, the proportional values in the oldest and youngest groups differed significantly. Previous results suggest that these are in the wrong direction. A possible explanation may be that the younger subjects were more alert and apprehensive in the novel test situation. This could have resulted in more eye movement recorded in the low delta band and less alpha activity than in the less alert and thus less anxious older subjects.

Although the power in the alpha band increased with age in our

sample, the reactivity of the alpha band to eye closure tended to decrease with age (Table 23). If alpha reactivity was examined in relation to EEG abnormality, a decrease in reactivity is seen with increasing abnormality (Table 26), with the most abnormal group showing almost no reactivity. The relationship between alpha reactivity, as measured by the FFT, dementia and normal ageing needs to be examined further, as this measure may be useful in discriminating between normal and pathological ageing and has not been considered by others.

The relationship between FFT measures and clinical EEG abnormality was examined by comparing FFTs of subjects catagorised according to EEG abnormality (Table 24, Figures 13 and 14). No statistically significant differences in FFT were seen, probably because of the large variance in each group. However, it appears that the FFT measures associated with clinical EEG abnormality are an increase in high delta activity, an increase in both low and high theta activity and a decrease in alpha activity.

The PRVEP was the least sensitive of the procedures examined. According to the usual clinical criteria, only 29% of the subjects had abnormal PRVEPs. The number of abnormalities within each group showed no relationship to clinical group (Table 27) or to age (Table 28). The tendency for the number of PRVEP abnormalities to increase with increased CT scan (Table 29) and EEG abnormality (Table 30) may be because the subjects with CNS dysfunctions had relatively more difficulty

cooperating on the test. It is also possible that an abnormal PRVEP reflected peripheral visual dysfunction rather than CNS dysfunction. Most subjects wore glasses but had not had their vision recently checked and visual acuity was not assessed before the PRVEP was recorded. Some subjects with abnormalities had recently had cataracts removed. Visser et al. (1985) found the early peaks (<100msec) of the PRVEP to be within normal limits in demented subjects and point out that these reflect activity in the primary visual cortex. They state that normality of these peaks in demented subjects is good evidence for adequate cooperation in the recording procedure. Our results underline the importance of checking visual acuity before recording PRVEPs and of noting the level of cooperation of each subject.

Although most studies have reported no change in the early peaks of the PRVEP with dementia, the later peaks of the PRVEP and most of the peaks of the flash VEP have been reported to be abnormal (Visser et al., 1985; Harding et al., 1985). These peaks appear to originate in the visual association area rather than the primary visual cortex (Coben et al., 1983). It appears, therefore, that intra-cortical neural transfer is slowed in AD.

The etiology of AD is still uncertain. Kaufman (1985) points out that many statistical associations have been noted in AD patients. For example, head trauma, hyperthyroidism and increased aluminium concentrations have been found more frequently in AD patients than in age-matched controls. The

role of the neurotransmitters in AD has also recently been demonstrated.

Berdjis and Demisch (1985) observed a slowing of the flash VEP, but not of the PRVEP, after what they describe as a subchronic administration of the MAO type A inhibitor, pirlindole, in 6 healthy male volunteers. The pattern of a normal PRVEP and an abnormal flash VEP had previously only been seen in patients with presenile dementia (Harding et al., 1985). Pirlindole affects the de-amination of serotonin and noradrenaline and has serotonin reuptake inhibiting properties (Berdjis and Demisch, 1985). They conclude that this indicates that different neuronal systems are involved in processing flash and pattern stimuli in man. This finding also suggests that the adrenergic and serotonergic pathways are involved in AD.

Wright and Harding (1985) have shown similar results in young volunteers after an injection of the centrally acting anticholinergic drug, Hyoscine Hydrobromide. They conclude that the delay in the flash VEP seen in dementia could be due to a reduction in cholinergic activity. It has previously been shown that there is a reduced concentration of acetylcholine (ACh) and the enzyme choline acetyletransferase (CAT) in the brains of demented individuals (Kaufman, 1985). Besides producing the VEP pattern typically associated with dementia, the administration of anticholinergic drugs in normals temporarily interferes with memory, and produces the clinical and pathological

the loss of cholinergic neurones (Heilman and Valenstein, 1985).

It is possible that AD is not a single entity. One form may, therefore, be based on a loss of cholinergic fibres and the second more related to the norepinephrine system (Bousfield, 1982). Filley et al. (1986) suggested the possible existence of two forms: a form with early onset and one with later onset. Those individuals with early onset AD had significantly more language impairment compared with those individuals with late onset disease, who had more visuoconstructual deficits (Filley et al., 1986). Treves et al. (1986) also showed the existence of two forms of the disease.

Recently, infectious agents known as prions have been identified. These, unlike viruses, are slow to produce symptoms and they contain no nucleic acid. They are considered to be responsible for at least two other degenerative neurological illnesses, namely, scrapie (a disease of sheep and goats) and Creutzfeldt-Jakob disease. Under the electron microscope, the senile plaque amyloid found in the brains of demented individuals, resembles these prions (Kaufman, 1985).

The presence of cognitive decline in patients with dementia has led to an interest in the role of the Cognitive Evoked potential (also known as the Event Related Potential or ERP) in the diagnosis of dementia. Pfefferbaum et al. (1984) claim that it has been well established that the P3 component of the ERP is greatly influenced by cognitive factors and they have also

shown that P300 latency increases with age. If the ERP is to be useful in the identification of patients suffering from dementia, then it will have to be shown that the increase in P300 latency in these patients is greater than in normal ageing. There is some evidence for this (Clair et al., 1985, Lai, 1985), although negative findings have also been reported (Sleats and Forgens, 1984).

REFERENCES

ADAMS R. D. AND VICTOR M. (1977). Principles of Neurology. New York, McGraw Hill.

AMERICAN PSYCHIATRIC ASSOCIATION. (1980). Diagnostic and Statistical Manual of Mental Disorders, Third Edition. Washington, D.C., APA.

BASSI P. AND SBRASCINI S. (1983). EEG in subjects over 80 years. Electroencephalography and Clinical Neurophysiology, 55, 6p-7p.

BERDJIS H. AND DEMISCH L. (1985). Changes in flash but not pattern evoked cortical potentials after subchronic application of a monoamine oxidase (mao) type A inhibitor in man. Electroencephalography and Clinical Neurophysiology, 62, 77-80.

BERG L. (1985). Does Alzheimer's disease represent an exaggeration of normal aging? Archives of Neurology, 42, 737-739.

BESDINE R. W., (1980). Geriatric medicine in America: An overview. In: Perspectives on geriatric medicine. NIH Publication No. 81-1924 (pp. 4-10).

BOUSFIELD D. (1982, June 3). Old age, dementia and the brain. New Scientist, p 648.

BUSSE E.W. (1973). Round table discussion: EEG in gerontology. In Everts W. H. (Moderator), Seventh anual meeting of the American Medical Electroencephalographic Association, Denver. Clinical Electroencephalography, 4, 152-162.

CELESIA G.G. (1986). EEG and event-related potentials in aging and dementia. Journal of Clinical Neurophysiology, 3, 99-111.

COBEN L.A., DANZIGER W.L. AND HUGHES C.P. (1983). Visual evoked potentials in mild senile dementia of Alzheimer type. Electroencephalography and Clinical Neurophysiology, 55, 121-130.

COBEN L.A., DANZIGER W. AND STORANDT M. (1985). A longitudinal EEG study of mild senile dementia of the Alzheimer type: Changes at 1 year and at 2,5 years. Electroencephalography and Clinical Neurophysiology, 61, 101-112.

CUMMINGS J. L. (1985). Clinical Neuropsychiatry. Orlando, Grune & Stratton.

FEIN G., GALIN D., YINGLING C.D., JOHNSTONE J. AND NELSON M.A. (1984). EEG spectra in 9-13 year old boys are stable over 1 to 3 years. Electroencephalography and Clinical Neurophysiology, 58, 517-518.

FEIN G., GALIN D., YINGLING C.D., JOHNSTONE J., DAVENPORT L. AND HERRON J. (1986). EEG spectra in dyslexic and control boys during resting conditions. Electroencephalography and Clinical Neurophysiology, 63, 87-97.

FILLEY C.M., KELLY J. AND HEATON R.K. (1986). Neuropsychological features of early- and late-onset Alzheimer's disease. Archives of Neurology, 43, 574-576.

GASSER T., BACHER P. AND STEINBERG H. (1985). Test-retest reliability of spectral parameters of the EEG. Electroencephalography and Clinical Neurophysiology, 60, 312-319.

GEREBY G. (1980). Electroencephalographic and neuropathological correlations in dementias. In Lechner H. and Aranikar A. (Eds.), EEG and Clinical Neurophysiology (pp 553-559), Amsterdam, Excerpta Medica.

GIAQUINTO S. AND NOLFE G. (1986). The EEG in the normal elderly: A contribution to the interpretation of ageing and dementia. Electroencephalography and Clinical Neurophysiology, 63, 540-546.

HARDING G.F.A., WRIGHT C.E. AND ORWIN A. (1985). Primary presenile dementia: The use of the visual evoked potential as a diagnostic indicator. British Journal of Psychiatry, 147, 532-539.

HUBBARD O., SUNDE D. AND GOLDENSOHN E.S. (1976). The EEG in centenarians. Electroencephalography and Clinical Neurophysiology, 40, 407-417.

JASPER H.H. (1958). The ten-twenty electrode system of the International Federation. Electroencephalography and Clinical Neurophysiology, 10, 371-375.

JOYNT R.J. AND SHOULSON I. (1985). Dementia. In Heilman K. M. and Valenstein E. (Eds.), Clinical Neuropsychology (pp 453-479). New York, Oxford University Press.

KACHATURIAN Z. S. (1985). Diagnosis of Alzheimer's Disease. Archives of Neurology, 42, 1097-1104.

KAUFMAN D. M. (1985). Clinical Neurology for Psychiatrists. Orlando, Grune & Stratton Inc.

LAI J.A. (1985). P3A influences on P300 differential diagnosis in geriatric clinical syndromes. Dissertation Abstracts International, 46, 2069-B.

LEVINSON B., WRIGHT P. AND BARKLEM S. (1985). Effect of Buflomedil on behaviour, memory, and intellectual capacity in patients with dementia. South African Medical Journal, 68, 302-307.

MARKAND O.N. (1986). Electroencephalogram in dementia. American Journal of EEG Technology, 26, 3-17.

MOSTERT W. P. AND VAN TONDER J. L. (1982). Moonlike bevolkingsgroei in Suid-Afrika tot die middel van die 22e eeu (Possible population growth in South Africa until the middle of the 22nd century). Pretoria: HSRC (Report S-83).

MUNDY-CASTLE A.C., HURST L.A., BEERSTECHER D.M. AND PRINSLOO T. (1954). The electroencephalogram in the senile psychoses. Electroencephalography and Clinical Neurophysiology, 6, 245-252.

MUNDY-CASTLE A.C. (1962). Central excitability in the aged. In Blumenthal H.T.(Ed.), Medical and clinical aspects of aging (pp 575-595). New York, Coumbia press.

MURDOCH B. D. (1981). The pattern reversal evoked potential in multiple sclerosis. Johannesburg, CSIR (Special report PERS 332).

MURDOCH B. D. (1985). The long-term effect of medication on epileptic patients. Johannesburg, HSRC (Research finding PERS 389).

NATIONAL INSTITUTE ON AGING. (1980). Changes...research on aging and the aged. NIH Publication no. 81-85.

OBRIST W.D. AND HENRY C.E. (1958). Electroencephalographic findings in aged psychiatric patients. Journal of Nervous and Mental Disease, 126, 254-267.

OTOMO E. (1966). Electroencephalography in old age: Dominant alpha pattern. Electroencephalography and Clinical Neurophysiology, 21, 489-491.

PENTTILA M., PARTANEN J.V., SOININEN H. AND RIEKKINEN P.J. (1985). Quantitative analysis of occipital EEG in different stages of Alzheimer's disease. Electroencephalography and Clinical Neurophysiology, 60, 1-6.

PFEFFERBAUM A., WENEGRAT B.G., FORD J.M., ROTH W.T. AND KOPELL B.S. (1984). Clinical application of the P300 component of event-related potentials . II Dementia, depression and schizophrenia. Electroencephalography and Clinical Neurophysiology, 59, 104-124.

POLISH J., EHLERS C.L., OTIS S., MANDELL A.J. AND BLOOM F.E. (1986). P300 latency reflects the degree of cognitive decline in dementing illness. Electroencephalography and Clinical Neurophysiology, 63, 138-144.

ROUBICEK J. (1977). The electroencephalogram in the middle-aged and the elderly. Journal of the American Geriatric Society, 25, 145-152.

SCHOENBERG B. S., ANDERSON D.W. AND HAERER A.F. (1985). Severe dementia: Prevalence and clinical features in a biracial population. Archives of Neurology, 42, 740-743.

SCIARRETTA G. AND ERCULANI P. (1978). A proposal for calibrating EEG spectrograms. Electroencephalography and Clinical Neurophysiology, 45, 674-676.

SLEATS J.P.J. AND FORTGENS C. (1984). On the value of P300 event-related potentials in the differential diagnosis of dementia. British Journal of Psychiatry, 145, 652-656.

ST CLAIR D.M., BLACKWOOD D.H.R. AND CHRISTIE J.E. (1985). P3 and other long latency auditory evoked potentials in presenile dementia Alzheimer type and alcoholic Korsakoff syndrome. British Journal of Psychiatry, 147, 702-706.

TOLLMAN S.G. (1985). Neuropsychology and the ageing process. In Grieve K.W. and Griesel R.D. (Eds.), Neuropsychology II: Proceedings of the second South African neuropsychology conference Pretoria 1985 (pp 211-220). Pretoria, University of South Africa.

TREVES T., KORCZYN A.D., ZILBER N., KAHANA E., LEIBOWITZ Y., ALTER M. AND SCHOENBERG B.S. (1986). Presenile dementia in Israel. Archives of Neurology, 43, 26-29.

VISSER S.L., VAN TILBURG W., HOOIJER C., JONKER C. AND DE RIJKE W. (1985). Visual evoked potentials (VEPs) in senile dementia (Alzheimer type) and in non-organic behavioural disorders in the elderly: Comparison with EEG parameters. Electroencephalography and Clinical Neurophysiology, 60, 115-121.

WRIGHT C.E. AND HARDING G.F.A. (1985). Visual evoked potentials in normal aging and dementia. Electroencephalography and Clinical Neurophysiology, 61, S46.

WEINREB H.J. (1985). Fingerprint patterns in Alzheimer's disease. Archives of Neurology, 42, 50-54.

HSRC PUBLICATION LIST

A complete list of HSRC publications or a list of publications of a particular Institute of the HSRC is available from the Head: Technical Services

Doc no 5657
Copy no. 154110

RGN BIBLIOTEEK | HSRC LIBRARY

HUMAN SCIENCES RESEARCH COUNCIL
RAAD VIR GEESTESWETENSKAPLIKE NAVORSING

President	Dr J.G. Garbers	President
Deputy Presidents	Dr H.C. Marais, Dr J.D. Venter	Adjunk-presidente
Vice-Presidents	Dr K.F. Mauer, Prof. D.J. Stoker	Vise-presidente
Executive Director: Administration	J.G.G. Gräbe	Uitvoerende Direkteur: Administrasie
Chief PRO	Dr G. Puth	Skakelhoof

Functions of the HSRC

The HSRC undertakes, promotes, supports and co-ordinates research in the field of the human sciences. It also determines research priorities, disseminates the findings of human sciences research, facilitates and evaluates the implementation of research findings, stimulates the training of researchers, places the full spectrum of human sciences disciplines at the service of the inhabitants of the RSA and promotes science in general.

Institutes

Institute for Communication Research (ICOMM)

Institute for Educational Research (IER)

Institute for Historical Research (IHR)

Institute for Manpower Research (IMAN)

National Institute for Personnel Research (NIPR)

Institute for Psychological and Edumetric Research (IPER)

Institute for Research Development (IRD)

Institute for Research into Language and The Arts (IRLA)

Institute for Sociological and Demographic Research (ISODEM)

Institute for Statistical Research (ISR)

Bureau for Research Support Services (BRSS)

Administration

Head office

Private Bag X41, Pretoria 0001
Republic of South Africa
Telegrams RAGEN
Tel. (012) 202-9111
Telex 3-20893 SA

NIPR

P.O. Box 32410, Braamfontein 2017
Republic of South Africa
Telegrams NAVORSPERS
Tel. (011) 339-4451
Telex 4-25459 SA

Regional offices

Western Cape, Private Bag X5, Roggebaai 8012
Tel. (021) 419-2572/3/4/5 Telex 5-22260 SA

Natal, P.O. Box 508, Durban 4000
Tel. (031) 31-6926 Telex 6-28567 SA

NIPR Natal, P.O. Box 17302, Congella 4013
Tel. (031) 815970 Telex 6-28567 SA

NIPR Eastern Cape, P.O. Box 1124, Port Elizabeth 6000
Tel. (041) 53-2131 Telex 2-43203 SA

Funksies van die RGN

Die RGN onderneem, bevorder, ondersteun en koördineer navorsing op die gebied van die geesteswetenskappe, bepaal navorsingsprioriteite, versprei die resultate van geesteswetenskaplike navorsing, vergemaklik en evalueer die implementering van die resultate van navorsing, stimuleer die opleiding van navorsers, stel die volle spektrum van dissiplines in die geesteswetenskappe ten diens van die inwoners van die RSA en bevorder die wetenskap in die breë.

Institute

Instituut vir Geskiedenisnavorsing (IGN)

Instituut vir Kommunikasienavorsing (IKOMM)

Instituut vir Mannekragnavorsing (IMAN)

Instituut vir Navorsingsontwikkeling (INO)

Instituut vir Opvoedkundige Navorsing (ION)

Nasionale Instituut vir Personeelnavorsing (NIPN)

Instituut vir Psigologiese en Edumetriese Navorsing (IPEN)

Instituut vir Sosiologiese en Demografiese Navorsing (ISODEM)

Instituut vir Statistiese Navorsing (ISN)

Instituut vir Taal- en Kunstenavorsing (INTAK)

Buro vir Ondersteunende Navorsingsdienste (BOND)

Administrasie

Hoofkantoor

Privaatsak X41, Pretoria 0001
Republiek van Suid-Afrika
Telegramme RAGEN
Tel. (012) 202-9111
Teleks 3-20893 SA

NIPN

Posbus 32410, Braamfontein 2017
Republiek van Suid-Afrika
Telegramme NAVORSPERS
Tel. (011) 339-4451
Teleks 4-25459 SA

Streekkantore

Wes-Kaap, Privaatsak X5, Roggebaai 8012
Tel. (021) 419-2572/3/4/5 Teleks 5-22260 SA

Natal, Posbus 508, Durban 4000
Tel. (031) 31-6926 Teleks 6-28567 SA

NIPN Natal, Posbus 17302, Congella 4013
Tel. (031) 815970 Teleks 6-28567 SA

NIPN Oos-Kaap, Posbus 1124, Port Elizabeth 6000
Tel. (041) 53-2131 Teleks 2-43203 SA

ISBN 0 7969 0491 X

697/87