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THE EFFECTS OF EARLY AND PROLONGED MATERNAL ANTENATAL DECOMPRESSION ON THE ELECTROENCEPHALOGRAM OF FOUR-YEAR-OLD CHILDREN

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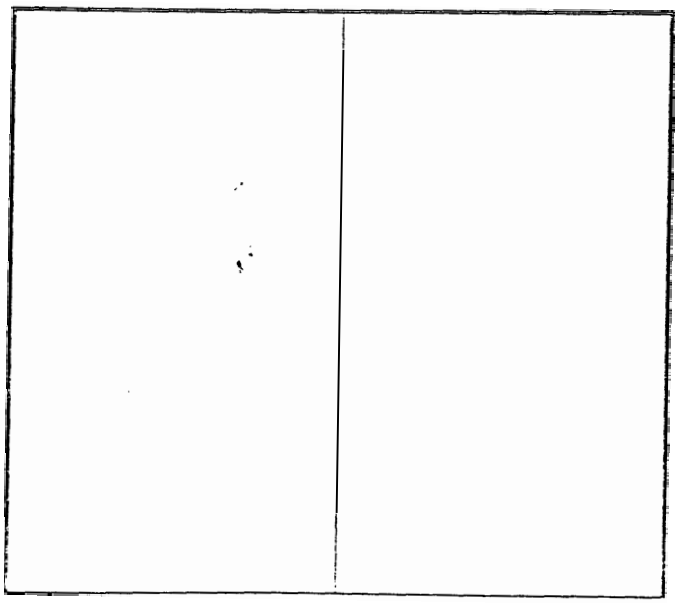

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B D MURDOCH

SUMMARY

Electroencephalograms (EEGs) were recorded from 44 four-year-old first-born children whose mothers had commenced decompression treatment before the 29th week of pregnancy and who had undergone a mean number of 148 treatments thereafter, and from 48 age-matched controls whose mothers had received no decompression during pregnancy. A comparison of the groups on 45 EEG variables revealed that only seven differentiated significantly between them at less than the 0,05 level. This is less than the number of variables which on a chance basis might have been expected to differentiate between the groups. Therefore it is suggested that the groups show no basic EEG differences, and that early and intensive decompression treatment produces no lasting EEG effects. A further detailed analysis of the significantly differentiating variables in relation to other group measures fails to support Heyns's contention that antenatal decompression leads to superior cognitive function and enhanced cortical development in children. It is possible that factors other than early and prolonged decompression treatment were involved in the production of the present results. However, due to the rigid control procedures necessary to check on the operation of such factors, further research into the effects of antenatal decompression would need to utilize animal subjects.

OPSOMMING

Die elektroenkefalogram (EEG) by 44 vierjarige eersgeborene proefpersone wie se moeders dekompressie-behandeling voor die 29ste week van swangerskap begin het en dit vir 'n gemiddelde aantal van 148 behandelinge daarna ondergaan het, en van 40 kontrole-groep kinders van dieselfde ouderdom wie se moeders geen dekompressie-behandeling ontvang het nie, is afgeneem. Die groepe is met mekaar in terme van 48 EEG-metings vergelyk maar by net sewe daarvan is verskille teen minder as die 0,05 beduidendheidsvlak tussen die groepe aangedui. Die aantal is minder as wat per toeval verwag sou word om tussen die groepe te onderskei. Dit word dus voorgestel dat geen basiese EEG-verskille tussen die groepe bestaan het nie en ook dat vroeë en intensiewe dekompressie-behandeling geen permanente EEG-veranderings meebring nie. 'n Nadere ontleding van die beduidende groepsverskille ten opsigte van die oorblywende tellings het geen ondersteuning vir Heyns se hipotesis van 'n verwantskap tussen voorgeboortelike dekompressie en superieure kognitiewe funksie en bo-normale kortikale ontwikkeling in kinders nie. Verder blyk dit dat vroeë en intensiewe dekompressie-behandeling nie die enigste veranderlike is wat vir die huidige resultate verantwoordelik was nie. Weens die noodsaaklikheid vir strenge kontrole om die inmenging van sulke faktore op te spoor word dit aan die hand gegee dat daar by verdere navorsing oor die uitwerking van voorgeboortelike dekompressie, gebruik gemaak word van diere.

INTRODUCTION

Previous attempts utilizing the electroencephalogram (EEG) as an index of developmental status to establish the validity of the claim (Heyns, 1963a[1], b[2]) that prenatal maternal decompression permits realization of the full developmental potential of the foetus, and, subsequently, the child, have failed. No consistent EEG differences between the children of non-decompressed mothers and children whose mothers had received twelve half-hour decompression sessions during the last two months of pregnancy (Nelson, 1961[3]) or between ten and thirty half-hour treatments during the last ten weeks of pregnancy (Murdoch, 1968[4]), were seen. The rationale for the use of the EEG as an index of cerebral maturation has been given elsewhere (Murdoch, 1973[5]) and it would appear to be potentially the most objective indicator of the effects of decompression currently available.

It had been previously suggested (Murdoch, 1968[6], 1973[7]) that measurable EEG effects might be produced by the earlier initiation and more intensive utilization of decompression during pregnancy. When children whose mothers had commenced decompression before the 29th week of pregnancy and who had utilized this treatment intensively thereafter were offered for EEG testing, it was therefore decided to initiate the study which is reported below. EEG characteristics of this decompression group were compared with those of an age-matched control group whose mothers had not received decompression during pregnancy.

SUBJECTS

The experimental (decompression) group comprised 44 children ranging in age from 47 months to 61 months (mean 53,48 ; SD 4,47). There were 16 males and 28 females in the group. The mothers of the children had had access at home to decompression apparatus. Decompression was initiated between the 23rd and 29th weeks of pregnancy, with mothers being instructed to apply treatment for half-hour periods daily from the time of initiation and twice daily during the last trimester of pregnancy. The total number of individual treatments ranged from 77 to 224 (mean 148,36 ; SD 39,05).

The control (non-decompression) group consisted of 40 children aged between 45,5 months and 61 months (mean 53,44 ; SD 4,19). Males numbered 25 and females 15. Control subjects were playmates of those in the decompression group, and were thus presumably reasonably well-matched in terms of variables related to socio-economic status. No mother of a child in the control group had undergone decompression treatment during pregnancy, although the incidence of those who had received physiotherapy or other specialized antenatal treatment was not ascertained.

The mothers of control and experimental children were matched on a number of variables which could have influenced congenital development, and hence possibly the EEG status of each group when tested. Control and experimental group mothers, therefore, did not differ significantly in terms of age, educational status, incidence of smoking during pregnancy, incidence of threatened miscarriage and of antepartum bleeding. The two groups were also matched for paternal educational status, weight at delivery, method of delivery, problems during the neonatal period, childhood illnesses and attendance at nursery school. All subjects were firstborns.

PROCEDURE

1. EEG Recording

EEGs were recorded in the laboratories of the Division of Neuro-psychology, NIPR. The experimental subject, accompanied by his control was brought to the NIPR, usually by the mother of the former. Control and experimental subjects were left to choose who would first have the EEG, and at no time were the recording personnel aware of

the group designation of the subject. Mothers were present while the recording was carried out if a balming influence was felt to be necessary. Both experimental and control subjects had been prepared for the recording situation beforehand, and most co-operated (only two subjects of the original groups selected refused the EEG).

Recordings were made on a Galileo E8b electroencephalograph using five different bipolar montages of electrodes positioned according to the ten-twenty system (Jasper, 1958[8]). Photic stimulation, consisting of a series of increasing and decreasing repetitive light flashes between one and 100 in frequency, was applied to all subjects except one decompression child. Standardized hyperventilation for three minutes was not performed, or performed inadequately, by only four decompression and six control subjects.

2. EEG Analysis

All EEGs were analysed by the same electroencephalographer by eye with the aid of a millimetre rule. At no time during the analysis was the electroencephalographer aware of the group designation of the EEG.

As an increase in frequency of the alpha rhythm relates predominantly to maturational processes (Petersén and Eeg-Olofsson, 1971[9]), particular attention was given in the analysis of the EEGs to the parameters of alpha activity. Accordingly, four alpha frequency and amplitude measurements were made from each of the derivations $T_4 - T_6$; $T_6 - O_2$; $C_4 - P_4$; $P_4 - O_2$; $T_3 - T_5$; $T_5 - O_1$; $C_3 - P_3$ and $P_3 - O_1$ (Jasper, 1958[10]). In addition, mean frequencies and amplitudes for left and right hemispheres were calculated by averaging the measurements from the relevant derivations. In some cases, mean left and right hemisphere scores were based on less than the total number of measurements indicated previously due to intermittent contamination of some EEG channels by electromyographic or movement potentials or the failure of apparatus channels. Other measurements of alpha activity derived were : number of alpha components (the number of different frequency components present within the 8 - 13 Hz band) and the alpha frequency range (the difference between the highest and lowest frequencies present in the 8 - 13 Hz band). Mean delta, theta and

beta frequencies were obtained by averaging, respectively, the frequencies present in the <4 Hz, 4 - 7 Hz and >13 Hz bands in each EEG. Similarly, mean delta, theta and beta amplitudes for left and right hemispheres were obtained by averaging the amplitudes of waves in each band. In effect, measurements relating to beta activity were discarded from further analysis as this type of activity was found in only one decompression subject, and only two control children. The incidences (indexes) of delta, theta and alpha activity in each record were rated. The relative indexes of the bands summated gave a total of 10.

EEGs during photic stimulation were rated in terms of degree of responsiveness to the flashing light on a four point scale ranging from zero (no response) to 3 (marked response). In addition, the presence or absence of harmonic and subharmonic responses, and the number of flash frequencies responded to, were ascertained. The degree of response to hyperventilation was assessed on a four-point scale (0 indicating no response and 3 a marked response), and the time taken to return to resting levels after hyperventilation was measured in seconds.

Abnormality ratings during rest, photic stimulation and hyperventilation were also made on four-point scales, with zero indicating a normal, and three a severely abnormal EEG or response. Judgements of abnormality were made in terms of focal (that is, activity appearing unilaterally in a specific cortical area and usually indicated by phase reversed potentials), paroxysmal (sudden changes in activity greater than 50% of the mean preceding background activity), sharp wave (sharp-tipped activity with a period of between 0,125 and 0,077 seconds), spike (with a period less than 0,077 seconds) and wave and spike activity. EEGs were not considered abnormal solely on the basis of slow activity unless this was localized or extreme, due to the dubious significance of slow activity in the EEGs of children (Petersén and Eeg-Olofsson, 1971[11]). However, EEGs were classified into 'supernormal', 'normal', 'slight increase in low frequency activity' and 'moderate increase in low frequency activity' categories on the basis of Petersén and Eeg-Olofsson's (1971[12]) criteria. At the present age these were applied as follows : supernormal - less than 10% slow

activity ; normal - 11 - 30% slow activity ; slight increase in low frequency activity - 31 - 50% slow activity ; moderate increase in low frequency activity - more than 50% slow activity.

3. Statistical Treatment

The measurements of alpha, theta and delta activity, responsivity during photic stimulation and hyperventilation, recovery after hyperventilation and ratings of abnormality during rest, photic stimulation and hyperventilation were all made on continuous scales. The t test (Guilford, 1956, p. 220[13]) was used to compare decompression and control group means. All other measurements were scored discretely, and chi-square, incorporating Yate's correction for continuity where necessary, (Guilford, 1956, p. 230, 234[14]) was used to assess the significance of group differences in incidence of these variables. Pearson's product-moment coefficient of correlation (Guilford, 1956, p. 138[15]) was used to assess the relationship between number of decompression runs and selected EEG variables in the experimental group. All calculations were made on a Hewlett-Packard 9100 B desk computer.

Group differences were regarded as significant if at the 0,05 level or less.

RESULTS

Results are presented in Tables 1 to 10

TABLE 1

Frequency measures for decompression and control groups

Derivation or Frequency Band	EXPERIMENTAL			CONTROL			t	N
	Range	Mean	SD	Range	Mean	SD		
C_4-P_4	8,0-9,75	8,47	0,47	8,0-10,0	8,70	0,64	1,53	53
P_4-O_2	8,0-9,5	8,42	0,45	8,0-10,88	8,70	0,76	2,04*	82
T_4-T_6	8,0-8,88	8,29	0,28	8,0-10,25	8,61	0,71	2,31*	60
T_6-O_2	8,0-9,63	8,45	0,47	8,0-10,5	8,69	0,65	1,95	79
Mean α Right Hemisphere	8,0-9,38	8,43	0,39	8,0-10,34	8,67	0,61	2,15*	83
C_3-P_3	8,0-9,5	8,48	0,48	8,0-10,75	8,64	0,70	1,18	73
P_3-O_1	8,0-9,75	8,38	0,41	8,0-10,25	8,64	0,69	2,09*	81
T_3-T_5	8,0-9,75	8,45	0,46	8,0-10,75	8,58	0,68	0,92	65
T_5-O_1	8,0-9,5	8,45	0,44	8,0-10,5	8,69	0,68	1,94	80
Mean α Left Hemisphere	8,0-9,34	8,43	0,36	8,0-10,56	8,65	0,63	1,89	83
Mean Theta	4,5-6,5	5,48	0,43	4,0-7,0	5,39	0,50	0,91	81
Mean Delta	2,0-3,5	2,64	0,36	2,0-3,75	2,73	0,48	0,48	24

* $p < 0,05$

TABLE 2

Amplitude measures for decompression and control groups

Derivation or Frequency Band	EXPERIMENTAL			CONTROL			t	N
	Range	Mean	SD	Range	Mean	SD		
C_4-P_4	12,5-32,50	21,72	4,85	12,5-35,0	22,08	5,32	0,26	53
P_4-O_2	17,5-56,25	30,26	8,70	17,5-60,0	31,28	8,71	0,53	82
T_4-T_6	15,0-43,75	26,46	6,97	15,0-47,50	27,33	8,49	0,44	60
T_6-O_2	20,0-48,75	30,21	7,15	16,25-75,0	32,40	10,64	1,09	79
Mean α Right	18,75-43,13	27,36	5,50	17,5-54,16	28,68	7,18	0,95	83
C_3-P_3	13,75-33,75	22,97	5,36	16,25-38,75	24,92	5,7	1,51	73
P_3-O_1	18,75-61,25	34,83	10,50	18,75-50,0	34,53	8,45	0,14	81
T_3-T_5	16,25-43,75	25,54	6,01	17,5-43,75	27,54	6,68	1,27	65
T_5-O_1	17,5-66,25	35,95	11,22	17,5-52,5	34,26	8,55	0,63	80
Mean α Left Hemisphere	18,13-50,63	30,26	7,64	19,38-40,83	30,3	6,21	0,03	83
Mean θ Right Hemisphere	20,0-92,5	55,32	17,35	20,0-105,0	56,29	17,07	0,25	79
Mean θ Left Hemisphere	25,0-113,33	60,69	19,02	25,0-97,5	59,73	13,97	0,26	81
Mean δ Right Hemisphere	35,0-150,0	86,58	31,26	40,0-215,0	116,57	54,28	1,50	19
Mean δ Left Hemisphere	30,0-170,0	107,12	39,78	90,0-180,0	119,54	29,54	0,79	22

No group mean difference approaches statistical significance.

TABLE 3

Alpha, theta and delta indexes for decompression and control groups

Index	EXPERIMENTAL			CONTROL			t	N
	Range	Mean	SD	Range	Mean	SD		
Alpha	3 - 10	5,60	1,95	0 - 10	5,56	2,29	0,09	84
Theta	0 - 7	4,24	1,86	0 - 9	4,03	2,22	0,48	84
Delta	0 - 2	0,16	0,43	0 - 2	0,23	0,53	0,63	84

TABLE 4

Experimental and control group responses to activation procedures

Procedure	Variable	EXPERIMENTAL			CONTROL			t	N
		Range	Mean	SD	Range	Mean	SD		
Photic Stimulation	Number of flash freq. evoking resp.	0-6	2,47	1,47	0-5	2,05	1,24	1,39	83
Photic Stimulation	Responsiveness (0 - 3)	0-2,5	1,07	0,68	0-2,5	1,03	0,53	0,33	83
Hyperventilation	Recovery (secs)	0-50	16,08	11,62	0-40	17,79	11,27	0,63	72
Hyperventilation	Responsiveness (0 - 3)	0-2	1,13	0,52	0-2,5	1,14	0,62	0,06	72

TABLE 5

Mean abnormality ratings for experimental and control groups

Condition	EXPERIMENTAL			CONTROL			t	N
	Range	Mean	SD	Range	Mean	SD		
Rest	0 - 2	0,50	0,67	0 - 1,5	0,20	0,42	2,44*	84
Photic Stimulation	0 - 2	0,12	0,39	0 - 1	0,05	0,22	0,94	83
Hyperventilation	0 - 2	0,35	0,57	0 - 3	0,28	0,62	0,50	78

* $p < 0,05$

TABLE 6

Alpha frequency range and number of alpha components for experimental and control groups

Variable	EXPERIMENTAL			CONTROL			t	N
	Range	Mean	SD	Range	Mean	SD		
α frequency range	0 - 3	1,60	0,75	0,5 - 4	1,88	0,94	1,03	46
Number of α components	1 - 6	3,56	1,16	2 - 6	4,29	1,23	2,06*	46

* $p < 0,05$

TABLE 7

Incidence of harmonics and subharmonics during photic stimulation

	HARMONICS		SUBHARMONICS	
	EXPERIMENTAL	CONTROL	EXPERIMENTAL	CONTROL
Present	4	7	10	11
Absent	39	33	33	29
Chi-Square	0,60		0,20	
df	1		1	

TABLE 8

Incidence of supernormal and normal EEGs, and those with slight and moderate increases in low frequency activity in experimental and control groups

CATEGORY	EXPERIMENTAL	CONTROL	
Supernormal	2	3	Chi-square
Normal	6	6	0,39 ; df 3
Slight increase in low frequency activity	23	20	
Moderate increase in low frequency activity	13	11	

TABLE 9

Incidence of types of abnormalities in experimental and control groups

	SHARP WAVE OR SPIKE ACTIVITY		FOCAL OR LOCALISED SLOW ACTIVITY		PAROXYSMAL ACTIVITY		WAVE AND SPIKE (LARVAL) ACTIVITY	
	Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
Present	12	3	17	9	4	1	4	1
Absent	32	37	27	31	40	39	40	39
Chi-Square	4,32		2,55		0,66		0,66	
df	1		1		1		1	
p	$< 0,05$		NS		NS		NS	

TABLE 10

Correlations between number of decompression runs and EEG variables in experimental group

		r	N
Number of Decompression Runs	P_4-O_2 Frequency	0,042	44
"	T_4-T_6 Frequency	0,071	30
"	Mean alpha Frequency Right	-0,002	44
"	P_3-O_1 Frequency	0,197	44
"	Mean alpha Frequency Left	0,090	44
"	Mean Theta Frequency	0,148	42
"	Mean Delta Frequency	-0,110	14
"	Alpha Frequency Range	0,237	25
"	Number of Alpha Components	0,322	25
"	Alpha Index	0,217	44
"	Theta Index	-0,246	44
"	Rest Abnormality Rating	-0,092	44

DISCUSSION

Experimental and control group differences in means or incidences were compared in respect of 45 EEG measures. Seven attained significance at less than the 0,05 level, an incidence which is less than the nine expected to reach significance for this number of variables on the basis of chance alone. This factor, together with the markedly similar group profiles on the remaining 38 measures, suggest the lack of any basic differences between experimental and control group subjects. This, in turn, suggests that early and extended antenatal decompression confers no lasting effects measurable in the EEG, a conclusion also previously advanced by Nelson (1961[16]) and by Murdoch (1968[17]) in relation to the effects of decompression applied at a more advanced stage of, and less intensively utilised during, pregnancy.

The statistically significant group differences (Tables 1, 5, 6 and 9) will be considered in detail and in relation to other differences between the groups to ascertain whether they contradict the above conclusion.

1. EEG criteria of maturation (Tables 1, 3, 8)

As increases in frequency, particularly those of the alpha rhythm, bear a direct and well-documented relationship with increasing age (see, for example, Petersén and Eeg-Olofsson, 1971, p. 264[18]), it is possible that consistent and significant differences between the groups on a number of frequency measures may be indicative of an enhanced cortical maturation. This, in turn, would provide a measure of support for Heyns's (1963a[19], b[20]) claims of the lasting benefits of antenatal decompression resulting from improved foetal cortical physiology. Table 1 indicates that the alpha frequencies measured from all derivations, as well as the means calculated for right and left hemispheres, were consistently slightly (0,13 - 0,32 Hz) and in four cases significantly ($p < 0,05$) faster in the control than in the experimental group. This is the antithesis of what might have been predicted if Heyns's (1963a[21], b[22]) contentions had been valid and negates any possible relationship between prenatal maternal decompression and advanced EEG maturity. Since the two groups were matched on most of the familial, congenital, peri- and post-natal

variables which might have influenced EEG development except that of prenatal decompression, the possibility arises that the latter treatment may be responsible for the depressed alpha frequency measures of the experimental group. A parallel has been drawn (Charlewood, 1973[23]) between the present experimental results and the high incidence of EEG abnormalities associated with the condition of retrolental fibroplasia (Boshes et al., 1967[24]), which apparently is the result of damage to developing retinal blood vessels from hyperoxygenation during the incubation of prematurely delivered infants. Such a parallel can only be regarded as extremely tenuous for the following reasons. Firstly neuroanatomical differences undoubtedly exist between retinal and cortical cells. It would appear illogical to assert that the reaction to hyperoxygenation of the latter must be as that of the former, at least until experimental evidence points otherwise. Next, it is inadvisable to generalize from results based on a sample of premature infants, who are known to have a high incidence of prenatal complications, to a normal sample. There further exists the possibility of a relationship between the defective or absent vision resulting from retinal damage in retrolental fibroplasia and cortical and, particularly, occipital EEG abnormalities. Finally, the significance of possible cerebral and other CNS changes associated with retrolental fibroplasia is by no means defined (Boshes et al., 1967[25]).

Experimental and control group alpha frequency differences may also be attributed, at least in part, to differences in the sex composition of each group : females comprised 37% of the control and 63% of the decompression group. Petersén and Eeg-Olofsson (1971[26]), have shown faster alpha frequencies in girls than in boys until puberty. Further, although all alpha derivations showed frequency differences in favour of the control group, all, in any one individual were the product of the same alpha-generating system, and the consistency of the results in the individual case is therefore not remarkable.

It has been argued to this point that the alpha frequency differences between control and experimental groups are : antithetical to Heyns's (1963a[27], b[28]) position ; not readily attributable to any possibly deleterious effects of antenatal decompression on EEG development in

the experimental group ; and, finally, may be partly the result of factors other than antenatal decompression. All of these arguments are compatible with the proposition that early and intensive antenatal decompression produces no lasting EEG changes. Further support for this statement may be found in a consideration of group differences in indices related to EEG maturation other than alpha frequency measures. Differences between experimental and control groups in mean theta and delta frequencies (Table 1), in alpha, theta and delta amplitudes (Table 2), in relative proportions of alpha, theta and delta activity (Table 3) and in incidences of supernormal and normal EEGs, and of those containing slight and moderate increases in low frequency activity (Table 8) all fail to reach significance at the 0,05 level.

2. Measures of alpha variation (Table 6)

A relationship between lack of variation in alpha activity as measured by the range of alpha frequencies and number of alpha components present in the EEG and the symptom constellation of minimal cerebral dysfunction (Murdoch, 1974[29]) has been advanced as tentative support for Walter's (1953[30]) contention that increased alpha variation is associated with superior intelligence. Significant differences between control and experimental groups suggesting greater alpha variation in the latter might therefore have been regarded as support for Heyns's (1963a[31], b[32]) statements regarding the superior cognitive ability of decompression children. However, only one of the measures of alpha variation reaches significance at the 0,05 level, and this shows more alpha components in the control group. This is, again, antithetical to Heyns's position.

3. Measures of EEG abnormality (Tables 5, 9)

Table 5 indicates that the experimental group had a significantly higher mean resting abnormality rating (0,5) in relation to the control group (0,2). This result may again be interpreted as providing evidence for a deleterious effect of antenatal decompression on the EEG, in terms of the rationale previously discussed. However, such an interpretation must further be assessed in the light of the results obtained in a previous investigation (Murdoch, 1968[33]) which indicated a higher incidence of

abnormality in the resting EEG in the control group in comparison with the decompression group. The presently obtained results may, of course, be ascribed to the earlier and more intensive decompression treatment than in the former study, but the contradictions between the two investigations in relation to resting abnormality could only be resolved by further experimentation. The utilisation of animal subjects in further research in view of the possibly adverse EEG effects of early and prolonged decompression treatment would appear indicated. It is of interest that both the previous (Murdoch, 1968[34]) and present investigations obtained no significant control and decompression group differences in abnormality of response to photic stimulation or hyperventilation. This accords, in the present study, with a general control and experimental group similarity in response to activation procedures (Table 4).

The decompression group shows a significantly ($p < 0,05$) higher incidence of sharp wave and spike activity during rest and in response to activation procedures than the control group. This is the only type of abnormal activity where group incidences show significant differences (Table 9). In most cases this activity was localized, suggesting a higher incidence of localized cerebral instabilities in the experimental than in the control group. Again a connection between this feature and decompression treatment is possible, but ultimate resolution of this question would have to await further research.

Experimental and control groups were matched on a number of variables which might be expected to produce lasting effects on the EEG. Theoretically the groups only differed in terms of the controlled variable of the early and prolonged decompression applied to experimental group mothers. That this was attained only to a limited extent has already been shown in relation to the sex composition of control and experimental groups. It can further be shown that other uncontrolled variables may, at least, be partly responsible for the results obtained by correlating the number of decompression treatments the mothers received with selected EEG measures in their children, including those which revealed statistically significant differences between the groups. If decompression treatment was the only factor responsible for group differences, marked and significant correlations between number of

treatments and those measures at least, on which the groups achieved significantly different scores should be obtained. Table 10 shows that all coefficients calculated were small and of apparently irrelevant direction and in no case approached the 0,05 level of significance.

CONCLUSIONS

1. The weight of evidence suggests that early and intensive decompression treatment in the mother produces no lasting changes in the EEGs of four-year-old children.

2. The majority of the statistically significant differences between decompression and control groups are antithetic to Heyns's contention (1963a[35], b[36]) that antenatal decompression produces enhanced cortical function and superior cognitive ability.

3. Although a number of statistically significant differences between control and experimental groups suggest possibly deleterious EEG effects of early and prolonged decompression treatment, it is highly unlikely that the decompression treatment is the only factor involved in producing these results. The relationship between this treatment and EEG changes could only be established by further experimentation, utilising animal subjects where more rigid control procedures are possible.

REFERENCES

	Order of Appearance
BOSHES, L.D., COHEN, J., ALFANO, J.E. and W-H. CHAO LEE (1967) Longitudinal appraisal of school-age children with retrolental fibroplasia. Dis. nerv. Sys., 28, 203-230.	24, 25
CHARLEWOOD, C.A.H. (1973) The Effects of Early and Extended Antenatal Abdominal Decompression on the Brain Function of the Child. MA Dissertation, Pretoria, University of South Africa.	23
GUILFORD, J.P. (1956) Fundamental Statistics in Psychology and Education. New York, McGraw-Hill.	13, 14, 15
HEYNS, O.S. (1963a) Abdominal Decompression - a Monograph. Johannesburg, Witwatersrand University Press.	1, 19, 21, 27, 31, 35
HEYNS, O.S. (1963b) How does abdominal decompression help the foetal C.N.S.? Leech, 33, 98-100.	2, 20, 22, 28, 32, 36
JASPER, H.H. (1958) The ten-twenty electrode system of the international federation. Electroenceph. clin. Neurophysiol., 10, 371-375.	8, 10
MURDOCH, B.D. (1968) The effects of prenatal maternal decompression on the electroencephalographic development of 3-year-old children. S.A. Med.J., 42, 1067-1071.	4, 6, 17, 33, 34
MURDOCH, B.D. (1973) The Effects of Prenatal Maternal Decompression on the Electrocortical Development of Three-year-old Children. MA Dissertation, Johannesburg, University of the Witwatersrand.	5, 7
MURDOCH, B.D. (1974) Changes in the electroencephalogram in minimal cerebral dysfunction : a controlled study over eight months. S.A. Med. J., 48, 606-610.	29
NELSON, G.K. (1961) Electroencephalograms of infants in relation to pre-natal maternal decompression. Confidential report. Johannesburg, NIPR.	3, 16

	Order of Appearance
PETERSÉN, I. and O. EEG-OLOFSSON (1971) The development of the electroencephalogram in normal children from the age of 1 through 15 years. Neuropadiätrie, 2, 247-304.	9, 11, 12, 18, 26
WALTER, W.G. (1953) The Living Brain. Harmondsworth, Penguin.	30