Visual Evoked Response in Children Subjected to Prenatal Maternal Decompression

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SUMMARY

The visual evoked response was studied in 41 children whose mothers had undergone antenatal decompression treatment, and in a closely-matched control group. The results showed no consistent statistically significant differences between the groups on a variety of latency and amplitude measures of the evoked response. It is concluded that neither group enjoyed any advantage in terms of maturation of the central nervous system, speed of neural conduction, or arousal level.

S. Afr. Med. J., **48**, 2591 (1974).

Prenatal maternal decompression has been pioneered in South Africa as a technique which facilitates not only labour but also fetal oxygenation[1-4] and which alleviates fetal distress[5] and the effects on the fetus of toxaemia during pregnancy.[6] In theory, a child having had the benefit of the technique would be more likely to realise its full developmental potential. Indeed, subjective reports from parents of children born after the treatment, and also a more objective assessment of the development of these children[7] suggest that this is the case.

However, other more refined studies argue against any advantage being enjoyed by children born after decompression treatment in either development or IQ, whether tested at an early age or later.[8-10] Similarly, no convincing evidence has been presented for less aberration in such children in brain function at an electrophysiological level, as indicated by electro-encephalographic abnormalities.[11-13]

Murdoch[12,13] attempted to use a detailed assessment of the electro-encephalogram (EEG) as 'the most convenient measure of cortical maturity presently available' to assess children subjected to prenatal maternal decompression. The results of these studies do not support Heyns's contention that antenatal decompression enhances cortical development. The objection could be raised that the EEG was assessed using traditional relatively subjective methods of measurement, and that, despite the reported changes in the EEG accompanying the development of the child, the EEG as a phenomenon is likely to be only a very gross indication of cortical function. There are, however, other electro-encephalographic phenomena which are more specific indicators of cortical function and which are measurable in an entirely objective way.

Due to technical advances made in the last decade, the electro-encephalographic response evoked in the brain by discrete visual stimulation can readily be recorded and studied. The very low voltage response is extracted from the background EEG by a computer technique relying on the improvement of the signal (response) to noise (background) ratio through the summation of the EEG immediately following each of a number of stimuli. The technique has been used successfully as both a clinical and a research tool.

Research on the ontogenesis of the visual evoked response (VER) has shown that the short-latency components develop later,[14] in keeping with the corresponding stages of morphological growth of the diffuse and specific nervous pathways.[15,16] Hrbek *et al.*[17] report that a 'mature' waveform is assumed between 1 and 2 years of age, but that further reductions in the latencies of components of the VER occur up to the age of 6 years.

Although normative data are still being accumulated to define VER characteristics at various stages of development, it would appear that this technique may provide an objective assessment of the degree of brain maturation.[18] Shorter-latency early (primary) components of the response, appear to indicate more complete myelinisation of thalamocortical radiation fibres.

Another characteristic of the VER has been identified as a useful objective measure. The majority of workers studying the relation of the amplitude of the late (secondary) components of the VER to the level of arousal or activation of a subject, report a positive correlation between the two variables.[19-21]

The present report concerns an attempt to use the VER to assess the developmental status of the central nervous system, and also the arousal level under standard experimental conditions of a group of children whose mothers had undergone decompression treatment during the last trimester of pregnancy.

SUBJECTS AND METHODS

An experimental group of 41 children (16 boys and 25 girls) whose mothers had undergone decompression treatment was compared with a control group of 20 boys and 19 girls. The mothers of the experimental group had undergone an average of roughly 148 decompression treatments each of half an hour per day and starting from the 23rd to the 29th week of pregnancy. The mothers of the control

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Date received: 26 August 1974.

group had not had any such treatment but were matched as nearly as possible with the mothers of the experimental group with regard to socio-economic status, age, educational level, and incidence of smoking during pregnancy, of threatened miscarriage and of antepartum bleeding. All subjects were firstborn. The two groups of subjects were matched for paternal educational status, weight at delivery, method of delivery, problems during the neonatal period, childhood illnesses and attendance at nursery school. The mean ages of the two groups at time of testing were very close (control 53,77 months, SD 4,40; experimental group 54,10 months, SD 4,33).

VER Recording

The VER was recorded during the course of a standard clinical electro-encephalographic examination. Results of the latter investigation are reported separately.[13] Both recording and analysis were done without knowledge of the identity of the group to which a subject belonged. The VER was recorded between electrodes P4 and O2,[22] amplified on a Galileo E8b electro-encephalograph and stored on magnetic tape using an Ampex SP-300 instrumentation recorder. The VER was later extracted from the magnetic tape recording by playing the data through a 1 - 20 Hz Krohnite model 335 bandpass filter into a Technical Instruments Computer of Average Transients (CAT). The paper tape output of the CAT was analysed on an IBM-360 computer.

The visual response was evoked by a silent, orange, 50-watt-second flash (0,32 ft-candles as measured at the distance at which the lamp was placed from the subject's eyes) with a flash duration of 0,028 msec. The flash-tube was placed approximately 20 cm from the subject's eyes. Subjects kept their eyes shut during the VER recording. The VER was derived from 50 flashes spaced at random in time but ranging from 2 to 5 seconds apart.

When necessary, mothers were present during the testing, but due to prior briefing, the subjects were prepared for the recording situation and generally most co-operative. Recording was done with the subject sitting in a quiet, normally lit, air-conditioned room, separated from the laboratory containing the recording equipment by a one-way screen. Communication between the two rooms was possible via an intercom.

VER Analysis

Analysis of the VER wave was done on an IBM-360 computer. One programme identified the major peaks occurring in the VER within specified time-bands relative to the onset of the stimulus according to criteria derived from previous normative studies.[23-25] The latency of each of these peaks was calculated.

Since an objection may be raised against using specified time-bands in the analysis when insufficient data on the VER of children exist for normative purposes, a second programme was used to identify all positive and negative peaks and list their latencies and peak-to-peak amplitudes, irrespective of time-bands. From the amplitudes, all waves were discarded which were not at least within one SD of the mean for all the waves.

A third programme calculated the area under the curve of the VER to provide an amplitude measure independent of the latencies of peaks present in the wave.

RESULTS

Results of the analysis of the VER latencies according to normative time-bands are presented in Table I. Odd-numbered peaks represent peaks with negative polarity. The variance of one group was not statistically different from that of the other for any of the measures. The means

TABLE I. LATENCIES (msec) OF MAJOR VER PEAKS IN SPECIFIED TIME BANDS FOR CONTROL AND EXPERIMENTAL GROUPS

Peak No.	Time band (msec)	Group	% Subjects in group with this peak present	N	Mean	SD
a	20 - 35	Experimental	14,63	6		
		Control	30,77	12		
II	36 - 85	Experimental	90,24	37	65,405	12,3
		Control	94,87	37	65,811	10,3
IV	86 - 125	Experimental	73,17	30	96,833	12,6
		Control	79,49	31	98,669	11,1
VI	126 - 200	Experimental	100,00	41	156,829	18,4
		Control	100,00	39	155,256	20,5
I	30 - 45	Experimental	7,32	3		
		Control	10,26	4		
III	70 - 117	Experimental	90,24	37	96,926	17,1
		Control	84,62	33	94,015	13,6
V	118 - 180	Experimental	95,12	39	144,199	19,1
		Control	89,74	35	147,179	22,4
VII	181 - 210	Experimental	53,66	22	199,602	7,5
		Control	64,10	25	194,500	8,3

were statistically different for the two groups on the latency of the negative late peak VII (t = 2,2117; $P<0,05$). Since peaks (a) and (I) were present for so few subjects, they were not included in the statistical analysis. (The groups did not differ significantly with regard to the number of subjects showing these peaks.)

The second programme discarded waves which could be regarded as 'noise'. From the remaining waves, the latencies of the first negative and positive waves occurring during the 'primary response' (within 100 msec of the stimulus) were calculated. The latencies of the first positive and first two negative waves occurring during the 'secondary response' (100 - 250 msec after the stimulus) were calculated. The latency of a late positive component was also determined. These results are given in Table II. The variance of the latency of secondary peak N1 was different for the two groups (f = 2,4; $P<0,05$) but for none of the mean latency scores did the groups differ statistically insignificantly.

From the above analysis a count was made of the number of components present in the VER. Results of this 'complexity' measure are shown in Table III. For none of the measures did the groups differ significantly.

Similarly, there were no statistically significant differences between the groups when the integrated amplitude of the VER was calculated (Table IV).

TABLE II. LATENCIES (msec) OF MAIN VER PEAKS FOR CONTROL AND EXPERIMENTAL GROUPS

Peak	Group	N	Mean	SD
Primary N1	**Experimental**	**24**	**61,718**	**28,3**
	Control	**19**	**51,118**	**25,5**
Primary P1	**Experimental**	**33**	**64,356**	**21,3**
	Control	**27**	**55,865**	**24,7**
Secondary N1	**Experimental**	**28**	**132,008**	**22,0**
	Control	**21**	**122,437**	**14,2**
Secondary P1	**Experimental**	**28**	**152,232**	**18,4**
	Control	**26**	**152,050**	**24,8**
Secondary N2	**Experimental**	**30**	**218,291**	**16,5**
	Control	**37**	**210,950**	**21,9**
Late P3	**Experimental**	**18**	**275,208**	**11,1**
	Control	**21**	**281,437**	**12,3**

DISCUSSION

From the above results it is clear that the children whose mothers had experienced decompression treatment did not differ from those whose mothers had not had the treatment, when characteristics of the visual evoked response in the two groups are compared. The one statistically significant mean difference (latency of the late P3 component) is attributed to a chance finding (1 out of 18 comparisons), and would have to be replicated before any importance can be attached to it.

The implication is that the speed of neural conduction (as indicated by the VER latencies) was the same for the two groups. This was true of both the primary response which is said to reflect the arrival of the stimulus signal in the primary reception areas, and the secondary response, which is said to reflect on the secondary elaboration within the cortex of the primary response. No evidence of advanced maturation was thus found in these measures in one or the other group. Furthermore, the level of arousal of the two groups was found to be essentially the same, as indicated by the VER amplitude measures.

Both the above measures have been linked by some workers to intelligence test performance.[24-29] Previous work has not shown any effect on IQ scores of antenatal maternal decompression.[8,10]

In the present study, a more intensive decompression treatment schedule, starting earlier in the pregnancy, was employed than in previous work. It seems likely, therefore, that if any effects were to have been induced in the fetus by decompression, they should definitely have been present. If such effects did indeed occur, they were not reflected in the measures used in the study.

TABLE III. NUMBER OF COMPONENTS IN THE VER FOR CONTROL AND EXPERIMENTAL GROUPS

Part of VER measured	Group	N	Mean	SD
Primary response (0 - 100 msec)	**Experimental**	**41**	**1,275**	**0,5**
	Control	**39**	**1,205**	**0,7**
Secondary response (100 - 250 msec)	**Experimental**	**41**	**2,561**	**0,6**
	Control	**39**	**2,256**	**0,8**
Total (0 - 250 msec)	**Experimental**	**41**	**3,829**	**0,8**
	Control	**39**	**3,462**	**0,9**

TABLE IV. INTEGRATED AMPLITUDE (MICROVOLTS) OF VER FOR CONTROL AND EXPERIMENTAL GROUPS

Part of VER measured	Group	N	Mean	SD
Primary response (0 - 100 msec)	**Experimental**	**41**	**1,850**	**1,3**
	Control	**38**	**1,914**	**1,1**
Secondary response (100 - 250 msec)	**Experimental**	**41**	**4,540**	**2,0**
	Control	**39**	**4,302**	**2,5**
Total (0 - 250 msec)	**Experimental**	**41**	**4,653**	**1,8**
	Control	**39**	**4,855**	**1,8**

CONCLUSION

The present findings support previous work[8-13] suggesting that prenatal maternal decompression does not necessarily allow a child to enjoy a neuropsychological advantage over a child who has not been exposed *in utero* to the treatment. These findings do not, of course, necessarily reflect on the potential of the technique as an obstetric aid.

We thank Mr B. D. Murdoch for negotiating and making arrangements necessary for this study and the staff of the Division of Neuropsychology of the National Institute for Personnel Research for their technical assistance.

REFERENCES

1. Heyns, O. S. (1961): Trans. Coll. Physns S. Afr., **5**, 11.
2. *Idem* (1962): Proc. Roy. Soc. Med., **55**, 459.
3. *Idem* (1963): *Abdominal Decompression—a Monograph*. Johannesburg: Witwatersrand University Press.
4. *Idem* (1963): Leech, **33**, 98.
5. Heyns, O. S., Samson, J. M. and Graham, J. A. C. (1962): Lancet, **1**, 289.
6. Blecher, J. A. and Heyns, O. S. (1967): *Ibid.*, **2**, 621.
7. Heyns, O. S., Samson, J. M. and Roberts, W. A. B. (1962): Med. Proc., **8**, 307.
8. Greenwall, P. S. (1968): 'The effect of abdominal decompression on children's intelligence'. M.A. thesis, University of South Africa.
9. Liddicoat, R. (1968): Psychologia Africana, **12**, 103.
10. Charlewood, C. A. H. (1973): The effect of early and extended maternal antenatal abdominal decompress.on on the brain function of the child, M.A. thesis, University of South Africa.
11. Nelson, G. K. (1961): *Electroencephalograms of Infants in Relation to Prenatal Maternal Decompress.on*. Johannesburg: CSIR.
12. Murdoch, B. D. (1968): S. Afr. Med. J., **42**, 1067.
13. *Idem* (1974): *The Effects of Early and Prolonged Maternal Antenatal Decompression on the Electroencephalogram of Four-year-old Children*. (PERS 204). Johannesburg: CSIR.
14. Rose, G. H. and Lindsley, D. B. (1968): J. Neurophysiol., **31**, 607.
15. Morrell, F. and Morrell, L. *in* Proctor, L. D. and Adey, W. R., eds (1965): *The Analysis of Central Nervous System and Cardiovascular Data Using Computer Methods*. Washington: NASA.
16. Scheibel, A. B. *in* Wortis, J., ed. (1962): *Recent Advances in Biological Psychiatry*. New York: Plenum.
17. Hrbek, A., Vitova, Z. and Mares, P. (1966): Activas Nervosa Superior, **8**, 39.
18. Regan, D. (1972): *Evoked Potentials in Psychology, Sensory Physiology and Clinical Medicine*. London: Chapman and Hall.
19. Bergamini, L., Bergamasco, B., Mombelli, A. M. and Sibow, F. (1964): Riv. Pat. Nerv. Ment., **85**, 573.
20. Ciganek, L. *in* Cobb, W. and Morocutti, C., eds (1967): *The Evoked Potentials*. Amsterdam: Elsevier.
21. Perry, N. W. and Childers, D. G. (1969): *The Human Visual Evoked Response*. Springfield, Ill.: C. C. Thomas.
22. Jasper, H. H. (1958): Electroenceph. clin. Neurophysiol., **10**, 371.
23. Ciganek, L. (1961): *Ibid.*, **13**, 165.
24. Kooi, K. A. and Bagchi, B. K. (1964): *Ibid.*, **17**, 638.
25. Jonkman, E. J. (1967): *The Average Cortical Response to Photic Stimulation*. Academisch Proefschrift ter verkrijging van de graad van Doctor in de Geneeskunde. Amsterdam: Universiteit van Amsterdam.
26. Ertl, J. P. and Shafer, E. W. P. (1969): Nature (Lond.), **223**, 421.
27. Bennet, W. F. (1968): *Ibid.*, **220**, 1147.
28. Rhodes, L. E., Dustman, R. E. and Beck, E. L. (1969): Electroenceph. clin. Neurophysiol., **27**, 364.
29. Griesel, R. D. and Clark, H. M. S. (1974): Psychologia Africana, **15**, 101.