

## THE ELECTRO-ENCEPHALOGRAM IN BLIND CHILDREN\*

BY

P. M. JEAVONS

*Consultant in Electro-encephalography, All Saints Hospital, Birmingham, and University Research Fellow, Department of Paediatrics, Birmingham*

THERE have been surprisingly few studies of the electro-encephalogram (EEG) in blind persons, and the first report appears to be that of Adrian and Matthews (1934) on the absence of alpha rhythm in totally blind subjects. Further reports on the alpha rhythm were made by Loomis, Harvey, and Hobart (1936), Lemere (1942), Callahan and Redlich (1946), Redlich, Callahan, and Mendelson (1946), Drever (1955), Bergman (1956), and Bergman and Jaffe (1961). Most of these studies concerned only a few patients. Lairy and Netchine (1961) reported on a group of fifty blind children, but gave little information about the alpha rhythm. There are a number of papers on the EEG findings in retrolental fibroplasia (Jim and Krause, 1954; Gibbs, Fois, and Gibbs, 1955; Kellaway, Bloxson, and Macgregor, 1955; Cohen, Boshes, and Snider, 1961), but these deal mainly with paroxysmal and focal EEG abnormalities and, with the exception of Cohen and others (1961), few authors report on the alpha rhythm. There is little information about the relation of the EEG findings to the degree of blindness or its time of onset, although Drever compared cases of early and late onset. Levinson, Gibbs, Stillerman, and Perlstein (1951), Stillerman, Gibbs, and Perlstein (1952), and Gibbs and Gibbs (1952) studied the EEG in a variety of eye disorders, especially strabismus, and Krill and Stamps (1960) and Lesný (1961) described the EEG findings in retinitis pigmentosa, the former authors commenting on the alpha rhythm.

The aim of the present study was to establish whether any particular EEG pattern was associated with blindness, to assess the alpha rhythm in relation to presence or absence of vision, and to see if there was any relation between the alpha rhythm and the degree and duration of the blindness. Since the series contains a large number of cases of retrolental fibroplasia, special mention will be made of these cases, and the relation of various causes of blindness to EEG abnormalities will also be examined. It is felt that some ophthalmologists may not be familiar with the terms used in electro-encephalography, and a brief account of these, and of the normal findings, is therefore given.

The EEG records the electrical activity of the brain by means of pens writing on moving paper at constant speed (usually 3 cm. per sec.). The brain rhythms occur at different frequencies, which have been divided into bands and named as follows:

- Alpha band : from 8 to 13 cycles per second (c/s)
- Beta band : faster than 13 c/s.
- Theta band : from 4 to 7 c/s.
- Delta band : slower than 4 c/s.

---

\* Received for publication May 16, 1963.

The normal adult EEG shows a regular alpha rhythm in the posterior regions equal in amount and amplitude on each side of the head. The rate of the alpha rhythm is usually constant for each individual and is usually at one dominant frequency (e.g. at 10 c/s or at 8 c/s), though it is quite normal to find an alpha rhythm which varies from 8 to 10 c/s or from 10 to 12 c/s.

The alpha rhythm is seen only when the individual is relaxed and has his eyes closed. It disappears or is "blocked" when the eyes are open. It will also disappear with the eyes closed if the individual listens attentively or has to perform mental calculations. If the eyes are open and staring occurs, the alpha rhythm may return. It is probable that the alpha rhythm is blocked by any meaningful stimulus requiring cortical activity, and the "blocking" can be regarded as an arousal response.

In adults there is often some fast activity in areas where alpha rhythm does not occur, but there is no slow activity in a normal adult record. Children show a variable amount of slow activity in the temporal, frontal, and central regions, especially during hyperventilation. Generally speaking, the younger the child the greater the amount of slow activity. By puberty the record is very similar to that of an adult, although some slow activity may occur in clinically normal adults up to the age of 25 years.

In brief, abnormalities in the EEG consist of too much slow activity, or asymmetry of any activity, or absence of normal rhythms, or transient discharges of high voltage slow waves, spikes, spike and wave. These transient discharges may be focal or generalized.

A normal record shows a regular alpha rhythm which disappears on opening the eyes (Fig. 1). The record of a sighted child whose eyes are open is shown in Fig. 2 (opposite). There is no alpha rhythm and the background activity consists of a low voltage mixture of frequencies.

### Method

Because the blind patient relies for his orientation on senses other than vision, it is clear that he will be extremely alert when he visits strange surroundings for a strange test. It was therefore thought that, on his first attendance at the EEG department, his alpha rhythm, even if he had any in normal circumstances, would most probably be inhibited. For this

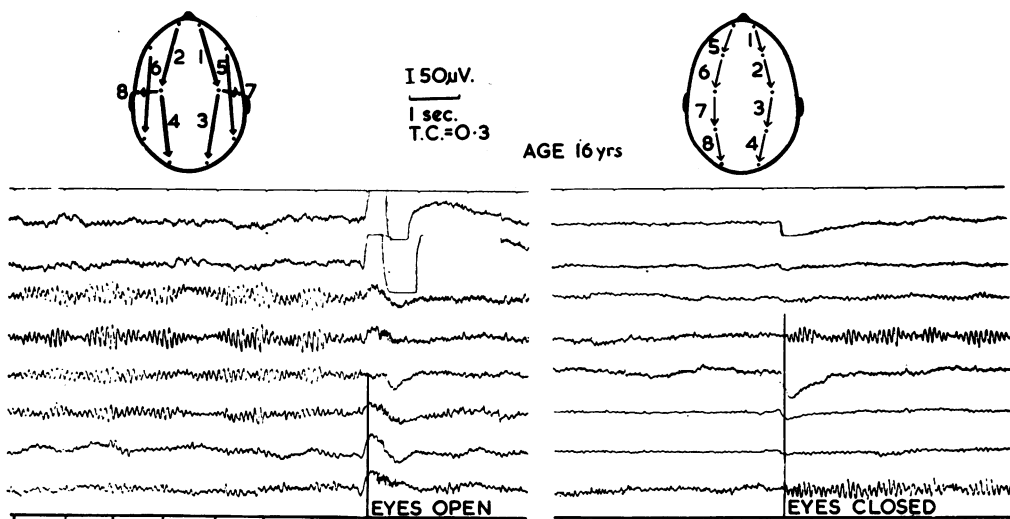


FIG. 1.—Normal electro-encephalogram, showing regular alpha rhythm which "blocks" on opening the eyes.

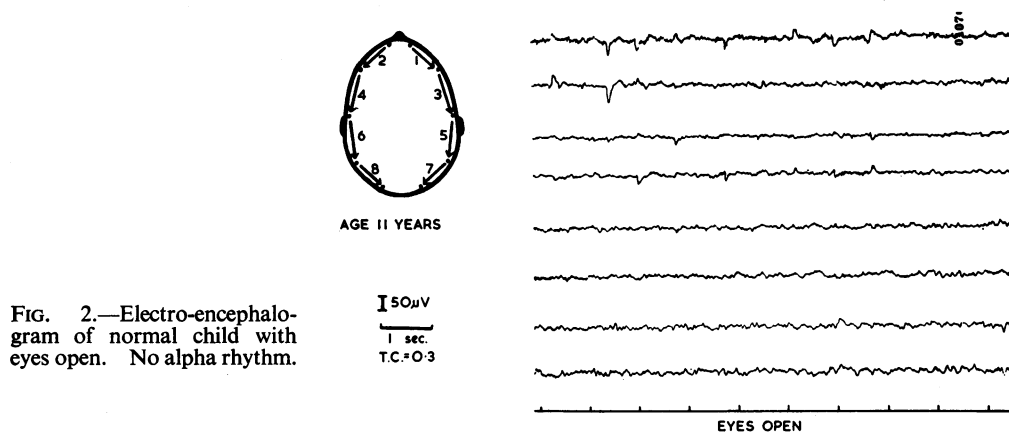


FIG. 2.—Electro-encephalogram of normal child with eyes open. No alpha rhythm.

reason all patients had two tests, usually within a month of each other. The children also attended in groups of six at a time, all members of the group knowing each other, in order to achieve maximum relaxation.

Recordings were made either on an Ediswan 8-channel machine or a Schwarzer 12-channel machine, using 21 electrodes, 5-7 montages, and bi-polar recording. Hyperventilation for  $2\frac{1}{2}$  minutes and intermittent photic stimulation were used as a routine and all records were taken with the patient awake.

Initially all the records were interpreted without the electro-encephalographer having any information about the child except his age, and an attempt was made at the time of interpretation to assess, from the EEG alone, whether the child had any vision.

Clinical information on each child was provided by the headmaster of each school, details being given as to the degree of blindness, age at onset, cause as given by consultant ophthalmologists, intelligence level with I.Q. whenever possible, history of epileptic fits with type and frequency, medication, and comments on any physical abnormalities other than those related to the eyes. The records were re-examined at a later date in the light of the above information.

### Material

The study is based on 192 children from schools for the blind.

79 came from normal schools for the blind, including grammar schools. Those of highest intelligence were totally blind, since only such cases of total blindness were originally selected for investigation. Later, however, some children with partial sight from normal schools were included. These children had very little sight, most of them only perceiving light, and a few perceiving hand movements. Twelve children from normal schools were of low intelligence.

72 children had a handicap in addition to their blindness and attended a special school for the blind (Condover Hall). Nearly all these children were of low intelligence, and some had cerebral palsy or other physical handicap. There was a far higher proportion of partially sighted children in this group compared to the group with normal intelligence, and furthermore, the subnormal children with partial vision tended to have better vision than those with partial sight from normal schools.

A group of younger children aged  $2\frac{1}{2}$  to 7 years came from an infant school for the blind (a Sunshine Home). These children had only one EEG.

Eleven blind and deaf children attended a special school (Pathways, Condover Hall).

The children in the series have therefore been divided into four groups:

- Group I. 79 of normal intelligence.
- Group II. 84 of low intelligence.
- Group III. 18 very young children who had one EEG only.
- Group IV. 11 both blind and deaf.

Some of the findings relate to Groups I and II only, because of the difficulty of assessing certain factors in the other groups. For example, it is difficult to estimate intelligence in the very young blind child or in the child who is both blind and deaf. It also seemed preferable to draw conclusions about alpha rhythm only when two records had been taken and for those who were able to hear verbal commands. On these grounds Groups III and IV have been excluded from the analysis of the alpha rhythm. Group IV is such an unusual group and so different from the others that it cannot be compared to the rest.

Table I shows the constitution of each group. The children in Groups I and II are similar in total number, average age, and sex distribution, but differ in intelligence.

TABLE I  
CASES STUDIED

Group		I	II	III	IV
Total No. of Cases		79	84	18	11
Age (yrs)	Range	7-20	7-16	2½-7	6-16
	Mean	12·7	12·7	5	10·6
Male:Female Ratio		2:1	2:1	2:1	1:1
Average I.Q.		116	65	—	—

Table II shows the degree and time of onset of blindness in the groups. "Totally blind" means that the child did not perceive light in either eye. "Partially sighted" includes all degrees of vision, unocular and binocular. The partially sighted in Group I had less vision than those in Group II. "Blind from birth" includes all cases in which so far as is known there was no evidence of vision from the first 3 months of life. It is, of course, extremely difficult to be sure that blindness does date from birth except in obvious cases

TABLE II  
DEGREE OF VISION AND TIME OF ONSET OF BLINDNESS

Group		I	II	III	IV	Total
Totally Blind	Total	53	29	12	1	95
	From Birth	29	25	9	1	64
	Later	24	4	3	0	31
Partially Sighted	Total	26	55	6	7	94
	From Birth	24	35	5	5	69
	Later	2	20	1	2	25
Uncertain					3	3

such as anophthalmos, and this is why the definition of blindness from birth must cover the possibility that it dated from the first three months.

In Group I, 67 per cent. of the children were totally blind compared to 35 per cent. totally blind in Group II, whilst the ratio is reversed with the partially sighted. This difference is entirely due to selection of cases as mentioned above. There is a considerable difference between the groups as regards onset of blindness for, although there is almost equal distribution in Group I of those totally blind from birth and those blind later, in Group II there were 25 cases totally blind from birth and only four blind later. The reverse applies to the partially sighted and there is no obvious explanation for these differences.

**Causes of Blindness.**—Table III shows the various causes of blindness and their distribution in the four groups. All diagnoses are exclusive. Where there are only one or two cases they are shown under "Other Causes" and this heading includes cases of localized infection of the eye, trauma, toxoplasmosis, etc. Cerebral tumours are included under secondary optic atrophy. Patients shown under T.B. meningitis all had secondary optic atrophy. Retinal lesions include retinal damage and chorio-retinitis but exclude retinitis pigmentosa.

TABLE III  
AETIOLOGY OF BLINDNESS

Group	I		II		III		IV		All Cases	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Retrolental fibroplasia	25	32	20	24	7	40	1	9	53	28
Retinoblastoma	16	20	—	—	1	5.5	—	—	17	9
Congenital cataract	3	4	6	7	2	11	6	55	17	9
Congenital glaucoma	9	11	6	7	—	—	1	9	16	8.5
Congenital malformation	7	9	5	6	3	16	1	9	16	8.5
Primary optic atrophy	3	4	8	9.5	1	5.5	—	—	12	6
Secondary optic atrophy	—	—	10	12	—	—	1	9	11	5.5
Retinal lesion	4	5	3	3.5	—	—	1	9	8	4
Congenital nystagmus	2	2.5	2	2.5	1	5.5	—	—	5	2.5
Myopia and Other abnormality	—	—	5	6	—	—	—	—	5	2.5
T.B. meningitis	1	1	4	5	—	—	—	—	5	2.5
Retinitis pigmentosa	3	4	1	1	—	—	—	—	4	2
Cerebral blindness	—	—	3	3.5	1	5.5	—	—	4	2
Cerebro-macular degeneration	—	—	3	3.5	—	—	—	—	3	1.5
Not known	—	—	3	3.5	—	—	—	—	3	1.5
Other causes	6	7.5	5	6	2	11	—	—	13	7
Total	79	100	84	100	18	100	11	100	192	100

It will be seen that retrolental fibroplasia is the commonest cause in all groups except Group IV, and that the distribution between the groups is much the same. This condition was equally divided between the sexes. Congenital malformations are equally distributed between the groups. Retinoblastoma is almost entirely confined to Group I, and cases of cerebral damage to Group II. The number of cases of congenital cataract in Group IV is high because these are mainly children with the rubella syndrome and have associated deafness. There are many cases of primary optic atrophy and most of them are in Group II. This, together with the high incidence of epileptic fits in these children, suggests that they may well have some brain damage to which all the symptoms are secondary and that they are not therefore cases of true primary optic atrophy. Further references to the causes of blindness are made below in the discussion of EEG abnormalities.

### Results

The EEG assessment was based on two records for each patient in Groups I, II, and IV, and on one record for Group III. It was found that a number of children showed alpha rhythm only in their second record, as had been expected, and that some showed abnormality in one record only. The number of cases in which there was a significant difference between the first and second record was 46 (28 per cent.) in Groups I and II, and four (36 per cent.) in Group IV.

Artefact was very common, more so than in sighted children, and was often due to eye movements, sometimes to restlessness, and occasionally to nystagmus. Some of the children of lower intelligence also made rocking movements and a few picked at the electrodes or at their eyes.

The records were assessed for the amount, distribution, and behaviour of alpha rhythm, and for the presence of abnormalities other than those related to the alpha rhythm. An attempt was made to assess whether vision was present from the EEG findings alone.

The findings concerning the alpha rhythm relate to Groups I and II only.

#### The Alpha Rhythm in Blindness

*Literature.*—Adrian and Matthews (1934) found no alpha rhythm in three patients who had been blind for a number of years though not from birth. They explained the absence of alpha rhythm on the grounds that this rhythm was due to the spontaneous beat of neurones in an area of the occipital cortex normally occupied by activities connected with pattern vision. When the area is unoccupied the neurones discharge spontaneously at a fixed rate, but when vision is lost the area does not remain unoccupied and must become more accessible to the rest of the brain. On the other hand, Loomis and others (1936) considered that the absence of alpha rhythm in the blind was due to the strangeness of the test and that once the subject was accustomed to the new situation the alpha rhythm appeared. They pointed out that the alpha rhythm reappeared in sighted subjects who kept their eyes open for any length of time and that inhibition of alpha rhythm was not due only to the stimulus of light. Baudouin, Halbron, Fischgold, and Mion (1939) stated that there was some alpha rhythm in the totally blind, but reported on too few cases. Lemere (1942) claimed that the EEG was a reliable objective test to differentiate true from hysterical blindness and said that persistence of the alpha rhythm when looking at an object indicated true blindness. However, Callahan and Redlich (1946) did not find any valid differentiation between true and hysterical blindness, using the response of the alpha rhythm to eye opening and closing. They found no change in alpha rhythm on opening and closing the eyes in three cases of bilateral enucleation. Bergman (1956) reported on twelve cases of cerebral blindness and found that when the patient was blind there was no occipital alpha rhythm and only two cases showed alpha rhythm at any time. In four of five patients who recovered their sight, the alpha rhythm returned. Whilst the patient was blind there was no effect from eye opening and closing or from photic stimulation. (Two of the four cases of cerebral blindness in the present series showed no alpha rhythm and the others showed a non-reactive alpha rhythm.) Bergman's conclusion was that in lesions of the eye the alpha rhythm is present but is unaffected by eye opening and closing; in hysteria the alpha rhythm is usually present and disappears on opening the eyes; and in cerebral blindness there is no occipital alpha rhythm.

Cohen and others (1961) found no alpha rhythm in nine of seventeen totally blind patients and none in seven of eleven partially sighted patients. Photic stimulation produced no

change in any patient, even the partially sighted. These authors drew attention to a change in distribution of the alpha rhythm in some cases and found more alpha rhythm in the parietal than the occipital regions. Bergman and Jaffe (1961) found unilateral abnormality of the alpha rhythm in unilateral cerebral lesions and no abnormality of the alpha rhythm in lesions of the optic nerve and chiasma. Krill and Stamps (1960) reported reactive alpha rhythm in their cases of retinitis pigmentosa but no relation between the degree or absence of alpha blocking and the visual status.

Drever (1955) appears to be the only author to have compared the amount or amplitude of the alpha rhythm in patients who had been blind early in life with those who were blind later. He found no difference between those blind before the age of 4 years and those blind after this age.

*Findings relating to the Alpha Rhythm in the Present Series.*—Only 8 per cent. of all the patients showed an alpha rhythm which reacted to light and fewer still showed an alpha rhythm reacting to sound only (Table IV). In 29 per cent. there was a non-reactive alpha rhythm (Fig. 3D, overleaf) which was fairly widely distributed throughout the post-Rolandic regions, in a few cases being more prominent in the parietal region than in the occipital areas. In the cases with a normal reactive alpha rhythm the distribution was normal, with dominance in the occipital regions, and there did not seem to be any tendency for better development of the alpha rhythm in the parietal areas.

TABLE IV  
ALPHA RHYTHM IN GROUPS I AND II

Alpha Rhythm		No. of Cases	Percentage
Reactive	To light	13	8
	To sound	5	3
Non-reactive		48	29
None		97	60
Total		163	100

The commonest pattern of EEG was a low amplitude tracing, lacking synchrony, consisting of a mixture of many frequencies with no frequency band dominating (Fig. 3A and B, overleaf). This type of record is very similar to that seen in sighted children whose eyes are open (Fig. 2) or in tense children whose eyes are closed, and it appears to reflect a state of arousal of the cortex. It occurred in 60 per cent. of cases and it is probable that it is the same type of record as that described by Lairy and Netchine (1961) as showing poor spatial organization.

Alpha rhythm was more likely to be present in the second record than the first, almost certainly because the situation was no longer strange.

*Assessment of Vision from the EEG Alone.*—The EEGs were originally examined without any knowledge of clinical details and an attempt was made to decide whether the child could perceive light or not. If the alpha rhythm appeared to block on opening the eyes or to increase in amount or amplitude on eye closure, it was thought

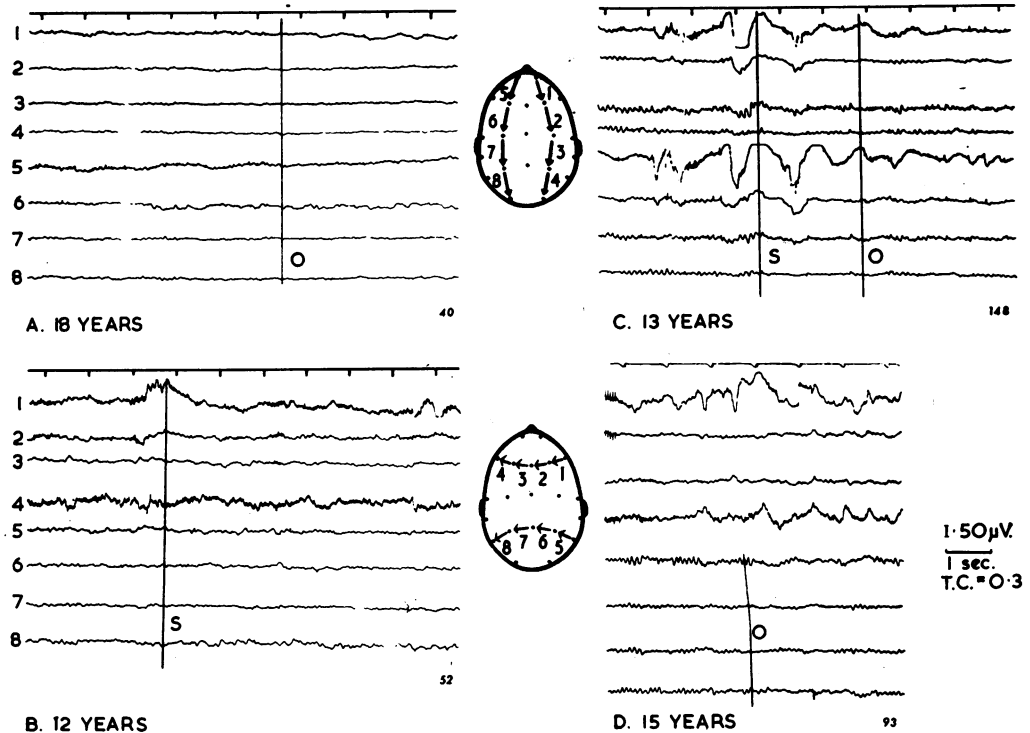


FIG. 3

(A) Totally blind from age 16 through trauma. A low voltage trace lacking organized rhythms with minimal alpha rhythm and little change on eye opening.

(B) Totally blind from birth. Retrolental fibroplasia. Low voltage trace lacking any regular alpha rhythm, with no change on eye closure.

(C) Totally blind from birth. Ophthalmia neonatorum. Alpha rhythm present. In lowest two channels, note that the alpha rhythm blocks on eye closure following verbal command.

(D) Partial sight from age 4. Cerebellar tumour. Regular alpha rhythm little affected by eye opening. (O=eyes opened S=eyes shut)

that some vision was likely to be present. Care had to be taken to distinguish those cases in which blocking of the alpha rhythm was due to the auditory stimulus of the recorder's command to open or close the eyes (Fig. 3C), but in these cases it was usual to find inhibition of the alpha rhythm on all occasions when sound occurred and there might be alpha rhythm while the eyes were open. Several children were encouraged to open and close their eyes spontaneously throughout the test without waiting for the auditory command.

Vision was suspected from the behaviour of the alpha rhythm in twenty cases and in four of these there was additional evidence of light perception from the occurrence of photic driving during intermittent photic stimulation. These four patients all had some degree of vision. Of the remaining sixteen, eleven did have some vision, but five were reported as being totally blind. The causes of blindness in these five cases were congenital cataract, iridocyclitis with detached retina, right-sided iridocyclitis with left endophthalmitis, retrolental fibroplasia, and retinitis with optic atrophy. It is perhaps possible that they may have had a little vision, particularly



since two of them were only 5 years old, and a third had an I.Q. of 62, so that clinical assessment of the degree of blindness may have been rather difficult.

There were 94 children who had some degree of vision, but only fifteen showed an alpha rhythm which was reactive to light. It is clear, therefore, that the EEG alone is unreliable in assessing the presence or degree of vision. The EEG is also unreliable as a test to differentiate true from hysterical blindness, because 60 per cent. of all patients have no alpha rhythm and some with psychological disorders also have none. Conversely, however, it is unlikely that a totally blind person would have an alpha rhythm which reacted to light.

*Alpha Rhythm in Lesions confined to the Eye.*—Of the 22 cases in which the lesion causing blindness was confined to the eye (see Table VIII, p. 99), nine (41 per cent.) showed no alpha rhythm and twelve (54 per cent.) had a non-reactive alpha rhythm. In nineteen of these patients the blindness had started after birth and this probably accounts for the greater tendency to show alpha rhythm (see below). These findings do not support the conclusions of Bergman (1956) that alpha rhythm is present in patients with lesions of the eye alone, but do agree with his findings that if alpha rhythm is present it will not react to eye opening and closing.

*Alpha Rhythm and Degree of Blindness.*—In the present series the partially sighted clearly show more alpha rhythm which is reactive to light, but there is little difference in the amount of non-reactive alpha rhythm shown by the totally blind and the partially sighted, and both groups include a similar number of records showing no alpha rhythm (Table V).

TABLE V  
COMPARISON OF ALPHA RHYTHM FINDINGS IN RELATION TO DEGREE OF BLINDNESS, TIME OF ONSET, AND INTELLIGENCE (PERCENTAGES)

Total No. of Cases	Category		Alpha Rhythm		
			Reactive	Non-reactive	None
163	All Cases		11.0	29.0	60.0
82	Totally blind		7.0	33.0	60.0
81	Partially sighted		14.8	26.0	59.2
79	Intelligence	Normal	6.3	38.0	55.7
84		Low	15.6	21.4	63.0
113	Blind	From birth	8.1	24.7	67.2*
50		Later	18.0	40.0	42.0*
54	Totally blind	From birth	7.4	22.2*	70.4*
28		Later	7.0	53.6*	39.4*
59	Partially sighted	From birth	8.4*	27.2	64.4
22		Later	31.8*	22.7	45.5

\*Difference significant at 1 per cent. level.

Table V also shows that there are more cases of reactive alpha rhythm among the partially sighted children of low intelligence than among those of normal intelligence. This is almost certainly due to the better degree of vision in those of low intelligence,

which is due to the selection of cases mentioned earlier. However, it is possible that those with partial sight and normal intelligence were generally more alert and that their alpha rhythm was therefore more inhibited.

*Relation of Alpha Rhythm to Time of Onset of Blindness.*—Fig. 4 and Table V show that cases in which the blindness dates from birth show considerably less alpha rhythm than those whose blindness started later.

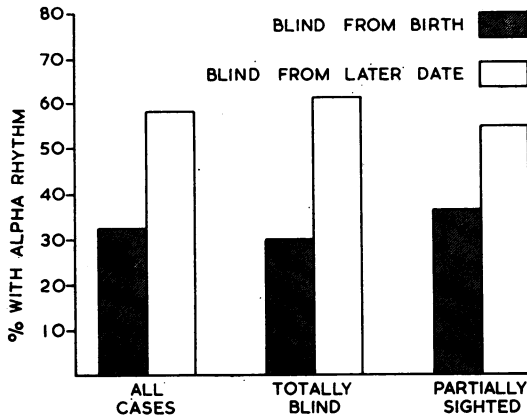


FIG. 4.—Comparison of alpha rhythm in cases blind from birth and blind later.

There was no alpha rhythm in 67 per cent. of those blind from birth compared with 42 per cent. of those blind later. Comparison of the totally blind from birth with those blind later shows the same trend (70.4 per cent. compared with 39.4 per cent.) and there is also a marked difference for non-reactive alpha rhythm (22.2 per cent. compared with 53.6 per cent.). Comparing the partially sighted from birth with those affected later, it is found that 31.8 per cent. of the latter show reactive alpha rhythm, compared with 8.4 per cent. of the former. All these differences are statistically significant at the 1 per cent. level.

In order to confirm the findings that the time of onset of blindness appears to be an important factor governing the amount of alpha rhythm, further statistical analysis was made. This showed that in the totally blind of normal intelligence whose blindness began later than the first 3 months of life, there was significantly more alpha rhythm than would be expected ( $P = .001$ ). The partially sighted of normal intelligence, whose blindness was of late onset, showed fewer cases with no alpha rhythm ( $P = .01$ ), and the partially sighted of late onset and low intelligence also had more alpha rhythm than was expected ( $P = .05$ ).

These findings suggest that the proper development of the alpha rhythm may be dependent on there having been some vision and visual stimulation at some time very early in life.

### EEG Abnormality in Relation to the Cause of Blindness

*Literature.*—Abnormalities in the EEG other than in the alpha rhythm have been reported in retinitis pigmentosa (Krill and Stamps, 1960; Lesný, 1961) and in a number of ocular disorders (Levinson and others, 1951; Stillerman and others, 1952). The former authors drew attention to the frequency of occipital foci in children with cerebral palsy and eye disorder, and similar findings were reported by Gibbs and Gibbs (1952), who stressed the frequency of occipital foci in children. Lairy and Netchine (1961) studied fifty blind children and found poor spatial organization in the EEG, the only exception occurring in some subnormal children. Slow spikes or posterior spikes on the left occurred in 25 per cent. of their cases. Levinson and others and Stillerman and others state that it is probable that ocular pathology limited to the peripheral organ does not affect the EEG patterns.

They quote one case of retinoblastoma and two of trauma with normal EEG tracings. Gibbs and Gibbs (1952) reported abnormality in cataract and strabismus but none in traumatic eye lesions. Kellaway and others, (1955) expressed the view that, where there was no retinal damage as in cataract, EEG foci did not occur, but they did find spiking in two cases of retinoblastoma and one case of anophthalmos. Gibbs, commenting on their paper, said that bilateral cataracts were associated with occipital foci, and that one case of anophthalmos had abnormal 30 c/s activity in the occipital regions.

Cohen and others (1961) found a low I.Q. in fifteen of 28 cases of retrolental fibroplasia and Gibbs and others (1955) reported 65 per cent. of their 51 cases as being of retarded intelligence, whilst 27 per cent. had fits and 10 per cent. had cerebral palsy. It is not therefore surprising that the EEG is frequently abnormal. Gibbs and others (1955) found EEG abnormality in 78 per cent. of cases, with occipital spiking in 72 per cent. These authors found that only 35 per cent. of children under 3 years old showed occipital spike foci, whereas the figure rose to 74 per cent. in children aged 3 to 14 years. They also noted the tendency for the spike foci to migrate to the temporal regions as the child grew older. This is a tendency found generally in children with a variety of disorders and occipital foci are more common in infancy than at any other time. Kellaway and others (1955) also found a high incidence of occipital foci and thought that the essential factor was bilateral destruction of the retina with total blindness in both eyes. Cohen and others (1961) found abnormal EEGs in all but one of their 28 cases and occipital foci were the most common abnormality. Ten of the seventeen totally blind cases had occipital spikes.

*Abnormalities in the EEG in the Present Series.*—Out of 192 cases, 73 (38 per cent.) showed abnormalities in the EEG. Absence of alpha rhythm was not regarded as abnormal but asymmetry of alpha rhythm was included under the heading of abnormality.

There was some difference in the proportion of abnormal records in the four groups. In Group I abnormal records occurred in 29 per cent., in Group II in 45 per cent., and in Group III in 45 per cent., but in Group IV in none. If one excludes the nineteen cases with a history of fits, the figures for abnormal records are as follows:

Group	No. of Cases	Percentage
I	25	32
II	22	26
III	7	39
IV	None	

The higher figure for the very young children is probably explained by the fact that four of the seven cases of retrolental fibroplasia in Group III had abnormal EEG records and this was a higher proportion than in the cases of retrolental fibroplasia in Groups I and II. It is known that abnormalities in the EEG are more common in this condition in the younger age group.

*Types of Abnormality in the EEG.*—These were broadly divided as follows:

(a) *Spike Abnormalities.*—All records showing spikes, spike and wave, and sharp waves, whether focal or generalized. These were records which would be classified as showing "epileptic" abnormalities: 29 cases.

(b) *Focal Slow Waves.*—The slow waves were constantly unilateral and usually confined to one area: 3 cases.

(c) *Diffuse Slow Activity*.—This is diffuse theta rhythm at 4–7 c/s, being more than normal for the age of the child. This type of abnormality is not at all specific: 18 cases.

(d) *Abnormal Alpha Rhythm*.—Usually an asymmetry of alpha rhythm, but one case showed an unusual distribution, and one showed too slow an alpha rhythm: 15 cases.

(e) *Abnormal Beta Rhythm*.—The fast activity was either asymmetrical or paroxysmal: 4 cases.

(f) *Paroxysmal Theta Rhythm*.—Episodic discharges of bilaterally symmetrical and synchronous theta activity: 4 cases.

(g) *Unstable*.—The record showed an irregular mixture of frequencies both slow and fast, sometimes on the right and sometimes on the left, and quite asynchronous: 3 cases.

Table VI shows the types of EEG abnormalities found in the various ophthalmic disorders. Since a history of epileptic fits is obviously relevant, the number of such cases is shown in brackets. Some patients showed more than one type of abnormality and these are indicated by an asterisk.

TABLE VI  
TYPE OF EEG ABNORMALITY, BY CAUSE OF BLINDNESS

Cause of Blindness	Spikes or Sharp Waves	Focal Slow Waves	Diffuse slow Activity	Paroxysmal Theta Rhythm	Abnormal Alpha Rhythm	Abnormal Beta Rhythm	Unstable	Total Abnormal Cases	Total Cases
Retinoblastoma	1			1	2		1	5 (30%)	17
Retinal detachment	1 (1)		1					2	4
Retinal fold			1					1	1
Retinitis pigmentosa	1 (1)	1						2	4
Chorio-retinitis			1		1			2	3
Congenital malformation of eyes	2 (1)		3 (1)					5 (33%)	15
Congenital glaucoma			1		5	1		7 (44%)	16
Congenital cataract	1 (1)			1			1	3 (17.5%)	17
Congenital nystagmus					1			1	4
Myopia and other abnormality			1 (1)					1	5
Infection of eye or trauma								0	4
Primary optic atrophy	3 (3)	1	2 (1)	1 (1)		1		8 (73%)	11
Cerebral tumour	1* (1)				1 (1)	2 (2)		3	5
Cerebral blindness	1 (1)		1					3	4
T.B. meningitis			1* (1)		2* (1)			3	6
Cerebro-macular degeneration	1 (1)		1				1	2	3
Other cerebral lesions	1 (1)	1	1					3	6
Retrolental fibroplasia	16* (1)		4	1	2*			22 (40%)	54
Totals	29 (12)	3 (0)	18 (4)	4 (1)	15 (2)	4 (2)	3 (0)	73	192

Figures in brackets show the number of cases in each category with a history of fits at some time. Some cases are shown twice if their EEG showed more than one abnormality.

\* Cases with another type of abnormality in addition to that shown in the particular column.

The relation of the various EEG abnormalities to the ophthalmic disorders can now be examined in more detail.

**SPIKE ABNORMALITIES (29 cases).**—The records showing spike and spike wave abnormalities can be divided into those showing bilateral and those showing focal discharges:

Abnormality	No. of Cases	No. with Fits
Bilateral discharges	2	2
Occipital spikes only	Bilateral	5
	Unilateral	5
Unilateral occipital spikes with unilateral spikes in other areas	5	1
Focal spikes other than occipital	7	5
Multiple spike foci	5	2

Most of the patients with foci in areas other than the occipital regions had temporal lobe foci, and it is noteworthy that of seven such cases five had a history of fits, whilst fits occurred in only two of the ten cases showing occipital foci alone, and in both of these cases the occipital foci were unilateral. It is surprising that only two of the five cases with multiple spike foci had fits.

If one excludes the patients who had fits, one finds that spike abnormalities are related to the following causes of blindness:

Occipital spikes only: seven retrolental fibroplasia; one retinoblastoma.

Occipital spikes plus other unilateral spikes: four retrolental fibroplasia.

Focal spikes, not occipital: one retrolental fibroplasia, one anophthalmos.

Multiple spike foci: three retrolental fibroplasia.

The marked preponderance of spike abnormalities in retrolental fibroplasia is discussed below.

**FOCAL SLOW WAVES** (3 cases).—In all three cases this abnormality was associated with a cerebral lesion. None of these patients had fits.

**DIFFUSE SLOW ACTIVITY** (18 cases).—This is such a common finding in children's records and is so non-specific that one can learn little from these cases. Table VI shows that this type of record occurred in patients with a wide variety of ophthalmic disorders, four of whom had fits.

**ABNORMAL ALPHA RHYTHM** (15 cases).—Asymmetrical or asynchronous alpha rhythm is often associated with organic cerebral damage and in four cases the cause of blindness supports this explanation (two T.B. meningitis, one cerebral blindness, one cerebral tumour). It is difficult to explain the abnormal alpha rhythm in the other cases. Five had congenital glaucoma which is not usually associated with a cerebral lesion, and one would not, in any case, expect a unilateral abnormality of alpha rhythm in the case of a bilateral eye lesion. One case of congenital glaucoma showed an unusual distribution of alpha rhythm which was mainly in the fronto-parietal regions. There were no reports of unusual clinical findings in these cases of congenital glaucoma, and none had fits. Two cases of retinoblastoma showed abnormal alpha rhythm, but as they had survived for more than 5 years after operation, one should be able to exclude a cerebral lesion, and they had no clinical symptoms. In the remaining cases with alpha abnormality, cerebral lesions could be present—congenital nystagmus, congenital cataract, retrolental fibroplasia, and retinitis with optic atrophy.

**ABNORMAL BETA RHYTHM** (4 cases).—The presence of this abnormality can be explained on the grounds of cerebral damage in the cases of T.B. meningitis, cerebral tumour, and optic atrophy, but one cannot explain the one case of congenital glaucoma.

**PAROXYSMAL THETA RHYTHM** (4 cases).—The number of cases is too small to draw any conclusions. In one case the abnormality was associated with a history of fits.

**UNSTABLE RECORD** (3 cases).—All three records were unusually unstable, showing irregular runs of slow and fast activity, sometimes on one side, sometimes on the other, with marked disorganization. One patient with an I.Q. of 51 had optic atrophy and hydrocephalus due to T.B. meningitis, and another with an I.Q. of 65 had congenital cataract and was emotionally disturbed. The third patient, however, in whom blindness was due to retinoblastoma, had above average intelligence and no evidence of any clinical abnormality apart from blindness.

### EEG Findings in Certain Ophthalmic Conditions

#### Retroental Fibroplasia (Table VII)

This was the commonest cause of blindness in the series, there being 53 cases (27 male and 26 female): 31 (59 per cent.) were of normal intelligence and 22 (41 per cent.) subnormal. The average I.Q. of those with normal intelligence was 98, compared to the average of 116 for Group I as a whole. On the other hand, the average I.Q. of the subnormal children with retroental fibroplasia was 72, which was higher than the average I.Q. of 65 for the whole of Group II.

TABLE VII  
EEG FINDINGS IN CHILDREN WITH RETROENTAL FIBROPLASIA

Intelligence	Alpha Rhythm	Totally Blind	Partially Sighted	Total
Normal	Reactive	1	—	1
	Reactive + abnormalities	—	1	1
	Non-reactive	—	2	2
	Non-reactive + abnormalities	2	—	2
	None	7	7	14
	None + abnormalities	5	5	10
	Abnormal	—	1	1
	Total	16	15	31
Subnormal	Non-reactive	2	—	2
	None	9	3	12
	None + abnormalities	4	4	8
	Total	15	7	22

One would expect cerebral pathology in retroental fibroplasia since it is a condition found in premature babies often weighing less than 3½ lb. at birth, and such infants are known to show a marked tendency to fits, mental subnormality, etc., if they survive. In addition to the tendency to lower intelligence mentioned above, there were four children with cerebral palsy and four were deaf as well as blind; there was nothing unusual in the EEGs of the latter. Three children had a history of fits and two of these had a normal EEG whilst the third showed focal spikes.

There was no alpha rhythm in 90 per cent. of the children with subnormal intelligence, compared with 77 per cent. of those with normal intelligence. There were fewer abnormal records (36 per cent.) in the children of subnormal intelligence than in those of normal intelligence (45 per cent.).

Thus, the commonest EEG abnormality was spikes, yet only one of the sixteen patients had had fits and the fifteen others showed spikes as follows:

Spikes		No. of Cases
Occipital	Bilateral	4
	Unilateral	3
Unilateral occipital with unilateral in other areas		4
Focal other than occipital		1
Multiple foci		3

Examples of the EEG abnormalities in three cases of retrolental fibroplasia are shown in Fig. 5.

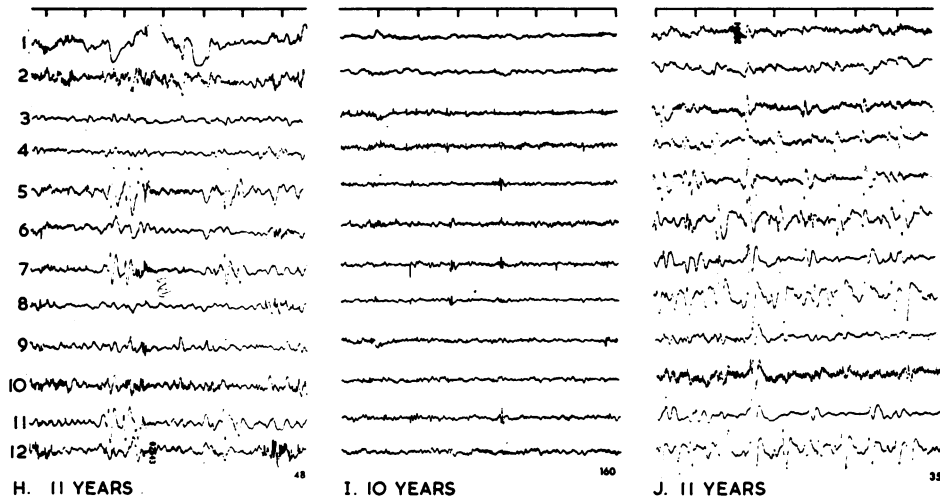


FIG. 5.—EEG abnormalities in three cases of retrolental fibroplasia (H, I, J).

(H) Partial sight from birth. No fits. Spike and wave in right temporo-parietal regions.

(I) Partial sight from birth. No fits. Tiny spikes mainly in posterior leads.

(J) Partial sight from birth. No fits. Multiple spikes, bilateral and widespread.

The number of cases showing EEG abnormality is lower than has been reported by other authors. Only 40 per cent. showed definite abnormality and only sixteen of these had spike abnormalities. Abnormalities were equally divided between the totally blind and the partially sighted. In seven cases there were occipital spikes only and in four there were spikes in other areas as well.

In view of the association of low intelligence, fits, cerebral palsy, deafness, and spikes in areas other than the occipital regions, it seems reasonable to explain the EEG abnormalities in retrolental fibroplasia on the grounds of early brain damage rather than to try to relate them to the effect of the eye lesion on the visual cortex as has been suggested by some authors.

### Congenital Glaucoma

This may be associated with a variety of other non-ocular conditions, including neurofibromata, naevi, intracranial angiomas, microcephaly, hydrocephalus, deformity of the extremities, and mental subnormality.

However, only mental subnormality was found in eight of the sixteen cases, and of these eight, three showed EEG abnormality. In one there was a diffuse non-specific abnormality, one had abnormal beta rhythm, and one had abnormal alpha rhythm.

It is possible that, in these three cases, the subnormality and the EEG abnormality reflected some cerebral abnormality associated with the congenital glaucoma.

Eight children were of normal intelligence, had no abnormal clinical signs other than those relating to their eyes, and had no history of fits. Yet four of them had unilateral abnormal alpha rhythm. It is difficult to explain a unilateral EEG abnormality in these circumstances. An asymmetry of alpha rhythm may be found in association with cortical damage and it is possible that the cases of congenital glaucoma showing an abnormal EEG may have an unsuspected subclinical cerebral abnormality.

### Primary Optic Atrophy

Eight of the eleven cases of primary optic atrophy had an abnormal EEG. This finding, combined with the high incidence of fits and of low intelligence, implies cerebral abnormality not confined to the optic nerve. Focal abnormalities were common in these cases (Fig. 6).

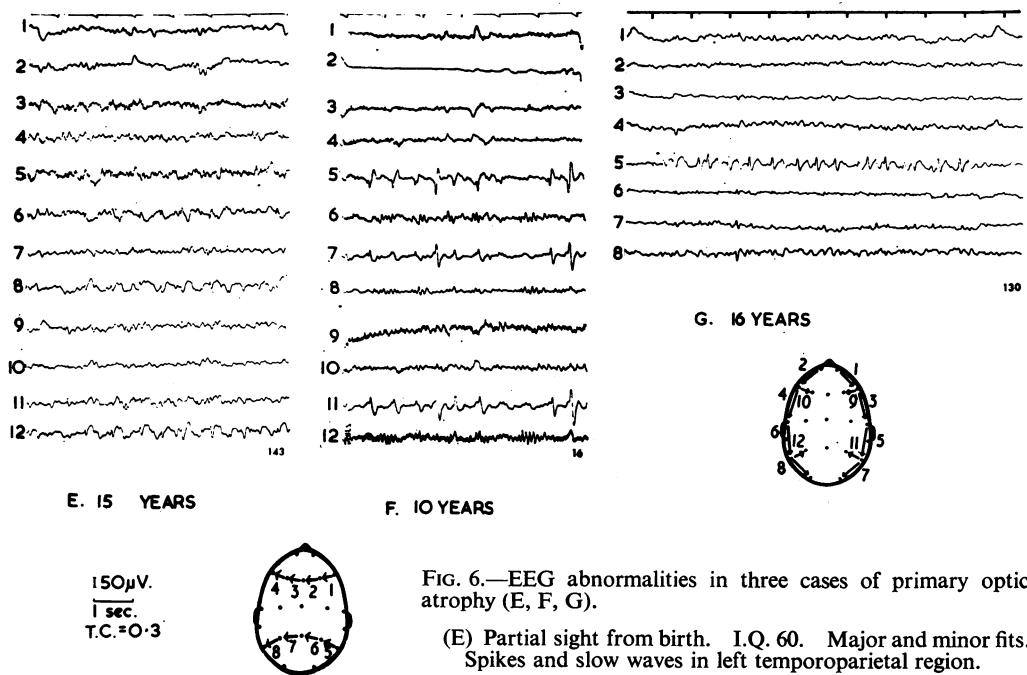


FIG. 6.—EEG abnormalities in three cases of primary optic atrophy (E, F, G).

- (E) Partial sight from birth. I.Q. 60. Major and minor fits. Spikes and slow waves in left temporo-parietal region.
- (F) Partial sight from birth. I.Q. 97. One fit 3 years ago. Focal spikes in right temporo-parietal region.
- (G) Partial sight from birth. I.Q. 78. Fits many years ago. Spike and wave in right temporo-parietal region.

It therefore seems that an EEG examination may be of value to the ophthalmologist in cases thought to be suffering from "primary" optic atrophy.

### Lesions Confined to the Eye

It is difficult to find many cases in which blindness can be said with any confidence to be due to lesions confined to the peripheral organ and very few are mentioned in the literature, but it was felt that some attempt should be made to substantiate the claim that such patients have normal EEG records.



In 22 of our cases one may presume that the lesion is or was confined to the eye. Sixteen had had bilateral enucleation for retinal glioma and had survived for more than 5 years, three had local infection of the eyes, one was blind from trauma, and two had retinal damage. All had normal intelligence and no history of fits.

The EEG was abnormal in seven of these 22 cases (32 per cent.). Abnormal alpha rhythm occurred in two cases of retinal glioma. The two cases of retinal damage showed too much theta rhythm for their age—a non-specific abnormality. Three cases of retinoblastoma showed other abnormalities: one had an extremely unstable record, one showed paroxysmal bilateral theta activity (a finding often associated with clinical epilepsy although there was no history of this), and one showed tiny bilateral spikes.

The alpha rhythm findings in these cases have already been mentioned. One may therefore say that, although spiking is rare in lesions confined to the peripheral organ, abnormalities of various types do occur in the EEG. The findings are summarized in Table VIII.

TABLE VIII  
EEG FINDINGS IN LESIONS CONFINED TO THE EYE

Cause of Blindness	Alpha Rhythm					
	None		Non-reactive		Reactive	
	Blind from Birth	Blind Later	Blind from Birth	Blind Later	Blind from Birth	Blind Later
Retinoblastoma		6 (1)		10 (4)		
Retinal Damage		1	1 (1)	1 (1)		
Infection of Eye	1	1				1
Trauma		1				
Total	1	8 (1)	1 (1)	11 (5)		1

Figures in brackets show number of cases with abnormal records.

#### Deaf and Blind Children (Group IV)

This small group of eleven children cannot be compared to the other children because so many factors were different. Two EEGs were taken in each case but it was almost impossible to establish contact with the child in the circumstances of the test, and no assessment of alpha rhythm reaction in response to a command could be made.

Seven had partial sight and only one was totally blind. In three the degree of blindness had not been fully established. It is curious that none of the eleven children had an abnormal EEG and that six showed some alpha rhythm, for this is a higher proportion than was found in the other groups. It is possible that this latter finding is associated with the greater degree of sensory isolation, and that alpha rhythm is inhibited mainly when only sight is missing and more use has to be made of hearing for orientation. However, the number of cases is too small for any conclusions to be drawn.

### Epilepsy

Of the 192 children 26 had had fits at some time. The initial report on the EEG was made without knowing whether the child had fits or not.

Seven children had normal or near normal records, four showed a non-specific diffuse abnormality, one showed paroxysmal theta abnormality, one had abnormal alpha rhythm, one had abnormal beta rhythm, and twelve had spike or spike-and-wave abnormalities. The causes of blindness were varied but in seven cases the blindness and fits were clearly secondary to cerebral damage and all these children had subnormal intelligence (3 cerebral tumours, 1 cerebral blindness, 1 cerebro-macular degeneration, 1 T.B. meningitis, 1 toxoplasmosis).

Six children were said to have primary optic atrophy and only one of these had normal intelligence. There were three cases each of retrolental fibroplasia, congenital cataract, and congenital malformation, one case each of congenital glaucoma, retinal detachment, retinitis pigmentosa, familial myopia, and in one the cause was unknown.

There was no particular relation between the cause of blindness and the EEG patterns nor was there any definite tendency for EEG abnormalities to occur in the occipital regions. In only three cases was the abnormality confined to the occipital regions, whilst five showed temporal lobe abnormality and one had an occipital and a temporal focus. Two showed multiple foci and one had bilateral 3 c/s spike and wave.

### Conclusions

Alpha rhythm is uncommon in blind children, whether the blindness is total or partial, and the commonest type of EEG record is one which is similar to that of a sighted child whose eyes are open. Although partially sighted children show more reactive alpha rhythm, there is little difference between the totally blind and partially sighted in the number of cases showing a non-reactive alpha rhythm or no alpha rhythm.

Alpha rhythm is more common in those whose blindness started after the first 3 months of life, and it is possible that the proper development of alpha rhythm is dependent on there having been some vision at some time early in life.

The EEG is probably of very limited value in assessing the presence or degree of blindness since alpha rhythm is so often absent in partial and total blindness, but it may assist in diagnosing the cause of blindness, and it was noteworthy that abnormal records were found in 73 per cent. of cases labelled as *primary* optic atrophy. Abnormalities were also found in cases of congenital glaucoma.

Abnormal EEGs were found in 38 per cent. of all 192 cases but the proportion of abnormalities is less if one excludes the cases with a history of epileptic fits. The commonest abnormalities were spikes, spike-and-wave, and sharp waves, and occipital abnormalities predominated. The commonest cause of blindness associated with spike abnormalities was retrolental fibroplasia. The presence of spike abnormalities is not indicative of clinical epilepsy, nor do focal spikes necessarily indicate focal lesions, despite the frequency of occipital abnormalities.

In cases in which the lesion causing the blindness could be regarded as confined to the peripheral organ, abnormal records were uncommon and spikes were rare.

### Summary

The EEGs of 192 blind children were examined, and the findings related to the cause, degree, and duration of the blindness. 60 per cent. of all cases showed no alpha rhythm and the commonest pattern was one similar to that seen in a sighted child with eyes open. The partially sighted showed more reactive alpha rhythm, and alpha rhythm was commoner in those whose blindness did not date from birth. 38 per cent. of all cases showed abnormalities in the EEG and these are described. The number of abnormal records in 53 cases of retrolental fibroplasia was less than that reported by other authors. The relationship between the cause of blindness and EEG abnormalities is discussed with particular reference to cases in which the lesion was confined to the peripheral organ.

The children involved in this investigation came from the following Schools or Colleges for the Blind: Condover Hall, Worcester College, Rowton Castle, Albrighton Hall, Lickey Grange, Sunshine Home, Kingswinford.

I am very grateful to the staff and principals of these schools for their co-operation, and especially to Mr. S. O. Myers of Condover Hall, who initiated my interest in the subject and whose help throughout the years has been invaluable.

Dr. J. A. Waterhouse of the Department of Medical Statistics, University of Birmingham, made the statistical evaluation and I am most indebted to him.

I appreciate the support, encouragement, and advice which I have received from Mr. P. Jameson Evans, and am grateful to Dr. B. D. Bower and Mr. M. J. Roper Hall for their helpful comments on this paper.

Finally, my thanks go to my EEG recorders, Mrs. U. Trilloe, Mrs. M. Darby, and Mrs. A. Stevens, for the patience and skill which enabled this investigation to be undertaken.

### REFERENCES

- ADRIAN, E. D., and MATTHEWS, B. H. C. (1934). *Brain*, **57**, 355.  
 BAUDOIN, A., HALBRON, P., FISCHGOLD, H., and MION, R. Y. (1939). *Bull. Soc. Ophtal. Paris*, **51**, 176.  
 BERGMAN, P. S. (1956). "Transactions of the American Neurological Association, 1956" (81st annual meeting), p. 30.  
 ——— and JAFFE, R. (1961). *Electroenceph. clin. Neurophysiol.*, **13**, 823.  
 CALLAHAN, A., and REDLICH, F. C. (1946). *Amer. J. Ophthalm.*, **29**, 1522.  
 COHEN, J., BOSHER, L. D., and SNIDER, R. S. (1961). *Electroenceph. clin. Neurophysiol.*, **13**, 914.  
 DREVER, J. (1955). *Quart. J. exp. Psychol.*, **7**, 91.  
 GIBBS, E. L., FOIS, A., and GIBBS, F. A. (1955). *New Engl. J. Med.*, **253**, 1102.  
 GIBBS, F. A., and GIBBS, E. L. (1952). "Atlas of Electroencephalography", 2nd ed., vol. 2, p. 223. Addison-Wesley Press, Cambridge, Mass.  
 JIM, V. K. S., and KRAUSE, A. C. (1954). *Amer. J. Ophthalm.*, **38**, 337.  
 KELLAWAY, P., BLOXSOM, A., and MACGREGOR, M. (1955). *Electroenceph. clin. Neurophysiol.*, **7**, 469.  
 KRILL, A. E., and STAMPS, F. W. (1960). *Amer. J. Ophthalm.*, **49**, 762.  
 LAIRY, G. C., and NETCHINE, S. (1961). *Rev. neurol.*, **105**, 198.  
 LEMERE, F. (1942). *J. Amer. med. Ass.*, **118**, 884.  
 LESNY, I. (1961). *Electroenceph. clin. Neurophysiol.*, **13**, 139.  
 LEVINSON, J. D., GIBBS, E. L., STILLERMAN, M. L., and PERLSTEIN, M. A. (1951). *Pediatrics*, **7**, 422.  
 LOOMIS, A. L., HARVEY, E. N., and HOBART, G. (1936). *Science*, **83**, 239.  
 REDLICH, F. C., CALLAHAN, A., MENDELSON, R. H. (1946). *Yale J. Biol. Med.*, **18**, 367.  
 STILLERMAN, M., GIBBS, E. L., and PERLSTEIN, M. A. (1952). *Amer. J. Ophthalm.*, **35**, No. 5, pt 2, p. 54.