THE ELECTRICAL ACTIVITY OF THE BRAIN IN SOME DISORDERS RELATED TO COMMUNICATION

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Few developments in the field of medicine and particularly of psychiatry have been attended with more initial enthusiasm than the discovery of the electrical activity of the brain. Although Caton's revelation in 1870 that it was possible to record electrical impulses from the brain received little attention, the painstaking research of Berger[4] provided the impetus for the sudden and rapid development of electroencephalography from the early thirties. Berger was a psychiatrist and his profession had very high hopes of the electroencephalogram (EEG) especially when it was finally accepted as a genuine phenomenon. Yet it is neurology and neurosurgery that received the more spectacular benefits of the technique which revolutionized the practical and theoretical problems of epilepsy and greatly facilitated the detection of cranial neoplasms and the assessment of head injuries. Meanwhile the specific problems of psychiatry were only partly clarified by the EEG which has shown itself to be of value mainly in the differential diagnosis of organic, epileptic and so-called functional disorders such as schizophrenia. Nevertheless certain findings in the psychoses do hold promise of ultimately assisting in the understanding of these conditions. While this field is beyond the scope of the present paper, it is important to take note of the application of the EEG in the related field of psychology, in both clinical appraisal and basic research on the determinants of behaviour. The purpose of this paper is to review briefly the general significance of the EEG and to evaluate its specific contribution in the field of disorders of communication.

General Significance of the EEG

The normal EEG comprises complex signals of approximately sine-wave form, these waves being recorded at the scalp (Fig. 1) at frequencies up to about 30 c/sec and voltages of a few microvolts. EEG characteristics are defined in terms of frequency, amplitude, wave-form, location, persistence and responsiveness. There are 4 main rhythms:

1. Alpha. A rhythm at 8-13 c/sec from the parieto-occipital regions of normal adults (Fig. 2). It is present only when the person is awake, is maximal during relaxation and disappears when the eyes are opened. This

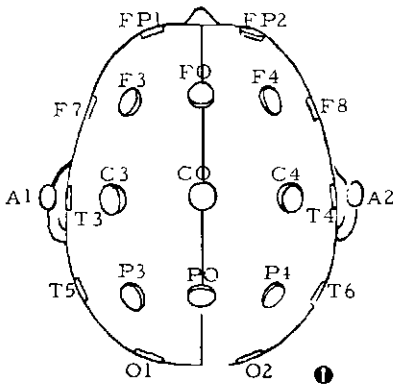

Fig. 1. Placement of EEG Electrodes: Electrode positions and names are in accordance with the recommendations of the International Federation of Societies for Electroencephalography and Clinical Neurophysiology. Recordings may be bipolar (connecting electrodes in serial chains, e.g. FP1–F3, F3–C3, C3–P3, P3–O1, etc.) or unipolar, referring each cortical electrode to a "neutral" or reference electrode. Unless otherwise stated, all EEG figures in this paper show bipolar recordings.

is known as the blocking response of the alpha rhythm and implies an important role of the functions reflected by this activity in the processes of visual perception and imagery, since alpha blocking may also be seen during visual imagery. While other perceptual modalities such as hearing may also show such changes in the alpha rhythm, the blocking response is most consistent in association with visual processes, providing the basis for the hypothesis that this electrical oscillation represents a scanning system in the visual projection area of the cerebral cortex, which is employed in the integration and coding of information received from the eyes.

The frequency of the alpha rhythm varies from person to person, and there is some evidence that individual mean frequency is a measure of central nervous speed reflected in temperament, such that relatively rapid reaction, impulsiveness, a wide span of attention and distractibility are associated with higher alpha frequencies, while individuals with lower mean alpha frequencies tend to be cautious, methodical and systematic (Mundy-Castle[43]).

Amplitude of the alpha rhythm is related to level of arousal in a curvilinear manner, being maximal during relaxed wakefulness and minimal at the two extremes of sleep and hyperexcitation.

Characteristic imagery mode is reflected in the index or percent-time of alpha rhythm, i.e. the number of seconds in a 100-second sample of EEG during which alpha rhythms are apparent. While most persons have indices of 30-90, low indices are associated with predominantly visual imagery, while high indices, and even alpha rhythms that do not block at all, are found in persons who make use mostly of verbal, kinesthetic or other types of non-verbal imagery.

More recently positive correlations of alpha frequency with intelligence test performance have been reported by Mundy-Castle[41] and Mundy-Castle and Nelson.[45]

2. Beta. These are rhythms of 14-30 c/sec, generally of lower amplitude than alpha and not specifically characteristic of any part of the brain.

Beta rhythms are associated with cortical activation, but are also found in light sleep. When more prominent than alpha activity, beta rhythms are regarded as a non-specific EEG abnormality.

3. Theta. Theta activity, at 4-7 c/sec is predominant in the EEG's of young children, and in sleep in persons of all ages. In a waking adult diffuse theta rhythms are abnormal and suggestive of epilepsy, organic brain disease or severe immaturity of the cortex. Focal theta rhythms are found in head injury and focal epilepsy and may indicate sub-cortical disturbance.

4. Delta. Delta activity comprises any potential fluctuation of $3\frac{1}{2}$ c/sec or slower. It is abnormal except in very young children and in deep sleep at all ages. Otherwise focal or diffuse delta activity is generally an accompaniment of organic pathology.

In addition to these main rhythms, other EEG features are of great clinical significance. These include such abnormal features as spikes, sharp waves, and the distinctive wave-and-spike pattern. In general such phenomena are linked with the epilepsies, but are also sometimes found in behaviour disorders. Abnormalities in the EEG are seen in the EEG's of 5 to 15 % of the normal population.

The rough classification of EEG abnormalities and their clinical correlates shown in Table 1 is a convenient although very inadequate summary.

TABLE 1: EEG ABNORMALITIES AND THEIR MAIN CLINICAL CORRELATES

Type/Location	*Diffuse or Generalized*	*Localized or Focal*
Episodic or Paroxysmal	Centrencephalic epilepsy	Focal epilepsy
Non-Episodic or non-Paroxysmal	Organic pathology Epilepsy Immaturity	Organic lesions Epilepsy Maturation defects

Developmental Aspects. The EEG appears to be the result of largely hereditary factors as shown by the similarity in general pattern in identical twins. Nevertheless the brain rhythm record is a highly individual trait, as unique as the fingerprints. Environmental factors may however bring about temporary or permanent alterations. Temporary changes are produced by drugs such as alcohol, and by mild head injury, while severe head injury, disease, malnutrition and toxic agents such as lead may cause permanent changes. Apart from the effects of such hazards, the record remains relatively stable from early adulthood to old age.

Rhythmic activity is first seen in the infant at the age of about 2 months at a frequency of 2 to 3 c/sec. This frequency increases with age (Fig. 3) until the adult (usually alpha-dominant) pattern becomes established at between 18 and 25 years of age. It is known that this

development can be adversely affected by such disorders as protein deficiency (Nelson[46]).

Activation Methods. Routine clinical EEG examination includes a comprehensive recording of the brain waves in the resting state. In order to reveal possible latent abnormalities two activation methods are generally used to supplement this recording:

(*a*) Photic stimulation (by means of a special stroboscope directed at the closed eyes);

(*b*) Hyperventilation (deep breathing for three minutes).

These techniques are described in full elsewhere (Nelson[47]).

Main Areas of the Brain Concerned with Communication

Vision and hearing are the two primary sensory modalities involved in the development of ordinary human communication. Disturbances of a sensory and motor kind related to these functions form the main theme of this account of the significance of the EEG. A brief consideration of the representation of these functions in the brain is a necessary preliminary to the survey of EEG findings. For reasons of brevity, almost exclusive attention is given to cerebral cortical representation.

1. Vision. Reference has already been made to the apparent role in visual perception of a cerebral neural process giving rise to the alpha rhythm of the EEG. The alpha rhythm originates in or near to the calcarine cortex of Area 17 at the occipital pole, but may also arise from the more anterior visual association regions in Areas 18 and 19 of the parietal lobe. Lesions in the more posterior regions tend to produce complete blindness if they are bilateral, or hemianopsia if unilateral. More anterior regions are implicated in binocular vision, while there is evidence that both frontal and temporal areas are involved in visuo-motor co-ordination. A full account of the neuro-anatomical relations is given by Humphriss.[24]

2. Hearing. The area of cortex primarily concerned with the reception of auditory signals lies in the temporal lobe, in Area 41 on the upper surface of this lobe which forms the lower bank of the Sylvian fissure. Auditory perception is mediated by the surrounding Area 42, covering most of the middle part of the upper surface of the temporal lobe.

3. Speech. Broca's area (Brodmann's area) is often considered to be of utmost importance in speech, but Jefferson[26] considers it to be a highly overrated localized functional area since excision of it in man leads to only transient aphasia, while stimulation of the region results in movements of the larynx and crude vocalization without words. Penfield and Jasper[49] point out that vocalization has representation of equal value in the dominant and non-dominant hemispheres and that either of the two areas may be removed without interference with speech. On the other hand these authors emphasize that stimulation of a speech area produces aphasia, and that such speech areas are in the left hemisphere in right-handed persons, while in left-handed persons they may be in either hemisphere. When the left hemisphere is severely injured in a right-handed child, speech may be lost for a period of months and is eventually re-established in the cortex

of the right hemisphere. Otherwise the "speech" areas of the non-dominant hemisphere are functionally "silent".

Four speech areas in the dominant hemisphere were described by Penfield and Jasper[49] and Penfield and Roberts,[50] in the frontal, parietal and temporal regions. Krieg[31] describes Area 40 as a semantic area, because obstruction of this region results in loss of "the highest level of speech". Area 22 is a syntactic area where injury may result in interference with the understanding of speech, or merely the distortion of sounds. A lesion in Area 39 produces nominal aphasia, the inability to assign names to common objects.

Fulton[15] found that unilateral lesions of the 3rd left frontal convolution in monkeys and chimpanzees led to no apparent impairment of their capacity to vocalize. On the other hand, bilateral lesions in these cortical areas led to confusion and inability to carry out complex motor manouevres, a defect comparable to apraxia in right-handed humans with lesions in Broca's area.

Electrical stimulation of the cingular gyrus in monkeys produces vocalization responses identical to those that the animal might be expected to make under normal conditions (Smith[56]). Yet Penfield and Jasper[49] found that stimulation of a cortical speech area in humans produces aphasia. These observations should not be allowed to obscure the role of subcortical mechanisms in speech; for example the subtle effects of stimulation of some areas around the 3rd ventricle were described by Sem-Jacobsen.[54]

The present paper is nevertheless primarily concerned with cortical manifestations as detected by the EEG.

The Contribution of the EEG in Communication Disorders

This brief report of EEG findings is based on data obtained from the following samples (Table 2). Most of these cases were referred to the N.I.P.R. for clinical EEG examination but a number were tested in the course of unrelated research projects.

TABLE 2

Disorder	*No. of Cases*
Visual	15
Auditory	2
Delayed speech	21
Aphasia	10
Dyslexia	3
Stuttering	5
Total:	56

Each defect will be considered separately in the light of concomitant clinical syndromes, with evaluation of the EEG findings. The nature of many of these cases necessitates a detailed individual description.

SENSORY DEFECTS

Vision. The question is often asked: In view of the apparently intricate involvement of the alpha rhythm in the process of visual perception, what are the EEG findings in blind persons?

One of the earlier studies was carried out on Helen Keller (Still[57]). As most people know, Helen Keller became blind and deaf after a sudden brief illness at the age of 19 months. An EEG recorded when she was 64 showed relatively low-amplitude occipital alpha rhythms, while "alpha" activity was prominent in the more anterior areas. This, it was suggested reflected changes in the functional organization of the cortex corresponding to the emphasis on the sense of touch or tactile discrimination which she developed.

The EEG findings in six cases of left occipital lobectomy were described by Masland *et al.*[40] Sharp waves and beta activity were seen in the temporal, central and frontal areas, and "overactivity" of the left temporal lobe was striking—alpha-like waves and high-voltage "sleep waves" were prominent in this region.

The EEG in amblyopia was studied by Parsons-Smith[48] who found abnormal records in 31 out of 50 cases, beta activity being particularly common. Bergman's[7] account of unilateral loss of alpha rhythms in hemianopia was criticized by Cobb[9] who believed that in Bergman's cases the lesion responsible for hemianopia almost certainly extended beyond the primary visual system. In 30 blind and partially blind 8 to 9 year old children with retrolental fibroplasia, Metcalf[41] found a 90% incidence of EEG abnormality which was not related to intellectual or other characteristics, but was characterized by the unusual prominence of focal disturbances, generally maximal in the occipital regions. In five cases of retinitis pigmentosa associated with juvenile cerebral lipoidosis (Spielmeyer-Vogt disease) uniformly abnormal records with diffuse paroxysmal disturbances and runs at 2.5 to 4 c/sec as well as waves-and-spikes and occasional variable interhemispheric asynchrony are described by Ellingson and Schain.[11]

Other recent studies include that of Subirana and Oller-Daurella[59] who obtained EEG's from two anophthalmic children, whose parents were first cousins. The records showed a badly organized background with diffuse fast activity. Both cases also showed occipital spikes and wave-and-spike activity. It was suggested that this reflected neurological lesions and/or "functional" focal activity in the cortical visual areas. Jeannerod and Courjon[25] describe similar abnormalities in five blind children, proposing that occipital spikes in this context represent neural degeneration of the cortex. Lairy *et al.*[34] studied partially sighted and blind children of school-going age, finding occipital spike foci on the left side in 25% and on the right side in 4%. It was concluded that cases of dyspraxia with sight occipital spikes are more or less reversible with re-education of the psycho-motor difficulty.

EEG Findings in the Present Series. Results from 15 cases of visual defect are given in Table 3. Two of the three cases with a clinical diag-

nosis of brain damage (1, 2, 3) had abnormal EEG's, raising the possibility of post-traumatic epilepsy in both cases. Cases 4, 5, 6 and 7 had a clinical diagnosis of athetosis, quadriplegia and hemiplegia. All but one had abnormal EEG's suggestive of localized cerebral abnormalities, in each case involving the parietal area (Fig. 4). Case 7 had a normal record. The following three patients, Cases 8, 9 and 10 were epileptics and all had abnormal EEG's; although the record of Case 8 did not show any EEG sign of the tumour, the EEG revealed localized epileptogenic abnormalities in both Case 9 and Case 10 (Fig. 5).

The remaining five miscellaneous disorders with accompanying visual defects are of interest: while it is not surprising that the patient with Riley's disease had a normal EEG (this is a transient disorder of childhood) the findings were of positive value in the remainder: Case 12, diagnosis unknown, had a remarkable EEG in that occipital beta rhythms seemed to have supplanted alpha activity; the record of Case 13 pointed to clear brain damage with post-operative epilepsy appearing as a disorder of concentration and memory; in Case 14 the EEG confirmed the presence of an organic abnormality in the right parietal area; Case 15 provided an opportunity for the application of another electrophysiological technique, namely electroretinography (ERG), the measurement of the electrical activity of the retina (Fig. 6). The EEG, including the response to photic stimulation, was normal, but the ERG, although showing a normal waveform on both sides, had an amplitude reduction of 50 per cent on the right. Evoked responses in the cerebral cortex showed a corresponding inequality when the eyes were independently stimulated by a flashing light, but both occipital lobes appeared capable of responses of equal amplitude. It was therefore concluded that the disturbance was peripheral and not central, but that the right eye was not totally inactive.

Comment. The incidence of EEG abnormality in these 15 cases was extremely high, 11 cases having abnormal records (73%), but this should be viewed in the light of the major clinical features, especially brain damage and epilepsy. From the point of view of visual perception, useful diagnostic information was nevertheless supplied by the EEG in 7 instances, viz.: Cases 2, 9 and 10, in whom visual disturbances were shown to be of epileptic origin or at least related to epilepsy*; cases 4, 13 and 14 in whom a localized brain disturbance was demonstrated; case 15, in whom the combination of EEG and ERG resolved the problem of central or peripheral locus of a disturbance resulting in a visual defect. Thus the EEG was of positive value in 47% of these cases.

Hearing. The EEG has been held to be of value in the investigation of hearing disorders related to speech difficulties. Abnormal EEG's were found

*A case described by Strauss[58] is called to mind here—an 11-year-old boy suffered attacks of complete blindness, with preservation of consciousness, lasting from 2 to 3 minutes; the EEG showed almost continuous high-voltage 1.5-3 c/sec occipital, temporal and more rarely parietal wave-and-spikes; although neurological examination revealed no abnormality, the interpretation was of a centrencephalic disturbance with maximal effect in the occipital regions; the EEG abnormalities and clinical phenomena were promptly controlled by anticonvulsant drugs.

in 19 of 53 children with severe hearing loss (Marcus[39]). Disturbances had a predilection for the temporal lobes. Marcus held that the EEG arousal response (desynchronization of alpha activity) was an adequate index of hearing. The use of "EEG audiometry" (to be described in relation to case 17) is considered by Fisch[12] and Gordon and Taylor[19] to be of great assistance in differentiating between peripheral and central deafness. The findings of Marcus and of Gottlieb *et al.*[20] support the hypothesis that the cerebral mechanisms relevant to speech acquisition are simplified, facilitated and less prone to interference when control of speech, hand and eye is localized primarily in one hemisphere of the brain. Generally speaking the alpha rhythm shows a reduced amplitude in the dominant hemisphere, so that the consistency and/or degree of interhemispheric difference in amplitude of the alpha rhythms might well form a profitable basis for a systematic study of communicative disorders.

EEG Findings in the Present Series. Two cases of hearing defects are detailed in Table 2. The first of these (Case 16) was reported by the speech therapist to have a fluctuating ability to speak. The patient was intelligent and it was felt that deafness was not the whole trouble. The EEG was suggestive of a localized organic abnormality.

The second patient, Case 17, who was apparently deaf, had an EEG suggestive of epilepsy associated with a brain abnormality in the left frontal area. Two months later he was given a "hearing EEG", signals at a variety of frequencies and intensities being applied to each ear independently in an attempt to assess cortical responsiveness. On this occasion averaging techniques were not used, but an apparent attenuation or augmentation of spontaneous electrical activity was seen in response to 81 of the 142 stimuli presented. The range of frequencies covered by these 81 stimuli suggested that, if the cortical responses were genuine, there was little if any hearing loss in this case. Although the findings are similar to those of Lesny and Odvarkova[35] in normal children and adults, the results of such simple tests are to be treated with considerable reserve owing to the variability of the spontaneous EEG pattern, especially in children. A series of studies utilizing automatic averaging and integrating techniques has been planned to determine more precisely the contribution to be expected from the EEG in this area. Preliminary experiments by Appleby *et al.*[3] have yielded promising results.

Comment. In only one of these patients with suspected hearing defects was an electrical abnormality demonstrated in an area approximating the auditory reception region in the superior portion of the temporal lobe. This was Case 16, where a left parieto-temporal abnormality was detected. However, it must be stressed that such focal EEG signs should not lightly be invested with specificity, since they may occur in a variety of syndromes such as epilepsy, psychopathy and delinquency, and their aetiology may be found in head injury, malnutrition (Nelson[46]) or genetic factors (Mundy-Castle[45]). Nevertheless Gervasio and Marenzi's studies[17] have shown the value of the EEG in differentiating defects in structures and functions up to and including the cochlea from those more centrally located. In a series

of 165 deaf-mute children EEG abnormalities were found in 74%; the results suggested that spreading or localized EEG disturbances indicated deafness consequent on brain injury caused by infective, toxic or traumatic factors; excessive beta activity was suggestive of congenital deafness (hereditary or otherwise) or inflammatory ear disease. Furthermore, independently of the nature of the injury, deafness due to cochlear lesions was characterized by a high incidence of normal or mildly abnormal EEG's, while interruption of the auditory fibres at the post-cochlear or cortical levels was associated with irritative abnormalities in the form of "slow spikes" or bursts of beta, theta or delta activity.

The possible relationship of hearing difficulties to epileptic disorders should not be overlooked, particularly if there is any apparent fluctuation in auditory efficiency. Gowers[21] described auditory loss before seizures in 6 patients, in one of whom there was concomitant temporary loss of vision.

More specifically the frequent association of apparent hearing defects with more general behavioural problems deserves attention in view of the functions of the temporal lobe, which, besides containing the primary receptive area for auditory sensation, also plays a profound role in the behavioural and emotional life of the individual (Chatfield[8]).

This very brief survey of EEG findings in visual and auditory anomalies should serve as a cautionary introduction to a consideration of the EEG in communicative disorders proper. Only if the excessive optimism associated with the initial enthusiasm that generally surrounds the application of a technique such as the EEG in such a highly specialised field is first dispelled are the actual electro-clinical correlations likely to emerge.

The following is a brief survey of some types of communicative disorder, representing the more common syndromes of delayed speech, aphasia and stuttering in relation to the electrical activity of the brain.

DELAYED SPEECH

Speech usually develops between the ages of 1 year and 18 months, otherwise deafness is usually suspected. If this cannot be demonstrated the possibility of a mental defect is raised, or failing this, in the words of Adams[1], "some psychic disturbance or special neurological defect", but he goes on to point out that there is often no evidence of brain injury.

In a group of 41 children with severe speech retardation Webb and Lawson[64] found that 13 had generalized and 11 focal EEG abnormalities. A high incidence of EEG abnormality was also reported by Berges *et al.*[6] in 45 children including cases of delayed speech, stuttering and difficulties in articulation: nearly 50% showed an inter-hemispheric asynchrony, with or without a spike focus, while an equivalent proportion had paroxysmal EEG disturbances during hyperventilation and photic stimulation. Most of these signs were found to disappear with the normalization of speech. An interesting interference was that a normal EEG in a child with delayed speech connoted an unfavourable prognosis, a view reminiscent of that of Hill[32] in relation to psychopathy.

EEG Findings in the Present Series. EEG's were obtained from 21 patients aged 20 months to 10 years (Table 4). The overall incidence of abnormality was 17 out of 21 (81%), the EEG being severely abnormal in one case (spastic quadriplegia), moderately abnormal in 11, mildly abnormal in one and doubtful in the remaining four.

The four patients with normal EEG's comprised: behaviour problem, aged 7; mental defect, aged 5 years 11 months; post meningitis, aged 2 years 8 months; an otherwise normal child aged 6 years 9 months.

The most common abnormality was excessive parieto-occipital or diffuse slow activity, suggestive of cortical immaturity, found in 5 patients. Focal disturbances were seen in 8 patients, in the right parietal area in 3, and in the right post-temporal, right fronto-centro-temporal, left frontal, left temporal and left parieto-temporo-occipital regions in one patient each. Two children had marked inter-hemispheric asynchronies in their EEG's.

It is perhaps not surprising that five of these patients showed EEG evidence of delayed cortical development. In such cases little of prognostic value can be gained from the EEG without repeated studies at intervals of 3 to 6 months. The focal disturbances had no consistent location, either in a lobe or hemisphere. While some of these perhaps reflected localized maturation defects, three were of the type usually associated with epilepsy (Fig. 7).

Only one of these (Case 28) had any history of a disorder related to epilepsy, yet all three showed types of EEG abnormality usually associated with epilepsy. In this connection Adams[1] observed that if external electrical stimulation of the cortex is followed by inability to speak, it is logical to assume that spontaneous electrical dysrhythmia may be responsible for delayed speech in children.

Another two cases were brother and sister. Although the EEG abnormality was posterior in Case 19 and anterior in Case 20, the right temporal lobe was implicated in each, and the possibility is raised of a genetic contribution to a localized maturation defect in these two children.

Comment. Although the incidence of EEG abnormality in these patients with delayed speech must also be viewed against the background of their more general disorders, the EEG provided valuable information in almost all cases, either confirming dysfunction of the brain or differentiating between possible brain lesions and immaturity.

APHASIA

It is accepted that aphasia is often a manifestation of a localized dysfunction of the brain, although evidence for this may be absent in many cases. There is also reason to suspect that aphasia may at least be triggered by psychogenic factors in some cases.

From the neurophysiological standpoint, Adams[1] showed that electrical stimulation of a speech area of the cortex was accompanied by aphasia for the duration of the stimulus. The role of epileptic discharges in the brain was considered by Volterra[63] who concluded that there are critical and "paracritical" speech changes of a definitely epileptic nature, but also

"non-critical" disturbances with no manifest epileptic, other neurological, intellectual or sensory defect, although the EEG shows a "grave irritative epileptic dysrhythmia", generally localized in the brain regions concerned with speech.

Kreindler *et al.*[32] found marked interhemispheric asymmetries in the blocking response of the alpha rhythm in 19 of 25 aphasic patients. It was inferred that the absence of alpha rhythm blocking in the dominant hemisphere might be attributed to a unilateral alteration of reticulo-cortical interrelations.

Nevertheless severe aphasia of various types is commonly associated with a normal EEG (Gibbs and Gibbs[18]), but where the record is abnormal, these authors claim a frequent association with mid-temporal slow wave foci.

EEG's were obtained by Liberson[36] from 18 aphasic patients, 13 of whom had abnormal records. Of these 10 showed depression or suppression of evoked potentials in the affected hemisphere (all 10 suffering from middle cerebral artery thrombosis), while 3 had bilaterally abnormal EEG's. These findings revealed a high degree of concordance with the evaluation of the speech therapist.

EEG Findings in the Present Series. EEG's were obtained from 10 patients with aphasia, aged 3 to 37 years. There was a history of epilepsy in two cases, while the remainder comprised one case each of spastic quadriplegia, surgical anoxia, head injury, behaviour disorder, mental retardation, "bad home environment", uncomplicated speech and spatial difficulty, and febrile illness.

The EEG was normal in only two of these patients: an $8\frac{1}{2}$ year old child with a bad home environment and a 3 year old child with a history of convulsions. The incidence of EEG abnormality was thus 80% in this small sample, the record being severely abnormal in three cases (quadriplegia, behaviour disorder and surgical anoxia), moderately abnormal in three (speech and spatial difficulty, febrile illness and mental retardation), and doubtful in two (head injury, epilepsy).

The types of abnormality were, as in the group with delayed speech, varied: excessive non-paroxysmal slow activity was seen occipitally in the case of speech and spatial difficulty, in the left temporal area in surgical anoxia, and in the right hemisphere in head injury. Focal bursts at various frequencies and/or spikes and/or waves-and-spikes were seen in spastic quadriplegia (right-frontal), febrile illness (temporal and generalized), behaviour disturbance (right hemisphere), and mental retardation (left frontal and fronto-temporal).

Although there was again no consistent pattern of abnormality, the EEG was of positive value in 8 of these 10 cases:

Case 39: An 8-year-old right-handed boy had speech and spatial difficulty, with no other symptom. The EEG suggested a severe cortical maturation defect, or organic pathology, involving the left occipital area in particular (Fig. 8),

Case 40: A 3-year-old boy with spastic quadriplegia and aphasia had no epileptic manifestation but his EEG showed numerous runs of high-voltage delta

activity and waves-and-spikes, sometimes generalized but with a focus in the right frontal area.

Case 41: An 8½-year-old boy with expressive aphasia had a bad home environment. Since he had tried to formulate words his defect was regarded as probably not psychogenic. However, his EEG showed no evidence of either organic pathology or delayed maturation.

Case 42: A 4-year-old boy had had a febrile illness 2½ months previously, after which he stopped talking and walking. An organic brain lesion was suspected but the EEG showed generalized wave-and-spike outbursts indicative of epilepsy, with no sign of a focal disturbance (Fig. 9).

Case 43: A 30-year-old woman had a heart operation 3 years prior to her EEG. During the operation she suffered a cerebral haemorrhage with consequent total right paralysis and inability to speak, read or write. Speech therapy had been successful to a limited degree: while she was able to speak slowly but with some echolalia, her EEG (Fig. 10) showed a clear focus of delta, theta and alphoid rhythms in the left temporal area. Had it been possible to carry out repeat EEG's the patient's progress might have been more precisely charted and even predicted.

Case 44: A 23-month-old child had fallen off a table at the age of 17 months and ceased to speak, although he was able to convey information by signs. His behaviour deteriorated and he was reported to be losing his memory. The EEG was doubtful due to a persistent slight asymmetry between the hemispheres, delta activity arising chiefly from the right temporal area. This could reflect a maturation defect or an organic lesion. A repeat EEG at the age of 31 months resolved the problem. This record was normal, suggesting that the previous record had reflected maturation processes. He had meanwhile recovered the ability to speak.

Case 45: A 7-year-old boy was referred because of a speech defect which had appeared at an early age and resisted therapy. He was an illegitimate child, ill-treated by his mother and admitted to an orphanage. The first EEG was doubtful showing various disturbances of quite obscure significance. The second EEG, 3 weeks later, was similar, but raised the question of a left-sided cerebral abnormality. The third EEG, after a further 6½ months was unequivocally abnormal, showing runs and bursts of theta activity, chiefly of temporal origin, with bursts of delta waves often confined to the left temporal area. His speech had improved but his general behaviour had shown marked deterioration. The EEG abnormalities were interpreted as reflecting a severe maturation defect underlying the behavioural deviation.

Case 46: A 3½-year-old boy gave the impression of being retarded and aphasic. A neurological examination led to the provisional diagnosis of anterior cerebral abnormality. This was confirmed by the EEG which showed a clear left fronto-temporal focus of spikes and sharp waves.

Comment. Gowers[21] and Penfield and Jasper[49] described aphasia in epileptics, often forming the most important sign of the seizure, but always associated with an abnormal discharge in the dominant hemisphere. The latter authors stress that while an epileptic discharge produces activation and interference, the effect on speech is confined to interference. They quote a case in which the EEG contained spikes from the left inferior temporal lobe. As Adams[1] puts it, "epilepsy in a speech centre" does not produce a "pouring out of words" during the attack, but instead there is an inability to speak; in spite of the fact that stimulation of a cortical speech area causes movement of the peripheral speech apparatus, there is no external vocalization. Typical of such an effect is a case described by

Gastaut *et al.*[16] of a man with seizures accompanied by rhythmic left temporal and post-temporal spikes, during which he had expressive aphasia.

The potential contribution of the EEG in cases where a psychogenic factor is in question is well exemplified by a case cited by Bagchi *et al.*[5]: a highly intelligent girl with no personal or familial history of epilepsy developed sibling rivalry and stopped talking. There were no other behavioural changes. The EEG showed severe abnormality in the form of generalized wave-and-spike discharges. No follow-up data are available, but such a record is strongly suggestive of epilepsy and would provide a strong basis for therapy based on anticonvulsants even though overt epileptic symptoms are absent.

DYSLEXIA

The concept of cerebral dominance has received much attention in the literature relating to reading difficulties, but Hughes *et al.*[23] and Kennard and Rabinovitch[27] found no correlation between EEG abnormalities and either reading difficulty or cerebral dominance. The study of Webb and Lawson[64] previously cited was undertaken because of the frequent observation that degree of efficiency in reading in a given child often varies from day to day or even from hour to hour, "as though the deficit were intermittent or paroxysmal." They describe a case of a 13-year-old child with severe dyslexia whose EEG was abnormal due to left temporal spikes. An identical twin had a similar EEG but no other defect. In the opinion of these authors this suggests the possibility of an organic defect underlying dyslexia in the one twin which was demonstrable by the EEG alone.

A recent investigation by Knott *et al.*[30] of 50 children with reading disabilities revealed abnormal EEG's in 31 (84%), of whom 15 had 14 and 6 per sec positive spikes in their sleeping EEG's. Three groups were distinguished:

(a) Normal EEG's;
(b) Abnormal EEG's with no spikes;
(c) Abnormal EEG's with spikes.

There was however no significant difference in reading disability or any other characteristic between these groups. Of the 31 abnormal records 41% contained positive spikes while 50% showed posterior slow dysrhythmia. Three patients were studied. From the first of these only one EEG was obtained, but the other 2 were intensively investigated.

Case 47: A 21-year-old male mill-hand was stated to have congenital alexia. He could understand written and spoken speech very well but was unable to read or write. There were no clinical abnormalities and he gave the impression of having a good I.Q. He was right-eyed and right-footed. The EEG was normal but very unusual in that alpha rhythms at 9-10 c/sec were more prominent frontally than parieto-occipitally, amplitudes being 20 and 15 microvolt respectively (Fig. 11). Phase relationships showed that the anterior rhythms were identical with those of more orthodox location, and were thus not independent alphoid rhythms.

Case 48: A 12-year-old boy was referred because of dyslexia. His first EEG was moderately abnormal due to a number of medium-high voltage 2 c/sec runs

from the left parieto-occipital area, and some asynchrony of the alpha rhythms. At this stage the interpretation was of a maturation defect or organic abnormality in the left parieto-occipital area. A second EEG 5½ months later showed a reduction of slow activity which was now more prominent on the right side, although still of parieto-occipital origin. A third EEG after a further 8½ months showed little change, except that parieto-occipital slow activity appeared independently on both left and right sides, giving some evidence of sub-cortical involvement. The final interpretation can only be made with confidence after further EEG's have been obtained. There has been little change in the dyslexia.

Case 49: A 3-year-old boy with a fluctuating behaviour pattern and mild right hemiplegia following surgery for a left cerebral haemorrhage caused by a fall at the age of 10 months. His EEG showed several focal paroxysmal 1-2 per second wave-and-spike discharges from the right parieto-temporal region, being suggestive of an epileptogenic abnormality in this area (Fig. 12). His second EEG a year later was more abnormal due to an increase in number and voltage of the focal disturbances which had spread to the fronto-central areas as well. After another year the EEG was improved in that wave-and-spike disturbances were less prominent and confined to the right parietal area. A fourth EEG after another 10 months, at the age of 5 years 8 months, was still abnormal but showed further improvement due to reduced amplitude of abnormalities and dissociation of spikes and slow waves.

And this time dyslexia became apparent. There was some improvement in this respect, followed by a relapse prior to the fifth EEG at the age of 7 years. The record had changed, containing numerous isolated spikes and larval waves-and-spikes, especially from the left frontal area. After a further one and a half months isolated spikes were seen in the left temporal, fronto-temporal and parietal areas, with some slow activity in the homologous regions of the right hemisphere. Reading difficulties continue.

At no stage has this patient given any sign of epilepsy, yet the EEG abnormalities are those most commonly associated with epilepsy. Whether or not this is a case of latent epilepsy, it is striking that the EEG anomalies appear to arise in the regions of the dominant hemisphere concerned with reading. It is however important to note the fluctuating nature of his disorder, characterized by intermittent distractibility and reduced stamina.

Comment. These three cases illustrate once more the variety of EEG abnormalities associated with disorders of the aphasic variety. They also serve to underline the importance of serial studies to take account of changes due to maturation.

STUTTERING

An early paper by Travis and Knott[60] reported no difference in EEG frequency between normal speakers and stutterers, but in two stutterers tonic spasms without vocalization were accompanied by high voltage spikes in the EEG, suggestive of epileptic involvement. In 1937 a further report by Travis and Malamud[62] again indicated no difference in EEG frequency between stutterers and controls, and a third paper (Travis and Knott[61]) supported these results.

A subsequent investigation (Lindsley[37]) did however reveal interhemispheric differences in brain potentials in stutterers, and Freestone[13, 14] found that stutterers had a higher alpha and beta amplitude than normal

speakers and that this increased further during stuttering. In addition there was relatively little amplitude difference between the hemispheres in stutterers, who also showed less alpha blocking with speech. These features would suggest a diminished focus of attention in stutterers, but Freestone's view was not that the neurological differences suggested by the EEG results were the cause of stuttering but that they acted "as potential subsoils in which stuttering may arise".

In 1943 Douglass[10] reported that, in silence, normal speakers showed more alpha blocking in the right hemisphere, stutterers in the left. During speech this difference did not appear. His sample of 20 stutterers showed more bilateral alpha blocking during speech than in silence and he concluded that speech has a greater attention value for stutterers. In the same year Knott and Tjossen[31] published data supporting the view of Douglass and interpreted these as favouring the lateral dominance theory of stuttering.

In 1954 Knott and Correll[28] in a study of the EEG's of 14 stutterers found "no very striking difference" between these and the records of non-stutterers, although the former had a slightly higher alpha amplitude. Although Rheinberger *et al.*[52] were also unable to find any significant difference in the EEG's of 10 stutterers and 10 non-stutterers, and Scarborough's[59] study accorded with this, Pierce and Lipcon[51] found abnormal records in 22% of stutterers and only 8% of a control group. Other differences relating to clinical history and psychological assessment led Pierce and Lipcon to the conclusion that stuttering as a habit is attributable to interrelated psychic, social and organic factors. In line with this hypothesis is the recent report by Berges *et al.*[6] already cited in connection with delayed speech.

Morin[42] investigated 30 young stutterers before and after speech therapy which was successful in 9 cases, produced an improvement in 14 and failed in the remaining 7. Morin claims that the EEG has a prognostic value since all 7 failures had normal EEG's while most of the others had abnormal records with "slow paroxysmal discharges". Where the EEG was most abnormal success was spectacular and rapid.

In addition to these implications concerning the relationship between brain function and stuttering, there is no doubt that some cases of stuttering are associated with frank but unusual epileptic patterns. Two such cases are described by Anastopoulos *et al.*[2] Furthermore, assessments of capillary blood oxygen saturation in stutterers during stress resemble those of paranoid and epileptic patients rather than those of neurotics (Levett Doust[38]).

EEG Findings in the Present Series. EEG's were obtained from five male stutterers aged 8 to 29. In four patients the EEG was abnormal, and the remaining case had an unusual record:

Case 50: An 8-year-old boy was classified as brain damaged because of marked difficulty in conceptualization, mirror-writing and a history of seizures. His mother had resented her pregnancy and he was born after a five-day labour. He started to stutter not long before the EEG which was grossly abnormal due to

a focus of slow activity in the left parietal region suggestive of an organic disturbance. A repeat EEG 10 months later showed little change.

Case 51: A 13-year-old boy was referred because of stuttering and a tic. No history was available. His EEG was of doubtful normality owing to excessive theta activity, raising the question of cortical immaturity.

Case 52: A 19-year-old army recruit was normal except for a marked stutter. His EEG was severely abnormal due to an almost continuous focus of slow activity in the right post-temporal area, and severe slowing with hyperventilation (Fig. 13).

Case 53: A 19-year-old toolmaker had a history of sudden onset of stuttering at the age of 8 years, following the divorce of his parents and subsequent remarriage of his father. His EEG was moderately abnormal due to excessive sometimes paroxysmal theta and beta activity from the right post-temporal region. When asked to read aloud, verbal blocking was accompanied by brief bursts of temporo-occipital 6-7 c/sec activity.

Case 54: A 29-year-old man, normal in all respects except for his stutter, had a normal but unusual EEG. During reading there was marked lambda variant activity at 5-7 c/sec. At halts due to stuttering the following phenomena were seen:

(a) Brief bursts of occipital 12-14 c/sec activity (Fig. 14).
(b) Suppression of lambda variant activity.
(3) Traces of irregular occipital beta rhythms.

The first of these (12-14 c/sec bursts) preceded the halts slightly, while suppression of lambda activity generally followed them. Lambda activity is often seen during successful reading and has been reported to disappear during stuttering. The clear augmentation of alpha activity during stuttering is suggestive of fluctuating attention.

Comment. In these five cases the only abnormality of any apparent consistency is the temporal or post-temporal disturbance in three patients. All were right-handed, yet the focus was in the left, or presumably dominant, hemisphere in only one case. The significance of such localized abnormalities and of EEG changes accompanying stuttering will be dealt with in a subsequent report of an experimental study of stutterers.

Conclusions

The data presented here provide little reason for optimism on the part of those seeking straightforward correlations between communicative disorders generally and the electrical activity of the brain. In spite of the high incidence of EEG abnormality in these patients, this would appear to relate more directly to an underlying clinical syndrome such as epilepsy which need not necessarily be accompanied by any disorder of a sensory or motor kind concerned with communication.

Although some of these cases, particularly those with aphasia and stuttering, demonstrate focal abnormalities, such abnormalities may occur in the dominant or sub-dominant hemispheres, although the main speech centres are to be found in the dominant hemisphere. Such cases merely serve to support what is known of the significance of speech and other centres in the dominant hemisphere. On the other hand an actively abnormal discharge in the sub-dominant hemisphere can disturb the operation

of the crucial centres in the dominant half of the brain. Such an abnormal discharge could have its origin in an injured portion of brain tissue, in an epileptogenic focus or even in a region which has failed to mature at the same rate as the remainder of the brain.

Research in this field must however constantly take account of the recent warning of Stern[55] that the assumption that the brain is a "master organ" controlling other physiological systems, can not exclude the effect of these other systems on the activity of the central nervous system. In the following paper the question is raised of the relative contribution of brain dysfunction (as reflected in the EEG) to stuttering, and the effect of stuttering on concomitant brain activity.

It is the detection and localization of sites of abnormal brain function giving rise to a variety of clinical symptoms amongst which may be a disorder of communication, that the main practical benefit is to be derived from EEG examination at the present time. Concomitantly the common problem of the relative contribution of organic and psychogenic factors to the development of a communication disorder can often be resolved with the assistance of an EEG.

It remains to be discovered whether refinements of the basic approach to the EEG, such as the study of evoked responses and the analytical techniques of auto- and cross-correlation, will throw more light on the precise role of the electrical activity of the brain in the determination of the efficiency of man's communication with his world.

Summary

A general introduction to electroencephalography (EEG) is given, followed by a brief outline of the main brain areas concerned with vision, hearing and speech. Previous EEG findings in sensory defects, delayed speech, aphasia, dyslexia and stuttering are considered and discussed in the light of EEG's from representative cases. Finally the value of the EEG in the diagnosis and study of communication disorders is appraised.

Opsomming

'n Algemene inleiding tot elektroenkefalografie (EEG) word aangebied. Daarop volg 'n kort oorsig van die vernaamste breingebiede wat by gesig, gehoor en spraak betrokke is. Vorige EEG-bevindings by waarnemingsgebreke, vertraagde spraakontwikkeling, afasia, disleksia en gehakkel word beskryf en in die lig van EEG's van verteenwoordigende gevalle bespreek. Laastens word die waarde van die EEG in die diagnose en studie van kommunikasiesteurings oorweeg.

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APPENDIX

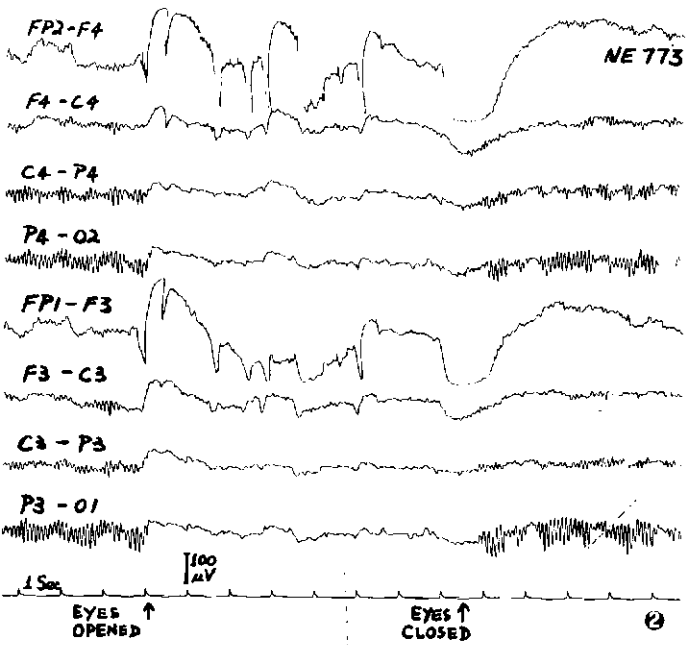

Fig. 2. Normal Adult EEG: Alpha rhythms, most prominent in the parieto-occipital leads, disappear or block with eye-opening. The large deflections in channels FP2–F4 and FP1–F3 are artifacts due to eye-movement. Voltage calibration is in microvolts as in succeeding figures.

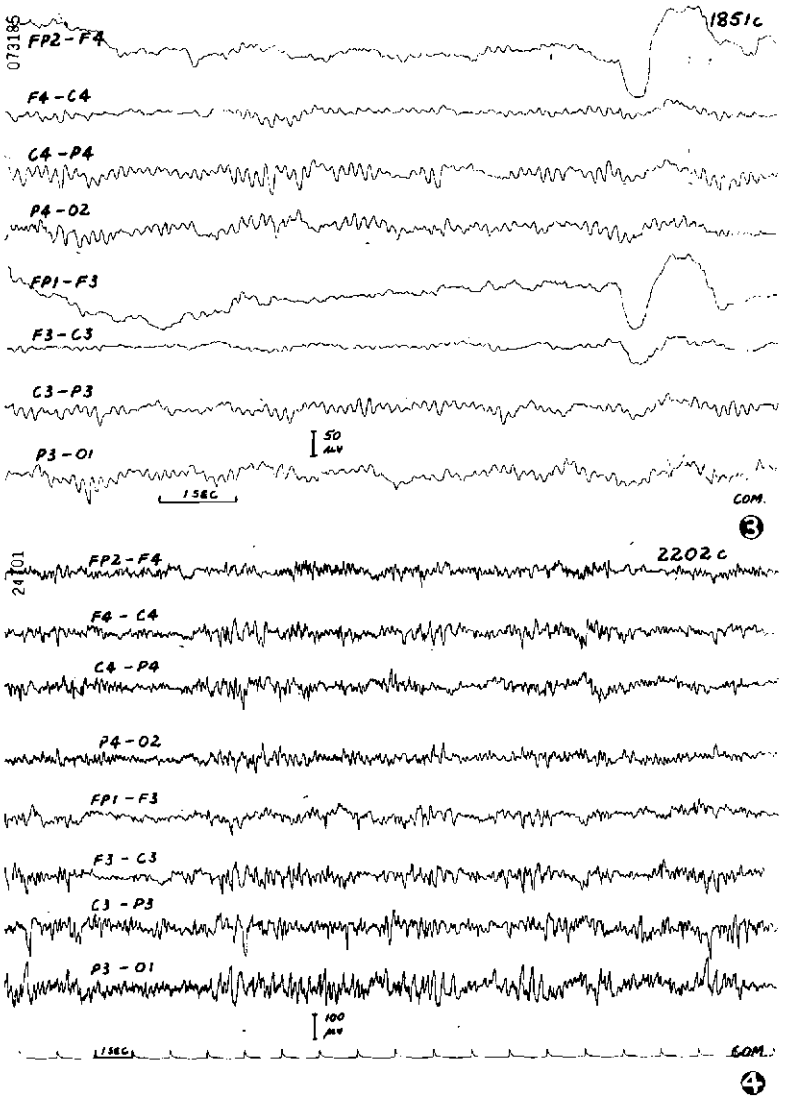

Fig. 3. Normal EEG from a Child Aged 9 Years: Dominant activity is at 7·5–8 c/sec. Large deflections in channels FP2–FP4 and FP1–F3 are due to eye-movements.

Fig. 4. EEG in Unilateral Blindness: Case 6: Girl, 9 years old, almost blind right eye (Table 3): focal sharp waves in the left parietal area (channels C3–P3 and P3–O1).

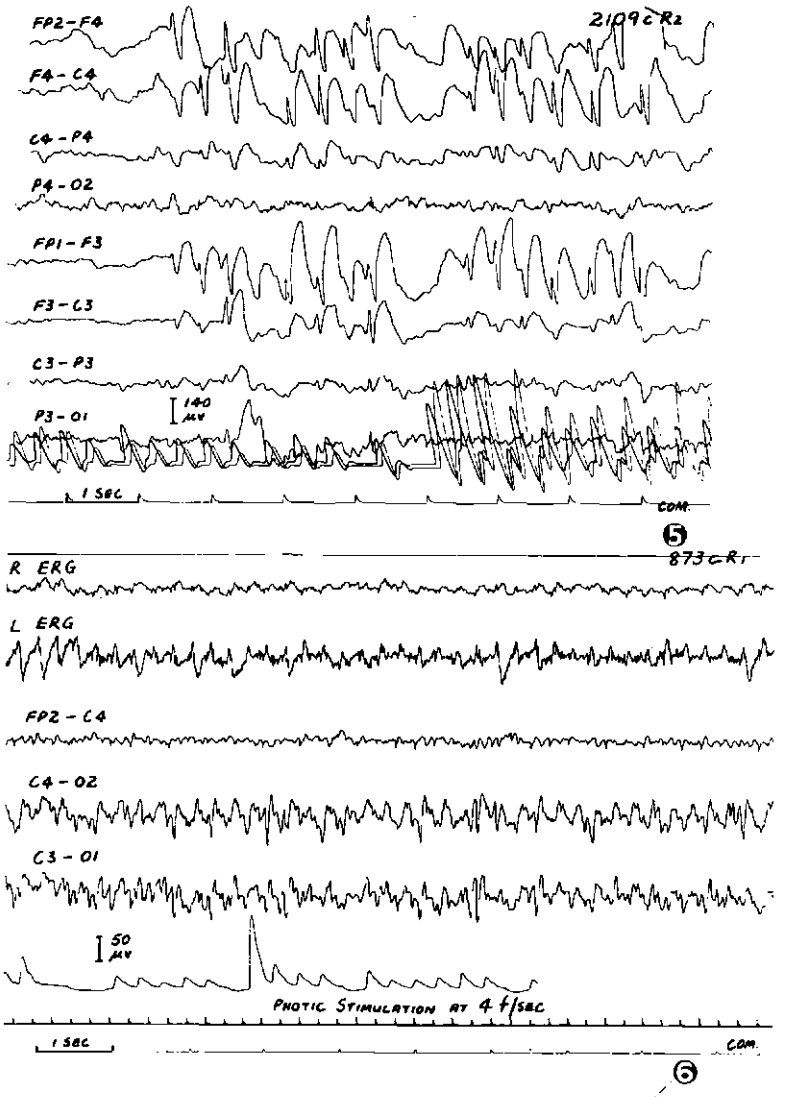

Fig. 5. EEG in Epilepsy with Visual Defect: Case 9: Boy, 7 years (Table 3): focus of slow waves and spikes in the right frontal area (channels FP2–C4 and C4–O2), suggestive of a localized epileptogenic abnormality.

Fig. 6. EEG and ERG in Unilateral Visual Defect: Case 15 (see Table 3 and text): Boy aged 14 years. Record shows approximately equal amplitude of cortical response to photic stimulation at 9 flashes per second, but marked reduction of ERG on right side. Irregular trace above photic stimulation registration is automatic frequency analyzer write-out, showing dominant activity at 4 c/sec. in R ERG.

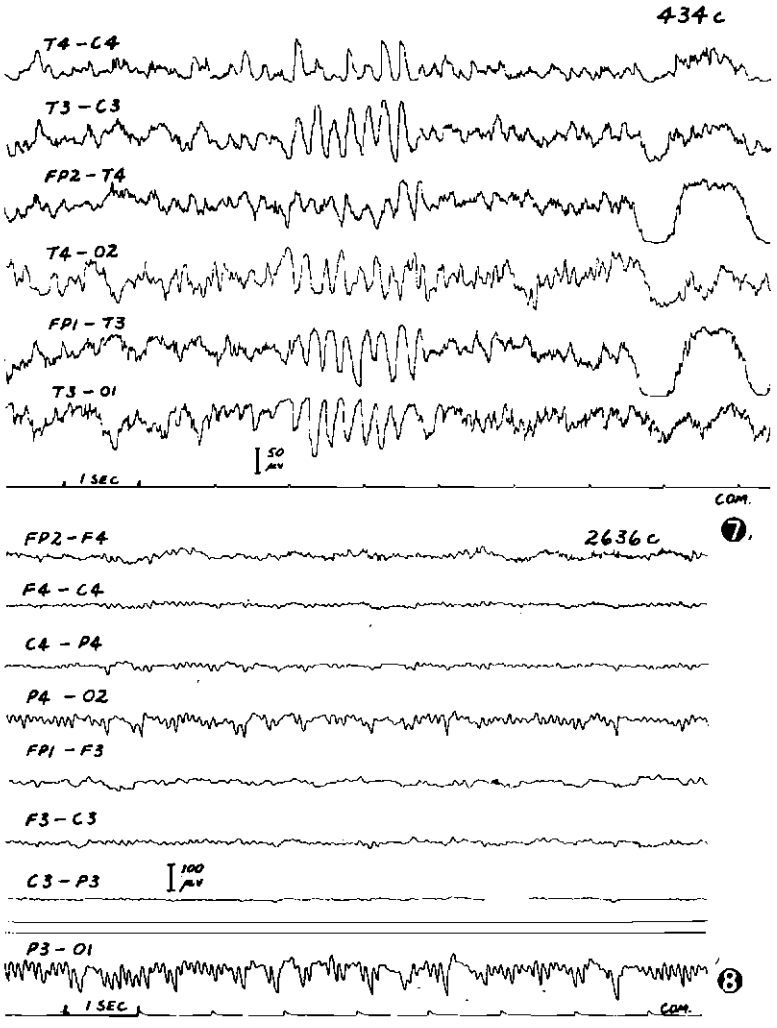

Fig. 7. EEG in Delayed Speech: Case 22: Girl, 7¾ years (Table 4). Excessive slow activity with occasional high-voltage 4–5 c/sec. bursts.

Fig. 8. EEG in Aphasia: Case 39 (see text): Focus of delta activity in left occipital region, spreading to right side.

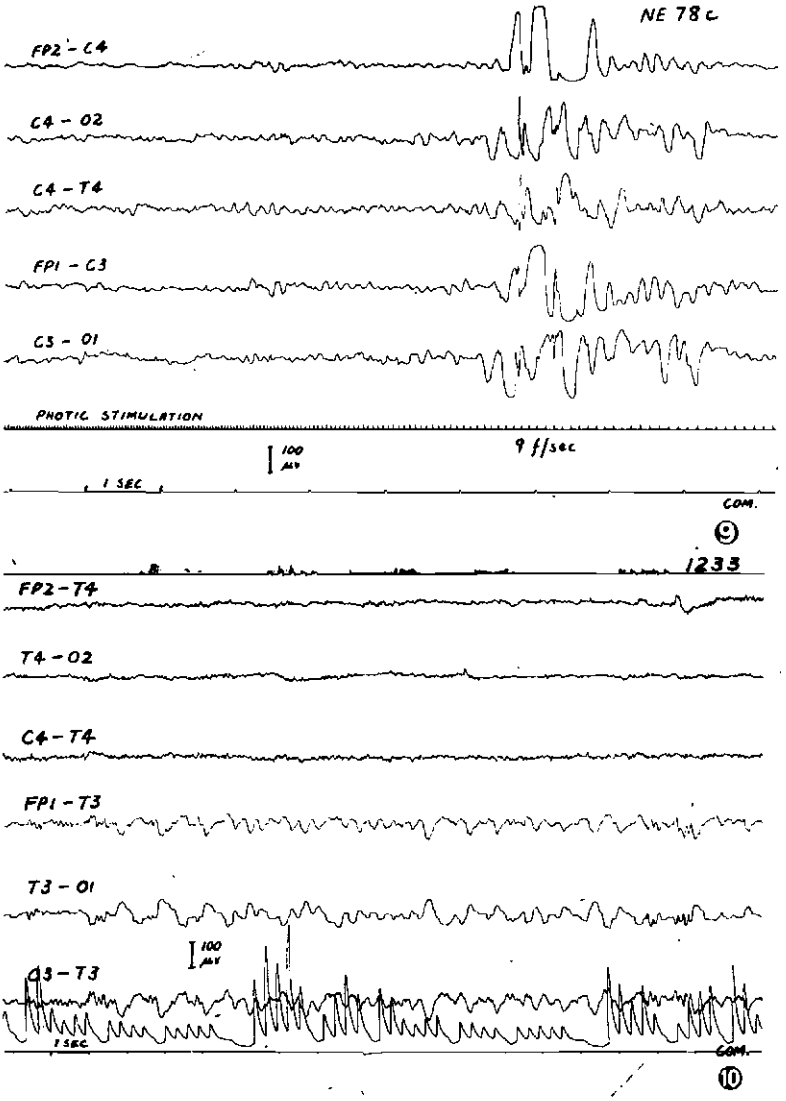

Fig. 9. EEG in Aphasia: Case 42 (see text). Photic stimulation at 9 flashes per second evokes high-voltage theta and delta burst with spikes.

Fig. 10. EEG in Aphasia: Case 43 (see text): Clear left temporal delta and theta focus. Automatic frequency analyzer shows that dominant activity in channel T3–O1 is at 1·5–6 c/sec.

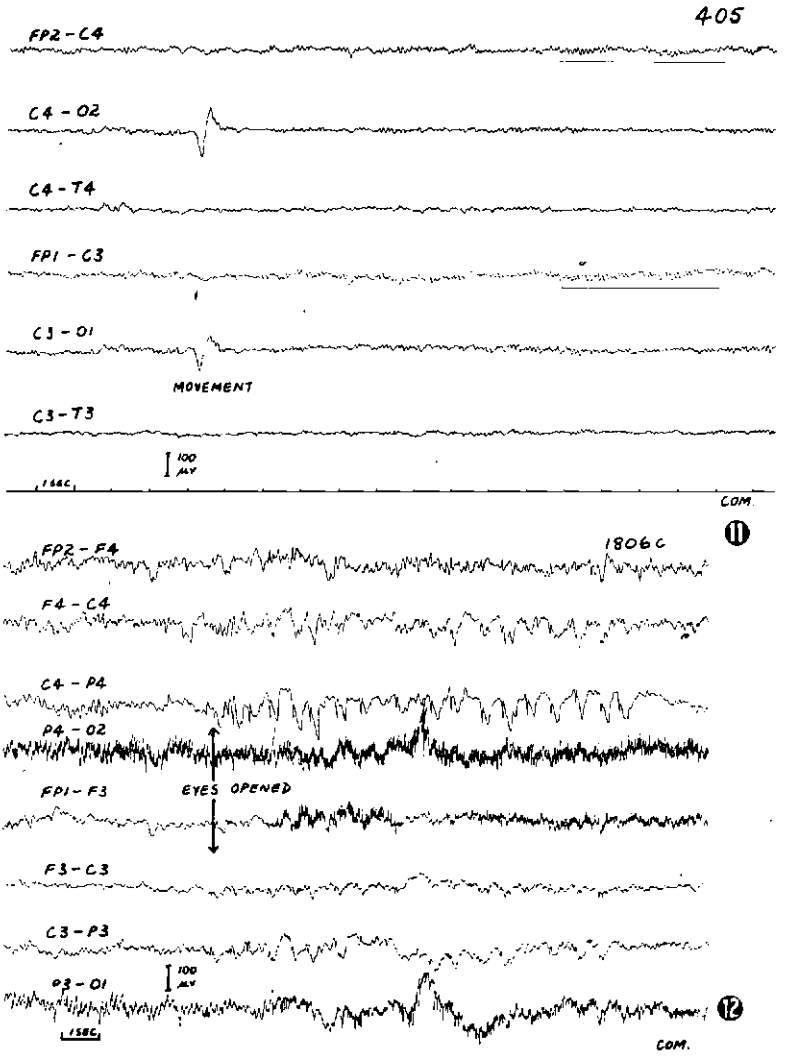

Fig. 11. EEG in Dyslexia: Case 47 (see text): Alpha rhythms more prominent anteriorly than posteriorly (anterior rhythms underlined).

Fig. 12. EEG in Dyslexia: Case 49 (see text): Focal 3–4 c/sec. wave-and-spike activity in the right parietal area on eye-opening.

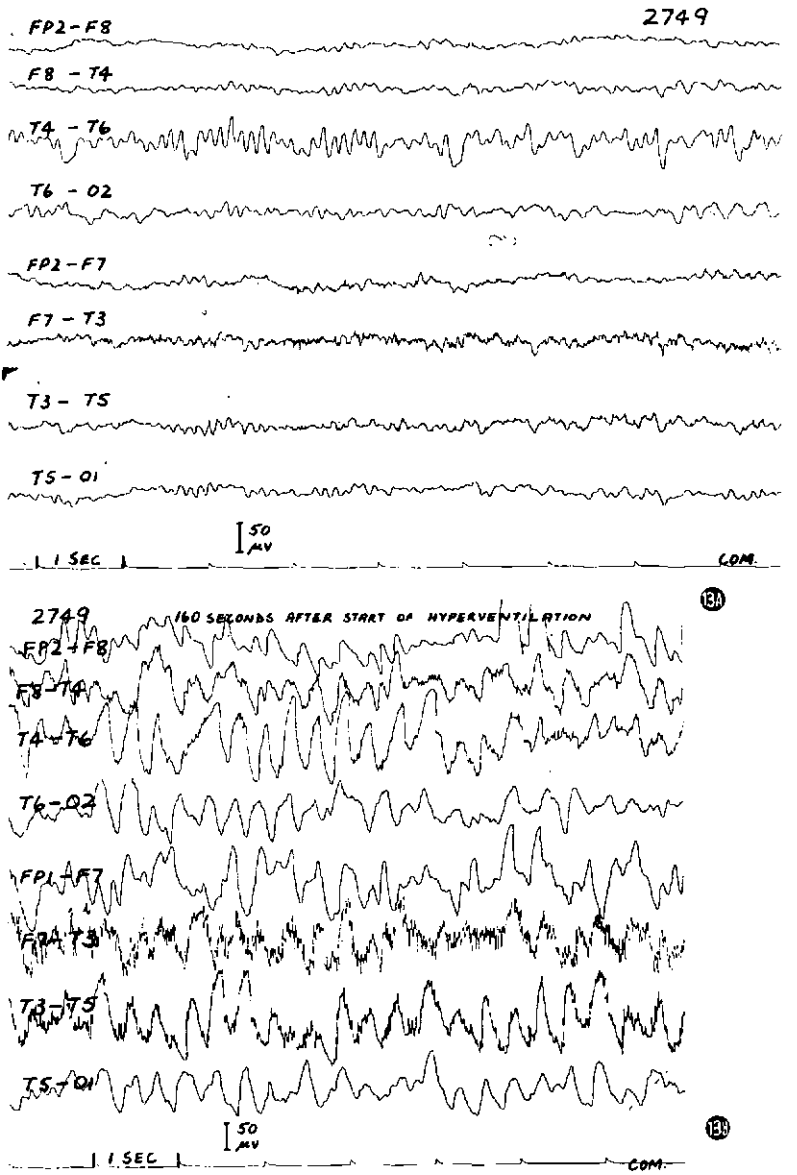

Fig. 13. EEG in Stuttering: Case 52 (see text): Upper sample: right post-temporal delta and theta focus in resting record; lower sample: severe slowing with hyperventilation.

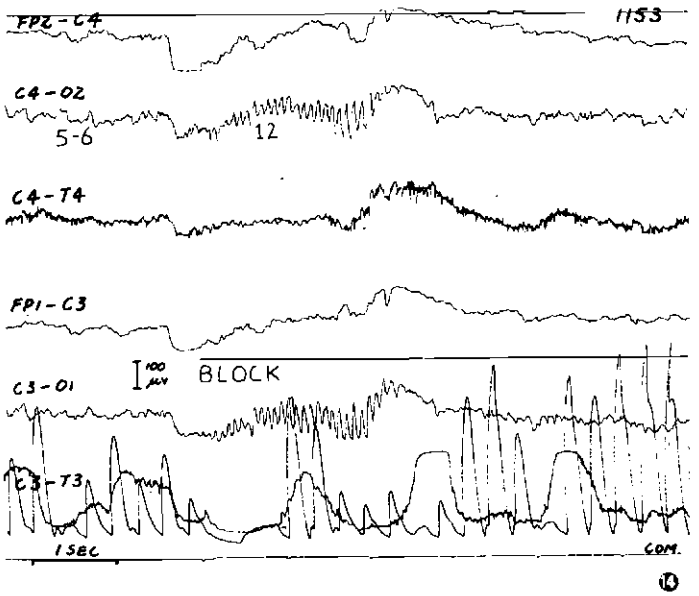

Fig. 14. EEG in Stuttering: Case 54 (see text): Eyes open: burst of 12 c/sec. alpha activity at start of a block during reading. Frequency analyzer trace shows wide range of EEG frequencies (channels C4–O2 analyzed).

TABLE 3: EEG FINDINGS IN SENSORY DISORDERS

Case No.	Age (years)	Sex	History and Clinical Picture	Sensory Defect	EEG	
					Analysis	Interpretation
1	8	F	Premature birth. Squint at birth. Brain damage with optic atrophy	Blind in right eye	Normal	Normal.
2	19	M	Head injury. Brain damage	Attacks of amblyopia	Abnormal: occipital 2–5 c/sec waves	Suggestive of epilepsy
3	19	M	Head injury. Brian damage	Degeneration of left retina	Abnormal: excessive diffuse 5–7 c/sec activity and occasional bursts of slow waves with photic stimulation	Compatible with epilepsy
4	9	F	Premature birth, fell at 13 months. Athetosis and ataxia	Congenital eye disease (unspecified)	Abnormal: excessive left fronto-parieto-temporal 4–6 c/sec activity	Suggestive of localized cerebral abnormality
5	4½	M	Breach birth, pyrexial episode at 14 days. Spastic quadriplegia and mental retardation with recent "blackouts"	Right-sided optic atrophy	Abnormal: numerous high-voltage 1.5–5 c/sec disturbances with sharp waves, maximal left fronto-parietal region	Suggestive of localized cerebral abnormality
6	9	F	Two "fainting spells"—right hemiplegia	Almost blind right eye	Severely abnormal: prolonged runs focal sharptipped 4–6 c/sec waves left parietal area, with occasional wave-and-spike episodes (Fig. 4)	Suggestive of left parietal epileptogenic abnormality
7	9	M	Premature birth, survivor of twins. Right hemiplegia, agraphia	Partly blind right eye	Normal	Normal
8	+25	F	Two epileptic seizures, 3 and 2 months previously. Retro-orbital tumour with epilepsy	Paralysis left abducens, with some involvement of rectus internus	Abnormal: excessive occipital 4–5 c/sec activity with rare generalized medium-voltage ½ second waves	Compatible with epilepsy—no focal signs
9	7	M	Frequent grand mal seizures—epilepsy	Visual defect	Abnormal: clear focus abnormal activity right frontal area (Fig. 5)	Suggestive of epileptogenic abnormality in right frontal area
10	10	M	Cyanosis at birth; ? petit mal attacks. Physical and mental retardation, epilepsy	Slight amblyopia	Abnormal: focal slow activity in right parieto-temporo-occipital area	Compatible with epilepsy, cortical maturation defect or both

TABLE 3: EEG FINDINGS IN SENSORY DISORDERS—*Continued*

Case No.	*Age (years)*	*Sex*	*History and Clinical Picture*	*Sensory Defect*	*EEG*	
					Analysis	*Interpretation*
11	5	M	Lack of tears, corneal anaesthesia. Riley's disease (familial dysautonomia)	Amblyopia	Normal	Normal
12	12	M		Complicated motor defect involving left eye	Abnormal: considerable occipital 14–18 c/sec activity	Unusual: occipital beta apparently performed function of the alpha rhythm
13	10	M	Surgery for pituitary tumour at 5 and 6½ years. Poor concentration and memory	Blind in left eye	Severely abnormal: focus of 1.5–3 c/sec waves and waves-and-spikes, right parieto-temporo-occipital area	Suggestive of localized brain damage with associated epileptic disorder
14	62	F	Corneal graft. Diabetes—right parietal haemangioma or glioma	Almost blind	Abnormal: right parietal slow wave focus	Suggestive of organic abnormality in right parietal area
15	14	F	Normal	Right amblyopia	Normal, but electroretinogram abnormal (see text and Fig. 6)	Peripheral (retinal) abnormality
16	11	F	Bad temper, "lack of desire to learn", fluctuating ability to speak—intelligent. Delayed speech	Deafness	Abnormal: excessive posterior delta, sometimes focal left parietal area	Raised possibility of organic abnormality left parieto-temporal area (see text)
17	3	M	Cerebral palsy. Delayed speech and development	Apparently deaf	Severely abnormal: high voltage left frontal spikes. (Also given "hearing EEG"—see text)	Suggestive of epileptogenic abnormality in left frontal region

TABLE 4: EEG FINDINGS IN CHILDREN WITH DELAYED SPEECH

Case No.	*Age (years)*	*Sex*	*Historical and Clinical Picture*	*EEG*	
				Analysis	*Interpretation*
18	$1\frac{8}{12}$	M	Quadriplegic—Pyramidal tract lesion	Abnormal: asynchronous frontal and temporal delta	Suggestive of dysfunction of fronto-temporal areas
19	4	F	Right hemiparesis	Abnormal: delta focus right post-temporal area	Suggestive of right post-temporal lesion
20	7	M	Spastic quadriplegia	Abnormal: delta focus right fronto-centro-temporal area	Suggestive of right fronto-centro-temporal lesion
21	5	F	Spastic quadriplegia	Abnormal: spike and delta wave focus right parietal area	Suggestive of right parietal epileptogenic abnormality
22	$7\frac{9}{12}$	F	Mentally retarded, fluctuating school performance	Abnormal: excessive fronto-central theta, and occasional bursts of sinusoidal 4 c/sec waves (Fig. 7)	Cortical immaturity with epilepsy
23	7	M	Physically and mentally retarded	Normal	
24	$6\frac{9}{12}$	M	"Nervous"	Normal	
25	$6\frac{9}{12}$	F	Borderline mental defective	Abnormal: excessive occipital delta and occasional right parietal theta	Cortical maturation defect
26	$5\frac{11}{12}$	F	Jaundice at birth—deterioration in I.Q.	Normal:	
27	$7\frac{6}{12}$	F	"Fits" associated with emotion	Mildly Abnormal: occasional left temporal theta and beta	Suggestive of left temporal abnormality, ? epileptic
28	10	M	Somnambulism—retarded	Abnormal: asynchronous bilateral post-temporal theta and spikes	Suggestive of epilepsy
29	8	M	Brain damage	Abnormal: excessive fronto-centro-temporal theta and delta	Severe cortical maturation defect
30	$10\frac{6}{12}$	F	Brain damage	Abnormal: excessive occipital theta	Cortical maturation defect

TABLE 4: EEG FINDINGS IN CHILDREN WITH DELAYED SPEECH—*Continued*

Case No.	*Age (years)*	*Sex*	*Historical and clinical Picture*	*EEG*	
				Analysis	*Interpretation*
31	$2\frac{8}{12}$	F	Post-meningitis, no speech	Normal	
32	2	F	Spastic quadriparesis	Abnormal: focal right parieto-occipital theta	Left parieto-occipital abnormality
	$3\frac{7}{12}$			Severely abnormal: right parieto-temporo-occipital spikes	Right parieto-temporo-occipital epileptogenic abnormality
33	$7\frac{11}{12}$	M	Mild athetoid cerebral palsy	Abnormal: excessive delta and theta	Severe cortical immaturity.
34	2	M	Spastic quadriplegia—no speech	Normal	
35	6	M	Difficult birth—uncle had "fits"	Abnormal: excessive delta and theta	Cortical immaturity
	$6\frac{9}{12}$			Abnormal: unchanged	Cortical immaturity
	7			Abnormal: slight increase in dominant frequency	Cortical immaturity
36	2	M	Falls frequently—no speech	Abnormal: low voltage irregular activity, with delta maximum left frontal area	Cortical agenesis?
	$2\frac{9}{12}$			Abnormal: suppression of sleep spindles on right, runs and bursts of theta and delta right fronto-parietal	Right fronto-parietal abnormality
37	9	F	Difficult birth	Abnormal: excessive delta and theta, maximal left parieto-temporo-occipital area	Organic abnormality left parieto-temporo-occipital
38	7	M	Mental retardation	Abnormal: excessive diffuse theta	Cortical immaturity