SLOWING OF THE ALPHA-RHYTHM OF THE ELECTRO-ENCEPHALOGRAM AND ITS ASSOCIATION WITH MENTAL DETERIORATION AND EPILEPSY.*

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INTRODUCTION.

THERE are few studies relating electroencephalographic characters with mental deterioration. Romano and Engel (1) (2) made an EEG and clinical study of "delirium" which they defined as the "toxic-infective state or psychosis associated with somatic disease" which was accompanied by "fluctuation in the level of awareness" and "intellectual, emotional and motor regressive behaviour"; the group included cases of chronic and acute cardiac decompensation, chronic alcoholism, hypertensive states, and severe Addison's disease. These authors considered that the EEG changes could be graded into three main groups, in order of severity: firstly, "decrease in frequency," with increase of slow 4–7 frequencies and normal blocking responses to visual attention; next, "disorganization," with irregular slow 2–7 frequencies and poor blocking responses; and finally, "reorganization at a lower energy level," with moderately high voltage slow 3–7 c./sec. activity, few or no normal frequencies, and absent blocking response to visual attention. This latter stage was, in some cases, found to be irreversible. Previously, Engel and Margolin (3) had published the results of an investigation into the effect of both oxygen and glucose-deprivation on the human electro-encephalogram. Both these conditions produced bursts of diffuse generalized 3–6 c./sec. activity which, in those cases with an acute onset, reverted to normal rhythms as clinical improvement occurred. However, 4 patients with chronic cerebral anoxaemia were examined (end-stages of chronic bronchitis with right-sided heart-failure) in whom the EEG abnormalities persisted; at an early stage, activity with a frequency of 5–6 c./sec. appeared, and still later the records became completely disorganized. There was one patient who ultimately recovered whose EEG showed a regular dominant alpha rhythm with a frequency of 7·5 to 8 c./sec. It had already been noticed that, in a few cases where the EEG had apparently returned to normal in prolonged anoxic and hypoglycaemic cases, there yet remained some degree of intellectual deficit on psychological testing. Hill (4) had observed that a type of EEG with a

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slower dominant alpha-rhythm than the normal 8–13 c./sec. (i.e. a rhythm at 6–8 c./sec.) was frequently associated with mental deterioration. An investigation was accordingly instituted to determine to what degree mental deterioration and slowing of the dominant rhythm in the post-central areas (alpha-rhythm) were associated.

Clinical Material and Method of Study.

A preliminary study of 1,500 consecutive EEG's at the Maudsley Hospital and 1,000 at the National Hospital for Nervous Diseases, a total of 2,500 in all, was undertaken, and amongst these were found 20 cases with a slowing of the alpha-rhythm. Since the Maudsley Hospital is predominantly a psychiatric hospital and the National Hospital is predominantly neurological, it was to be expected that organic cerebral disease, and hence irreversible EEG changes, would be more commonly found in the clinical material at the latter hospital. This was indeed the case, since only 5 of the 20 were Maudsley cases, despite the fact that 50 per cent. more EEG's had been examined at that hospital. Of these 5 patients, 3 were epileptics, 1 showed a post-encephalitic syndrome, and 1 patient suffered from multiple tics and had an early history of " chorea." The clinical notes of the 20 cases with slow alpha-rhythm were reviewed to determine the presence or absence of a degree of mental deterioration detectable on ordinary routine clinical examination. Finally, the 1,000 cases already investigated at the National Hospital (less the 15 cases with slow alpha-rhythm) were further reviewed to find those which had demonstrated mental deterioration—in other words, patients who were so dull and deteriorated that they were found to be obviously so during an ordinary neurological examination. These numbered 15. This second group of cases was then studied to determine how far mental deterioration was associated with EEG abnormalities other than the picture already found with the slowing of the alpha-rhythm. For purposes of comparison these two groups have been separated into Group I (Tables I and II) and Group II (Tables IV and VI).

The EEG's of patients under 20 years of age were excluded, since it was less likely that constitutional immaturity of the EEG would be present above that age which would invalidate the findings. Age and sex were taken into consideration to determine any possible abnormal distribution in relation to these factors. Since many cases were found to be epileptic, the severity of the disorder, where present, was evaluated and a family history of epilepsy was sought. Trauma to the brain may produce mental deterioration and EEG changes, and consequently such an association was investigated ; finally, factors which of themselves might modify the EEG, such as organic nervous disease, cerebral arteriosclerosis and increased intracranial pressure, were sought.

In the EEG records the frequency, amplitude and extent of the dominant postcentral rhythm were observed, as well as the response to visual stimulation. Other rhythms, fast and slow, were tabulated together with their respective locations ; and finally, voluntary overbreathing for 3 minutes was done (so far as the mental condition would allow) to bring out any latent abnormality.

TABLE I.—*EEG Findings in Preliminary Group of*

Case No.	Alpha-rhythm. Frequency. (c./s.)	Alpha-rhythm. Amplitude. (μv.)	Alpha-rhythm. Percentage.	Area of alpha-rhythm. Occipital.	Area of alpha-rhythm. Forward spread.	Area of alpha-rhythm. Including frontal.	Blocking.	Other rhythms: fast. (c./s.)
I	7–8	50	<50	+	+	−	Poor: variable	22
II	6–8	30–50	50	+	+	−	,, ,,	—
III	7–8	50	>50	+	+	+	Fair: variable	—
IV	6–8	50	50–75	+	+	+	Poor	—
V	7–8	25	30–40	+	+	−	Nil	—
VI	6–8	40–60	50–75	+	+	+	Poor: variable	—
VII	7	50	50–75	+	+	+	Fair	—
VIII	6–7	50–60	50	+	+	+	Nil	22
IX	8	60	75	+	+	+	,,	16–22
X	6–8	50	75	+	+	+	,,	—
XI	6–8	50–70	75	+	+	+	,,	16–18
XII	6–8	50	50–75	+	+	+	Fair: variable	—
XIII	7–8	50	>75	+	+	+	,, ,,	22
XIV	7–8	60	>50	+	+	+	Poor: variable	—
XV	6–7	40–50	75	+	+	+	Nil	—
XVI	7–8	50	50	+	+	+	,,	16–22
XVII	5–7	60	50	+	+	+	,,	—
XVIII	7–8	50	<50	+	+	−	,,	—
XIX	7–8	50	50–75	+	+	+	,,	16–22
XX	6–7	50	50–75	+	+	+	Poor: variable	—

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Cases, Presenting with Slowing of Alpha-rhythm.

Other rhythms: slow.	Location of other rhythms. Fast.	Location of other rhythms. Slow.	Symmetry.	Response to over-breathing. Stable.	Response to over-breathing. Unstable.
2–6 c./s., 50 μv.	Frontal	Frontal and diffuse	+	Not done	
3–6 c./s., 30–50 μv.	—	Diffuse: Postcentral > precentral	Right > left	+	—
2–6 c./s., 50 μv.	—	Diffuse: frontal +	Fair	—	Slight
3–4 c./s., 50 μv.	—	Diffuse: temporal +	+	+	—
2–6 c./s., 70–100 μv.	—	Diffuse: Right > left	Poor: Right > left	+	—
Rare: 3–6 c./s., 50 μv.	—	Diffuse	Fair	+	—
—	—	—	+	Not done	
Occasional, 3–4 c./s.	Fronto-parietal	Diffuse: frontal +	+	+	—
Occasional, 4 c./s.	Diffuse	Occipito-parietal	+	Not done	
3–4 c./s., spike and wave 100 μv.	—	Frontal: right > left, but bilaterally synchronous	Fair	,,	
3–4 c./s., spike and wave 100 μv.	Diffuse frontal	Diffuse	+	,,	
3–6 c./s., 50–70 μv.	—	Occipito-parietal: Left > right	Fair	—	+ (even after glucose)
Occasional, 3–6 c./s., 60–100 μv.	Frontal	Difiuse	,,	—	+ (1 spike and wave)
2–6 c./s., 60–80 μv.	—	,,	,,	+	—
2–6 c./s., spike and wave 100 μv.	—	,,	,,	Not done	
3–6 c./s., 100 μv.	Frontal	Fronto-parietal	+	+, spike and wave complexes	—
2–4 c./s., 60–80 μv.	—	Diffuse	+	+	—
2–6 c./s., 100 μv.	—	,,	Poor	+ (relatively)	—
Occasional, 3–6 c./s., one burst of 3 c./s., 100 μv.	Diffuse: more posteriorly	,,	+	+	—
Occasional, 3–6 c./s., 50–60 μv.	—	,,	+	+	—

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TABLE II.—*Clinical Findings in Preliminary Group*

Case No.	Age.	Sex.	Fits.	Duration of symptoms.	Diagnosis.	Family history of epilepsy.	Head trauma.	General systemic disease.
I	66	F.	−	4 years	Presenile dementia	Positive	Nil	Nil
II	59	M.	+	8-9	Ditto	Negative	,,	(1) Old T.B. lungs (2) B.P.—180/105 (3) Rheumatism
III	41	M.	+	4 months	Cortical atrophy	,,	,,	Nil
IV	65	F.	+ (1 fit only at onset)	3 years	Cerebral arterio-sclerosis	,,	,,	(1) *Hypertension*— B.P. 170/95 (2) *Heart* — Aortic systolic (3) Retinal arterio-sclerosis
V	60	F.	+	3 ,,	Hypertensive encephalopathy	,,	Minor H.I. 1940: short period of unconsciousness	(1) *Hypertension*— B.P. 240/100 (2) *Heart* — enlarged to left. Aortic second sound increased
VI	44	F.	−	2 ,,	Cerebral syphilis	,,	Nil	Optic atrophy
VII	56	M.	−	44 ,,	Multiple tics	,,	Head injury aged 10 years	Nil
VIII	23	M.	−	5 ,,	Post-encephalitic syndrome	,,	Nil	,,
IX	44	M.	+	Indefinite	Epilepsy with hysteria	Negative	Head injury 1944	Nil
X	25	M.	+	23 years	Epilepsy	,,	Head injury preceded fits; fell on stone floor—unconscious	,,
XI	28	M.	+	19 ,,	,,	,,	Nil	,, B.P. 130/98
XII	21	M.	+	13 ,,	,,	,,	Head injury aged 6 years	Nil
XIII	43	M.	+	17 ,,	,,	,,	Nil	,,
XIV	25	F.	+	22 ,,	,,	,,	Fall on forehead preceded first attack	,,
XV	22	F.	+	15 ,,	,,	,,	Nil	,,
XVI	41	M.	+	25 ,,	,,	,,	,,	,,
XVII	25	F.	+	13 ,,	,,	,,	,,	,,
XVIII	28	F.	+	11 ,,	,,	,,	,,	,,
XIX	48	M.	+	8 ,,	,,	,,	,,	
XX	25	F	+	1 year	,,	,,	,,	,,

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of Cases, Presenting with Slowing of Alpha-rhythm.

Mental deterioration.	Personality deterioration.	Neurological signs.	Raised intra-cranial pressure.	Special investigations.	Past illnesses.
Gross dementia (Shipley-Hartford)	Slowness and dullness	Nil	Absent	(1) *Pneumoencephalogram* —gross cortical atrophy (2) *X-ray skull*—N.A.D. (3) *X-ray chest*—N.A.D. (4) *C.S.F.*—normal	"Nervous breakdown" aged 46 years—lasted 6 months.
Gross dementia (simple testing including Babcock)	Slowness and dullness	Weakness and tremor of left side of body in fits	,,	(1) *X-ray chest*—old T.B. (2) *C.S.F.*—normal (3) *Pneumoencephalogram* —General dilatation of ventricles; enlargement of right parietal region	(1) Rheumatism aged 13 years. (2) Deafness right ear; long duration.
Gross dementia (simple testing including Babcock)	Nil gross	Slight left facial weakness	,,	(1) *C.S.F.*—normal (2) *X-ray skull*—N.A.D. (3) *Pneumoencephalogram* —more air in left than in right lateral ventricle. Suspicious shift of third ventricle to left (4) *Operation* — cortical atrophy only (5) *Fundi*—colloid body formations	Neurasthenia age 27 years: tendency to drop things, tiredness —lasted one month, nil since.
Moderate	Slowness	(1) Dysarthria (2) Left facial weakness (3) Hearing — bilaterally affected (4) Left pyramidal lesion. Left paresis and tremor (5) Conjugate movement of eyes to right impaired	,,	(1) *X-ray skull*—N.A.D. (2) *X-ray chest*—N.A.D.	Hypertension 3 years.
Difficult to estimate owing to deafness	Dull and slow	Nil	,,	(1) *X-ray skull*—N.A.D. (2) *Deafness*	(1) Neurasthenia 1919. (2) Bilateral mastoids.
Memory poor: orientation poor	Dullness	(1) Optic atrophy (2) Right anosmia (3) Numbness left	,,	(1) *C.S.F.* (Lange)—mild. Continuous. No comas (2) *Blood W.R.*—positive	Nil relevant.
Slight	Nil gross	Nil	,,	(1) *I.Q.*—low average (2) *Blood W.R.*—negative (3) *E.S.R.*—normal	Chorea aged 12 years.
Retardation mental and motor	Withdrawn obsessive thinking	,,	,,	(1) *Blood W.R.*—negative (2) *E.S.R.*—normal (3) *C.S.F.*—normal	Illness started as "nervous trouble."
Moderate	Psychopathic personality	(1) Slurred speech (2) Tremors (3) Minor fits	Absent	*I.Q.*—low average	? Encephalitis 1941.
Slight	Nil gross	(1) Right facial paresis: In fit head goes to right and right side of mouth is drawn up	,,	Nil	Nil relevant.
,,	,,	Nil	,,	(1) *X-ray skull*—N.A.D.	Recent psychomotor attacks.
Memory only fair: mental deterioration	Slowness	,,	,,	(1) *I.Q.*—below average (2) *C.S.F.*—normal (3) *X-ray skull* — slight oxycephaly. Suggestion of early cerebral atrophy	Nil relevant.
Mental slowness	Dull	,,	,,	Nil	,,
Very backward	Dull and mentally slow	Fits show activity on left side of body	,,	(1) *Blood W.R.*—negative (2) *X-ray skull* — strong suggestion of venous angioma left frontal and fronto-temporal areas	,,
Mental slowness	Dull	Nil	,,	*X-ray skull*—N.A.D.	Convulsions at age of 7 months.
Very poor memory and concentration	Irritable	,,	,,	*Psychological testing* shows organic deterioration	Nil relevant.
Poor concentration, 1944, but reported bright 29.ix.45	Moody and difficult 1944. Episodic changes	(1) No abnormal physical signs (2) Fits start left leg; consciousness lost as sensory phenomena reach up to knee	,,	(1) *X-ray skull*—N.A.D. (2) *C.S.F.*—normal	,,
Memory and concentration poor	Recent deterioration—dull and slow	Nil	,,	Nil	,,
Nil gross: Earning £650 per annum as commercial artist	Nil gross	,,	,,	*Blood W.R.*—negative	*Backache* November, 1943, and March, 1944.
Memory fair	Dull with temper outbursts	,,	,,	(1) *X-ray skull*—N.A.D. (2) *Blood W.R.*—negative	Nil relevant.

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A 3-channel Grass apparatus was used for recording, with standard bipolar electrode-placements in antero-posterior and transverse positions; the duration of the recording was 25–30 minutes. The blood-sugar level at the time of testing was not estimated, but subjects were examined after fasting for more than 4 hours in most cases.

Results.

Group I.

This was the group that was chosen primarily because of slowing of the alpha-rhythm.

EEG findings.—20 EEG's with a slowing of the dominant alpha rhythm were discovered in the 2,500 cases examined at both hospitals; the results of analysis are summarized in Table I.

The dominant alpha-rhythm was in the 6–8 c./sec. range in all cases except in one in which it was 5–7 c./sec.; the amplitude was mainly in the range of 50–60 μv., only 3 being below this; the per cent-time alpha was characteristically 50–75 per cent. Forward spread of the alpha-rhythm occurred in all cases, and failed to reach the frontal region in only 4 cases. The response to visual stimulation was characteristically absent or defective; half the cases did not respond at all and, of the other 10, 8 gave a variable response, in that blocking in some part of the records was better than in others, with a general tendency for the response to be poor earlier and better later in the recording. Fast rhythms in the 16–22 c./sec. range were present in 7 cases, mainly in the frontal areas, and slow rhythms with a frequency of 2–7 c./sec. were present in all cases except one. The slow rhythms showed a tendency to appear in bursts, were of high voltage in 15 of the 20 EEG's, and in 2, characteristic spike-and-wave discharges occurred; moreover, the slow activity was diffuse in all areas, with maximal location in the frontal regions, except in 2 cases where the main site of appearance was in the occipito-parietal areas. Symmetry of frequency and amplitude was good in 10 cases, fair in 7, and poor in only 3. Hyperventilation could only be performed in 14 of the 20 cases, and of these, 11 were stable; one epileptic produced a spike-and-wave response after a previously stable record. A tendency was noted for bursts of activity to appear, during which the fast rhythms doubled and then halved in frequency. The slow waves also often appeared in bursts with, frequently, fast waves superimposed upon them.

To summarize, a fairly characteristic EEG picture within this group can therefore be described associated with the slowing of the dominant alpha rhythm. It presents the following features:

A dominant occipital rhythm of 6–8 c./sec., having an amplitude of 50–60 μv., being present 50–75 per cent. of the time and spreading forward to the frontal regions, but showing poor response to visual stimulation. Slow rhythms are mainly located in the frontal areas, which show fair to good symmetry.

Clinical findings.—The clinical data in this group are summarized in Table II. The diagnoses show a preponderance of epileptic cases (Table III).

TABLE III.—*Diagnostic Classification of Cases in Group I.*

Epilepsy	12 cases.
Arteriosclerotic dementia	3 ,,
Postencephalitic syndrome	1 case.
Cerebral syphilis	1 ,,
Presenile dementia	1 ,,
Cortical atrophy	1 ,,
Multiple tics	1 ,,
Total	20 cases.

Cerebral dysfunction, in the form of idiopathic epilepsy or organic cerebral disease, was present in all the cases with slowing of the alpha-rhythm. Examination of out-patient and in-patient clinical records revealed mental deterioration of such a degree as to be obvious to ordinary clinical examination and simple psychological testing (memory-testing, serial sevens test and general knowledge) in all cases except one, who was holding down a job as a commercial artist. Unfortunately the patient could not be followed up and his intelligence could not be tested. One case of epilepsy presented as being dull on one occasion and brighter on a subsequent visit; again this patient was not subjected to psychological testing. The clinical records showed that one can certainly associate mental deterioration in this group with slowing of the alpha-rhythm; though the association is not an absolute one, it was present in at least 18 of the 20 cases. Of equal significance is the finding that fits were present in 16 of the 20 cases; 4 of these were organic cerebral states, but in all of them fits were infrequent, in contradistinction to the idiopathic epileptic group. Personality changes were marked in 13 of the 20 patients and consisted mainly of dullness and withdrawal, but 5 of the 13 showed in addition the excessive emotional instability of the patient with organic cerebral disorder. Clinical signs of impaired function in the central nervous system were definitely present in 8 of the 20 cases, in 3 supported by pneumoencephalographic studies. A family history of epilepsy was obtained in only one case. Head-trauma was a negligible factor. The duration of illness was variable, though all the epileptic cases were of long standing except one; the onset of dullness was generally a gradual one. Sex of the patient was not a factor in aetiology, and a study of age revealed nothing significant except that epilepsy occurred in the lower age-groups.

Group II.

This group of 15 cases, selected primarily because of the presence of mental deterioration, was found in the 985 cases remaining at the National Hospital after the 15 with slow alpha-rhythm had been examined.

EEG findings.—These are summarized in Table VI. The same factors as in Group I were investigated, but no characteristic EEG's were found, although 11 of the cases showed some abnormality. The dominant frequencies in the post-central areas were within the normal range of alpha rhythm in all cases

TABLE IV.—*Clinical Findings in Second Group*

Case No.	Age.	Sex.	Fits.	Duration of symptoms.	Diagnosis.	Family history of epilepsy.	Head trauma.	General systemic disease.
I	59	M.	—	5 years	Alcoholic dementia	Negative	Nil	Nil
II	61	M.	—	1½ „	Hypertensive encephalopathy	„	„	(1) *Heart*—hypertensive (2) *B.P.*—240/135
III	31	F.	—	15 „	Post-encephalitic Parkinsonism	„	„	Nil
IV	60	M.	+	15 „	Hypertensive encephalopathy	„	„	(1) *B.P.*—210/120 (2) *Heart*—hypertensive (3) *Fundi*—arterio-sclerot (4) Recent confusional ep sode
V	75	M.	—	1 year	Cerebral arterio-sclerosis	„	„	(1) *B.P.*—130/90 (2) *Cataracts*
VI	48	M.	—	3 years	Presenile dementia	„	„	(1) *B.P.*—120/80
VII	61	M.	+	2 „	Cortical atrophy	„	„	Nil
VIII	46	F.	+	2 „	„ „	„	„	„
IX	52	M.	+	6 „	Presenile dementia	„	„	(1) General arterio-sclero (2) *B.P.*—140/80
X	53	M.	+	23 „	Epilepsy with mental deterioration	„	„	(1) *Fundi*—arterio-sclerot (2) *B.P.*—160/95
XI	44	M.	—	7 „	Post-encephalitic Parkinsonism	„	„	Nil
XII	49	M.	+	6 „	Epilepsy with mental deterioration	„	„	„
XIII	60	F.	+	2 „	Cortical atrophy	„	„	„
XIV	62	F.	+	18 months	„ „	„	„	(1) *Cardio-vascular syste* —normal (2) *B.P.*—140/90
XV	56	F.	+	2 years	Presenile dementia	„	„	(1) *Cardio-vascular syste* —normal (2) *B.P.*—120/80

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Cases Presenting Primarily with Mental Deterioration.

Mental deterioration.	Personality deterioration.	Neurological signs.	Raised intra-cranial pressure.	Special investigations.	Past illnesses.
Dementia	Apathetic	Nil	+ (?)	(1) *X-ray skull*—? suggestive of increased intra-cranial pressure (2) *C.S.F.*—normal	Nil relevant.
Dullness and memory failure	Depression	(1) Early pseudo-bulbar palsy (2) Spasticity right arm	Absent	(1) *X-ray skull*—bilateral mild hyperostosis frontalis interna (2) Encephalography—moderate general dilatation of ventricles—general cerebral atrophy	,,
Dullness, slowness and poor memory—5 years	Depressive attacks	(1) Tremor right arm (2) Increased salivation (3) Oculogyric crises (4) Cogwheel rigidity	,,	Nil	Encephalitis 15 years ago.
Memory and judgment poor (improving)	Confused	Speech difficulty	,,	(1) *C.S.F.*—normal (2) *Kidney function*—good (3) *Blood urea*—35 mgm. % (4) *X-ray skull*—N.A.D.	Nil relevant.
Dementia	Apathetic	Slight left hemiparesis	,,	(1) *C.S.F.*—normal (2) *Fundi*—arterio-sclerotic	,,
,,	Facile and euphoric	(1) Ataxia (2) Diplopia (3) Coarse nystagmus (4) Plantars extensor	,,	(1) *C.S.F.*—normal (2) *Encephalography*—dilatation of all ventricles with cortical atrophy ++ in frontal region	,,
Memory failure	Emotional instability	Weakness right arm, right lower face, and right side tongue	,,	(1) *C.S.F.*—normal (2) *Ventriculography*—enlarged right lateral ventricle with slight displacement of ventricles to right; suggests atrophic process right hemisphere	,,
Poor memory	Dullness	(1) Difficulty in swallowing (2) Right leg slightly weak after fits (3) Nominal aphasia	,,	(1) *C.S.F.*—normal (2) *Encephalography*—slight dilatation of left ventricular system as a whole. Abnormal amount of air over cortex	,,
,,	Dullness and apathy	(1) Slight tremor: left, right (2) Right pupil: left (3) Slight right facial weakness	,,	(1) *X-ray skull*—N.A.D. (2) *C.S.F.*—normal	,,
Dementia (recent)	Ditto	(1) Early bilateral grasp reflexes (2) ? Left extensor response (3) Tremors of legs	,,	(1) *C.S.F.*—normal (2) *Blood count*—normal (3) *X-ray skull*—normal	,,
Gross memory impairment and orientation poor	Dull and seclusive	(1) Choreo-athetotic movements (2) Cogwheel rigidity	,,	*Blood W.R.*—negative	Started with infective illness (some weeks).
Poor memory	Dullness	Nil	,,	,, ,,	Nil relevant.
Gross dementia	,,	,,	,,	,, ,,	,,
,,	,,	(1) Tremor of hands (2) ? Left extensor response	,,	,, ,,	,,
	,,	Nil	,,	,, ,,	,,

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except 3, in which no dominant frequency at all could be found; one case showed a picture not unlike the Group I EEG, but at a steady frequency of 8 c./sec. Forward spread of alpha-rhythm and response to visual stimulation were less definite than in Group I and per cent-time alpha was low, an observation which was consistent with the presence of slow 2–7 c./sec. rhythms in as many as 12 of the 15 cases, these rhythms being generalized and with maximal incidence in the frontal regions. There was no marked difference between Groups I and II in relation to fast rhythms, symmetry, or response to hyperventilation.

Clinical findings.—The clinical data in this group are summarized in Table IV, and diagnostic classification is presented in Table V.

TABLE V.—*Diagnostic Classification of Cases in Group II.*

Cortical atrophy	4 cases.
Presenile dementia	3 ,,
Post-encephalitic syndrome . .	2 ,,
Hypertensive encephalopathy . .	2 ,,
Epileptic dementia	2 ,,
Cerebral arteriosclerosis . . .	1 case.
Alcoholic dementia	1 ,,
Total	15 cases.

Epileptic fits occurred in 9 patients, but in only 2 were these more than incidental to a progressive organic state, in contradistinction to Group I,

TABLE VI.—*EEG Findings in Second Group*

Case No.	Alpha-rhythm. Frequency. (c./s.)	Alpha-rhythm. Amplitude. (μv.)	Alpha-rhythm. Percentage.	Area of Alpha-rhythm. Occipital.	Area of Alpha-rhythm. Forward spread.	Area of Alpha-rhythm. Including frontal.	Blocking.	Other rhythms fast. (c./s.)
I	No dominant rhythm	50	—	—	—	—	?	—
II	10–11	30	25–50	+	+	Slight	Fair	Few,
III	9–10	30	50–75	+	+	+	Poor	—
IV	8–10	40	25–50	+	+	Slight	+	—
V	7–10	50	50+	+	+	+	?	—
VI	8–10	40	50–75	+	+	+	Fair	—
VII	8–9	40	50	+	+	+	+	16
VIII	No dominant rhythm	Very low	--	—	—	—	—	22 ar 11
IX	9–10	50	50	+	+	—	+	—
X	9–10	50	50–75	+	+	+	+	—
XI	No dominant rhythm	20	—	—	—	—		—
XII	8–9	40	50–75	+	+	+	+	—
XIII	8	50	50	+	+	+	Fair	—
XIV	9–11	40	<50	+	+	Slight	Poor	—
XV	10–12	50	25–50	+	+	,,	Fair	22

where 12 of 15 cases with fits were idiopathic epileptics. Personality changes of organic type were present in all (viz. variable apathy, depression, confusion, euphoria and emotional instability). Abnormal signs in the central nervous system were demonstrable in 11 of the 15 patients. The four without such signs were the patient with alcoholic dementia, one of the two patients with epileptic dementia, a patient with cortical atrophy, and one with presenile dementia. Increased intracranial pressure was not found or suspected in any of the patients. Although 10 of the 15 patients were males, the series is too small for this to be of any significance. The time-interval between the onset of the illness and the appearance of mental deterioration could not be assessed; 7 of the histories were of under 2 years' duration. None of the patients gave a family history of epilepsy, nor was there any association with head injury. The relationships between slowing of the alpha frequency, mental deterioration and epilepsy are shown in Table VII.

TABLE VII.

	Mental deterioration.	Epilepsy.	Mental deterioration + epilepsy	No mental deterioration; no epilepsy
Slow alpha . .	3	2	14	1
Normal alpha . .	6	0	9	0

DISCUSSION.

Of the 35 cases of cerebral dysfunction examined mental deterioration was present in 33, and 18 of these showed a slow dominant alpha-rhythm which was associated to a marked degree with established epilepsy. Gibbs and

ases, Presenting Primarily with Mental Deterioration.

Other rhythms: slow. (c./s.)	Location of other rhythms. Fast.	Location of other rhythms. Slow.	Symmetry.	Response to over-breathing. Stable.	Response to over-breathing. Unstable.
2–6	—	Generalized	Fair	Not done	
—	Generalized	—	+	+	—
3–5	—	+ Fronto-parietal	+	Not done	
1–5	—	Generalized (++ right temporal)	Right> left	,,	
3–6	—	Generalized	Fair	,,	
—	—	—	+	+	—
Occasional,	Frontal +	Generalized	Fair	+	—
½–3	Generalized	++ Fronto-temporal	+	+	—
Low amplitude, 4–6	—	Frontal	+	—	+
Few, 4–7	—	,,	+	+	—
Few, 4–6	—	Generalized	+	Not done	
—	—	—	+	+	—
4–6	—	Generalized	+	+	—
2–5	—	Frontal mainly	Fair	—	Moderate
5–7	Generalized parietal +	Generalized	,,	—	+

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Gibbs (5) state that deteriorated epileptics often show predominantly slow waves in the electroencephalogram—an observation which is supported by the present study; slow wave activity was present in the frontal regions in many cases, though it was by no means confined to deteriorated epileptics. Putnam (6) points out that one must guard against misinterpreting the mental dullness, which is really an epileptic equivalent and is an expression of a continuous stretch of cerebral dysrhythmia; but since the cases in this study have been followed up for prolonged periods, this renders such a misinterpretation unlikely. In a comparative study of the value of psychological abstraction tests and EEG in cases of brain damage, Greenblatt *et al.* (7) found that the only group where the EEG was more valuable diagnostically was in those cases where epilepsy was present. Any attempt to explain the phenomenon of the slow alpha-rhythm physiologically would be highly speculative; no special evidence could be obtained pointing to a "reorganization at a lower energy-level" consequent upon a destruction of cortical neurones. What has emerged from the study is that mental deterioration is not specifically associated with slowing of the dominant alpha-rhythm but, when the latter is present, mental deterioration is probable and, moreover, particularly so when the patient has suffered from fits. The important practical application of this is that when one encounters this phenomenon in the EEG of an epileptic one should consider the probability of mental deterioration.

Summary.

In an examination of 2,500 consecutive EEG's 20 were found with slowing of the dominant alpha-rhythm to 6–8 c./sec., and of these 18 had definite mental deterioration.

Further investigation showed that mental deterioration is often associated with EEG abnormality, though not specifically with slowing of the alpha-rhythm.

There is a marked association between slowing of the alpha-rhythm and deterioration in epilepsy.

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