A physiological study of brain activity

——a report on the special-class children's EEG and key reaction time——

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I. Introduction

In the education of the mentally retarded, teachers and parents have to know their children's physical and mental ability or developmental levels as compared with normal children of the same age, in order to bring up the children's potential ability. EEG is an excellent way to provide information roughly about the developmental level of the brain or to predict brain damage by a subclinical seizure discharge. Comparative EEG characteristics of problem juveniles[7], mentally retarded[2,3,4,5,6,8] or other kinds ef exceptional children[10,11,12] have been studied for a long time. As a result some kind of anticonvulsant treatment has been recommended especially for the epileptic, which appears often in these exceptional children. The problem of exceptional children is now a question of social welfare which has to be solved.

Almost every pupil studying in a special-class has his own etiology, combined with each handicap. Proper guidance may be provided for each pupil through an association of parents or teachers who make careful daily observations plus clinical diagnosis confirmed by EEG. With this point of view, pupils' EEGs in the special-class were studied as compared with those in the normal class in collaboration with their teachers. It showed a higher proportion of EEG abnormality than that of the control group, as from the serious patient, who has a severe EEG abnormality and needs integral medical treatment, to one with subclinical seizure discharges. It is necessary to take the EEG into account in order to prevent further progress of mental retardation or to understand the pupils' condition. This is a report of the actual circumstances about the pupils' EEG in the special-classes in Kochi. At the same time their key reaction times as induced by photic stimulation were studied as a means to provide information about their brain activity. The present report includes a quantitative data concerned with it.

II. Experimental subjects and methods

The present experimental subjects were composed of school children in the special-classes of A and B primary schools : fourteen children from 7 to 12 years of age from group A, nineteen children from 9 to 13 years of age from group B respectively. The A primary school is located in the poorest district in Kochi city. In its experimental subjects, 5 were children of families helped by the Livelihood Protection Law. This group included 4 children with the I.Q. of 60−70, 10 with the I.Q. of 70−90. Ten out of fourteen (71%) had some etiologies of brain defect in their infancies or positive hereditary factor. The rest had no etiology of brain damage but had common environment of noninterference, domestic trouble and poverty. On the other hand the B primary school is a representative of enrolling pupils

of good homes. Experimental subjects in B included 7 children with the I.Q. of 50−70, 8 children with the I.Q. of 70−90 and 4 unknown. There were 15 out of 19 (79%) who had some etiology of brain damage or positive hereditary factor. The remainders were composed of those grown up in the uninterference families or orphans who had no information about their infancies.

Control subjects were 55 school children at about the same ages, from 8 to 10 years old. Nine (16%) of them were found to have some etiology of brain defects (Table 1).

A nine channel EEG machine was used in the present experiment with time constant 0.3 sec. Monopolar recording referred to the ears was used with Gibbs' way of electrode distribution: on the frontal, parietal, occipital and temporal areas symmetrically with both cerebral hemispheres.

The recording of EEG were performed in a quiet state, awake with eyes closed. Hyperventilation for three minutes and photic stimulation were used as methods for activation of EEG abnormality. Gibbs' classification of EEG was referred to define its normality. Positive effects of hyperventilation were those build-ups during or after hyperventilation and those developed bursts of high voltage θ waves in the frontal area which appeared only after activation. In photic stimulation, EEG abnormality during or after flicker stimulation, which correspond to Gibbs' photoactivation and photoconvulsive, were observed.

Flicker apparatus, PS-101 type, Sanei Co. was used for photic stimulation in which frequencies, 5−16 Flickers/sec. were applied. For the measurement of key reaction time induced by single photic stimulation, the same methods were applied as those used in the former report.[9]

Table 1. Past histories of special-class children at the A and B primary school with those of the control, common primary school children.

Past history	A	B	Control
Medical history			
Encephalitis	1	2	
Cerebral palsy	2		
Difficult birth	2	1	1
Febrile disease	2	6	1
Jaundice		1	
Bruise	1	1	6
Positive hereditary factor	2	2	1
Positive hereditary factor + Fever or Bruise		2	
Psychological history			
Noninterference, Domestic trouble	4	1	
Negative medical history with brain defect or unknown		3	46
Total	14	19	55
Rate of those with positive medical history to the whole	10/14 (71%)	15/19 (79%)	9/55 (16%)

Febrile disease: autointoxication, acute pneumonia, scarlet fever, etc. in the infancy.
Difficult birth: perinatal injury, premature birth etc.
Bruise: fall from the upstair, traffic accident etc.
Positive hereditary factor: those who have relatives of mental illness or mental retardation.

III. Experimental results :

(a) EEGs of the control

In 55 controls, there were 48 (87 %) normal and 7 (13 %) abnormal EEGs (Table 2). EEG abnormalities were as follows : (1) moderately slow (6 c/s) for age (9 years old) (Fig. $1_{(1)}$) (2) slightly abnormal fast (F-1) (Fig. $1_{(2)}$) (3) diffuse paroxysmal slow (Fig. $1_{(3)}$(a), Fig. $1_{(4)}$(a)). Six of 48 normal control EEGs were different from others in the following points : (1) Appearance of many intermediate fast waves (15−20 c/s) in the frontal area. (2) Asymmetry (20−30 %) in the amplitude of the occipital basic wave. (3) Sporadic high voltage slow waves associated with slight asymmetry in the occipital basic waves. (4) Dominant fast waves (30 c/s) and bursts of high voltage θ waves in the frontal area (Fig. $1_{(5)}$).

As will be discussed later, these EEG abnormalities in the control express a fact that EEG abnormality in normal children is not always correlative with an organic or functional brain defect, especially for children. Diffuse paroxysmal slow waves, which appeared in the normal children, indicates their nature of subclinical epilepsy which sometimes subsides without producing symptome or becomes clinically expressed at a later date. After all, in 87 % the common primary school children had normal EEG.

In control children there were about 21 % of activation by hyperventilation in those who showed normal EEG, including slight, medium, big build-ups or bursts of θ waves

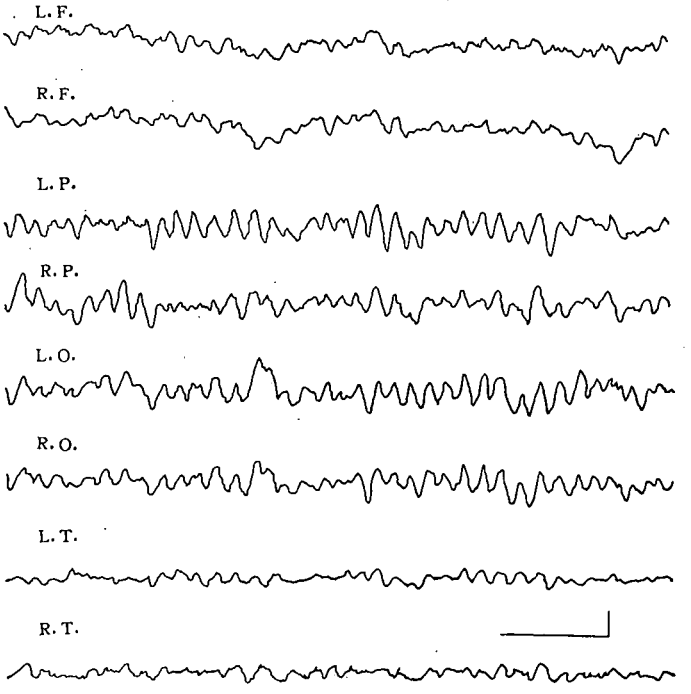

Fig. $1_{(1)}$ Normal boy, age 9 years-awake. Moderately slow waves (6 c/s) in all areas. Vertical and horizontal lines in the right bottom, Fig. 1−3 mean 50 μV and 1 sec. respectively. Abbreviations on the left in each Fig. indicate recording parts on the skull : the frontal (F.), parietal (P.), occipital (O.) and temporal (T.) areas of the left (L.) or right (R.) cerebral hemisphere symmetrically. All figures are retouched.

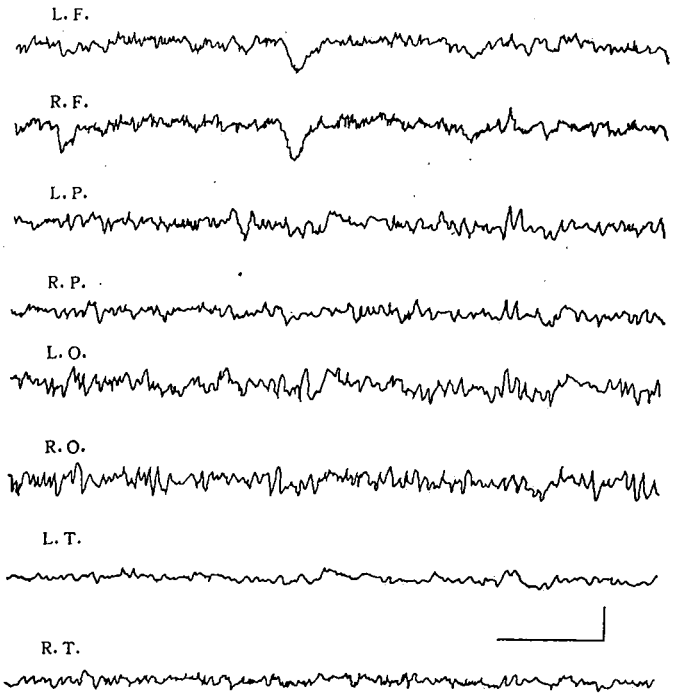

Fig. 1(2) Normal girl, age 9 years-awake. Slightly abnormal fast waves (F−1).

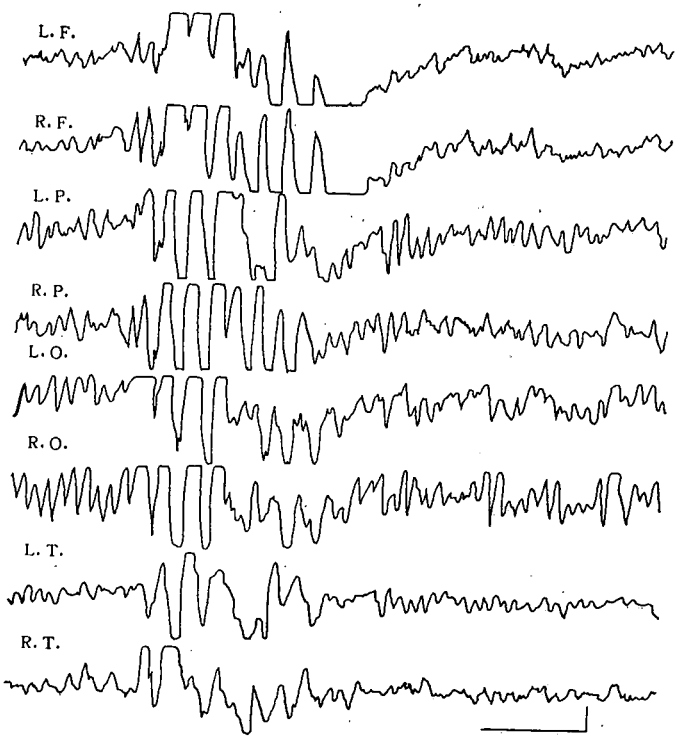

Fig. 1(3)(a) Normal girl, age 8 years-awake. Diffuse paroxysmal slow waves.

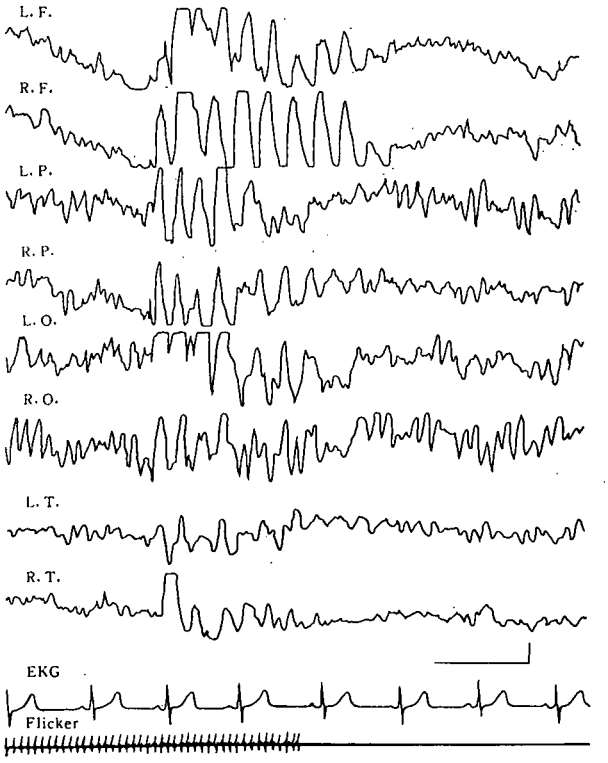

Fig. 1(3)(b) Same subject as in Fig. 1(3)(a). Photoactivation at the end of 14 c/s flicker stimulation for 10 sec.

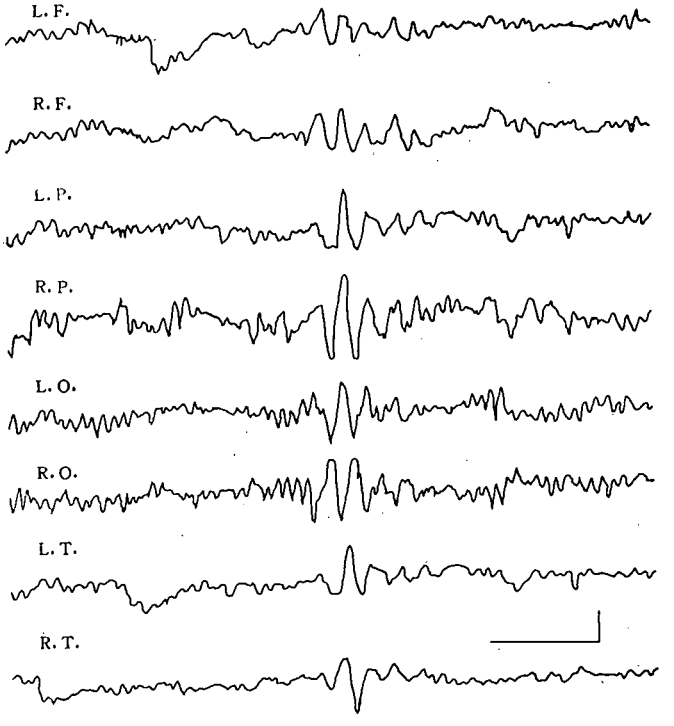

Fig. 1(4)(a) Normal girl, age 9 years-awake. Diffuse paroxysmal slow waves.

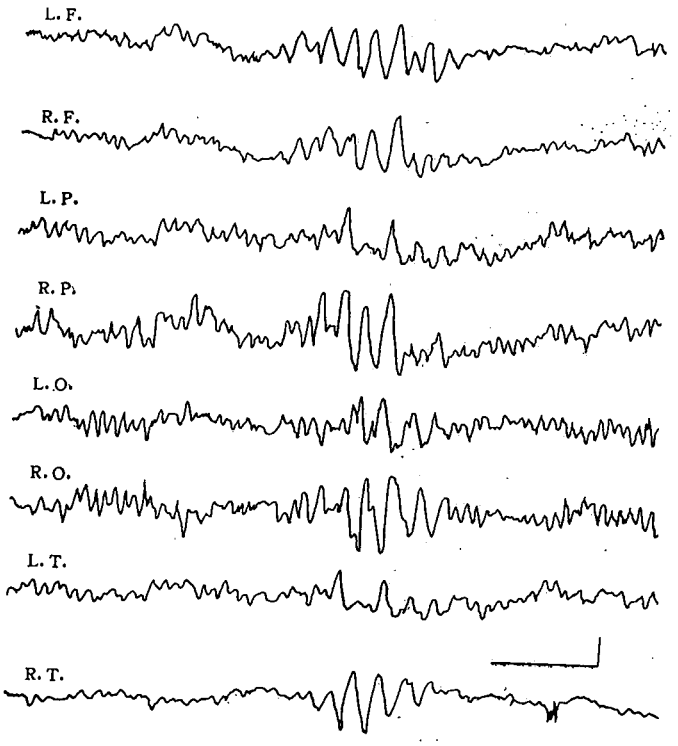

Fig. 1(4)(b) Same subject as in Fig. 1(4)(a). Build-up in the end of hyperventilation for 3 min.

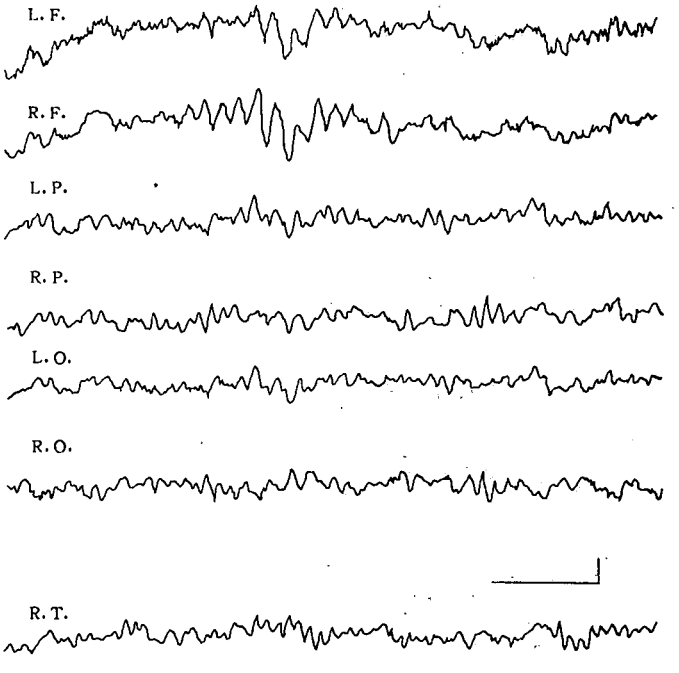

Fig. 1(5) Normal boy, age 8 years-awake. Fast waves (30 c/s) and high voltag, bursts of θ waves in the frontal area.

Table 2: EEGs of control children: (17 of 8 years, 25 of 9 years and 13 of 10 years of age)

	EEG finding	Age constituent 8 yr.	9 yr.	10 yr.	No.	Hyperventilation S	M	θ	B	Flicker stimulation S	M	θ	B
Normal	Normal	12	18	12	42	5	1	1	2	2	5	3	1
	Intermediate fast waves (15–20 c/s) in the frontal.			1	1							1	
	Asymmetry (20–30%) in the amplitude of occipital basic waves.		2		2								
	Sporadic high voltage slow waves in the occipital with amplitude asymmetry.		1		1								
	Fast waves (30 c/s) with bursts of θ in the frontal.	2			2	1							
	Total				48 (87%)	Positive rate: 10/48 (21%)				Positive rate: 12/48 (25%)			
Abnormal	Moderately slow waves (6 c/s basic wave of 9 years of age)		1		1								
	Slightly abnormal fast waves (F–1)		1		1								
	Diffuse paroxysmal slow waves	3	2		5	1	1		2				1
	Total				7 (13%)	Positive rate: 4/7				Positive rate: 1/7			
	Total of normal and abnormal EEGs.	17	25	13	55	7	2	1	4	2	5	4	2
						Total pos. rate: 14/55 (25%)				Total pos. rate: 13/55 (24%)			

Of 55 control children, 7 showed EEG abnormalities, 6 included in these with normal EEG, had background activities suggestive mentally retarded type. Effects of hyperventilation and flicker stimulation included small-(S), medium-(M), big(B)-build-ups and bursts of θ waves (θ). Same abbreviations were used in Table 3A and 3B.

in the frontal area. Its rate in total control was 25 % (Table 2, Fig. 1(4)b).

EEG abnormality activated by photic stimulation was about 25 % including from slight to big activations, photoconvulsive and bursts of θ waves in the frontal area which appeared only after stimulation (Fig. 1(3)b, Table 2).

(b) Special-class children's EEGs in the A primary school (Table 3A)

Four out of fourteen at A showed abnormal EEGs: (1) generalized very slow waves (No. 1. Table 3A) (2) diffuse paroxysmal slow waves (No. 2, Table 3A) (3) hypsarhythmia (No. 3, Table 3A) (4) bursts of high voltage θ waves in the frontal and parietal areas (No. 4, Table 3A) (Fig. 2(1)~(4)). Five showed EEGs of mentally retarded type as follows; (1) bursts of θ (5, 6 c/s) waves in the frontal and parietal areas (No. 5, 6, Table 3A) (2) mixture of slow (5, 6 c/s) and low voltage fast (25–30 c/s) waves (No. 7, 8, Table 3A, Fig. 2(8)) (3) anterior dominancy with low voltage fast waves (25–30 c/s) (No. 9, Table 3A). (4) diffuse α waves i.e. occipital dominant very regular high voltage α_s wave (No. 10, Table 3A, Fig. 2(10)). These were already assumed as EEG characteristics of mentally

Table 3A: EEG findings of special-class children in the A primary school accompanied with etiologies, symptoms, effects of hyperventilation (H. V.) or flicker stimulation (Fl.) and hereditary relation (Her.).

	No.	Male or Female	Age y.:m.	I.Q.	EEG Finding	Etiology	Her.	Symptom, Problem	H. V.	Fl.
Abnormal	1	M	12 : 4	61	Generalized very slow waves, 3, 4, 5 c/s.	Cerebral palsy		Diabetes insipidus, severe shortsightedness	+(M)	+(B)
	2	F	9 : 5	65	Diffuse paroxysmal slow waves	Scarlet fever	+			+(B)
	3	M	11 : 8	71	Hypsarhythmia	Difficult birth*, Bruise		Infantile speech, violent		+(M)
	4	M	11 : 6	69	Bursts of high voltage θ waves (7 c/s) in the F & P. §	Cerebral palsy		Nocturnal enuresis Pelvis dislocated		+(B)
Mentally retarded type	5	M	8 : 10	84	Bursts of θ waves (5, 6 c/s) in the F& P.			Noninterference	+(M)	+(M)
	6	M	10 : 3	72	〃	Difficult birth, Encephalitis		Thievish	+(S)	+(S)
	7	M	11 : 5	71	Slow (5−7 c/s) and low voltage fast (20−30 c/s) waves	Premature (7 m.) Febrile unconsciousness			+(M)	
	8	M	11 : 0	71	Burst of θ (5 c/s) waves in the F and P with low voltage fast waves		+	Nocturnal enuresis	+(S)	+(S)
	9	F	8 : 4	86	Anterior dominancy with low voltage fast (25−30 c/s) waves			Noninterference Thievish		
	10	M	11 : 6	66	Diffuse α_s waves			Quiet		
	11	M	7 : 4	76	Adult form (Abnormal for age)			Domestic trouble Vagrant habit Violent, Infantile speech		
Normal	12	M	7 : 4	88	Normal EEG	Acute pneumonia		Slight rigidity of hands & feet, Strabismus, Infantile speech	+(B)	
	13	M	11 : 1	80	Normal EEG	Bruise		Noninterference	+(M)	
	14	M	8 : 2	88	Normal EEG		+			

* Nephritis in the period of pregnancy and syncopic birth.
§ F, P or O: Abbreviations of the frontal, parietal and occipital areas in Table 3A and 3B.

retarded.[4,7] Moreover, No. 11, Table 3A was a problem boy of seven years old and was included in the abnormal because of its adult form (Fig. 2(11)).

The rate of abnormal and mentally retarded type EEGs to the whole EEGs in A was 78 % (11 out of 14), about 6 times that of control. Of those with abnormal and mentally retarded type EEGs 6 had etiologies of some kinds of brain deficiencies, 2 had positive

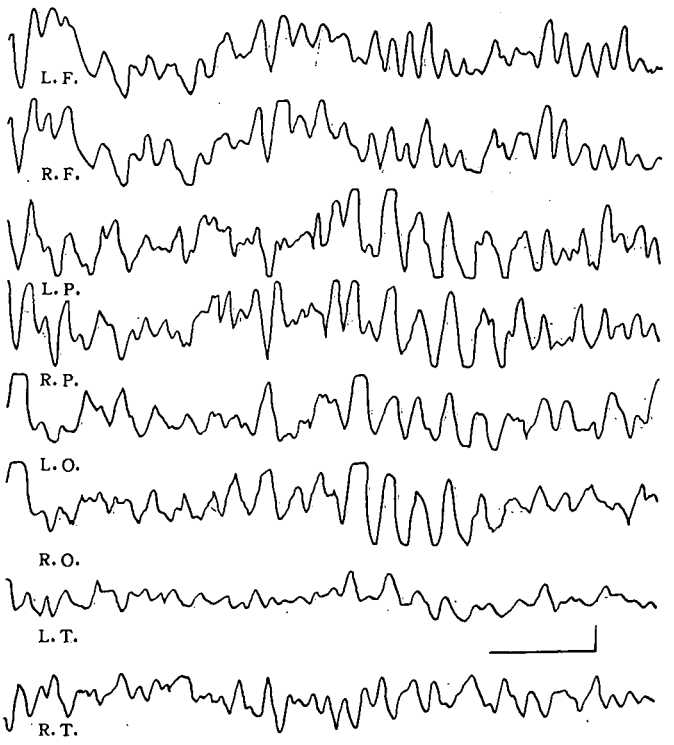

Fig. 2(1) Special-class boy, age 12 years-awake. Generalized very slow waves. Numbers in parentheses attached to Fig. 2 and 3 indicate individuals of those in Table 3A and 3B respectively.

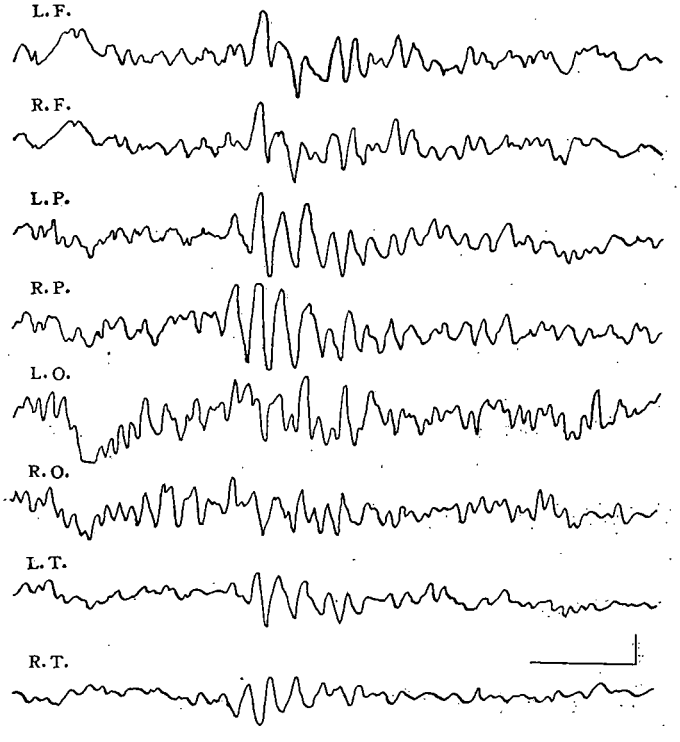

Fig. 2(2) Special-class girl, age 9 years-awake. Diffuse paroxysmal slow waves.

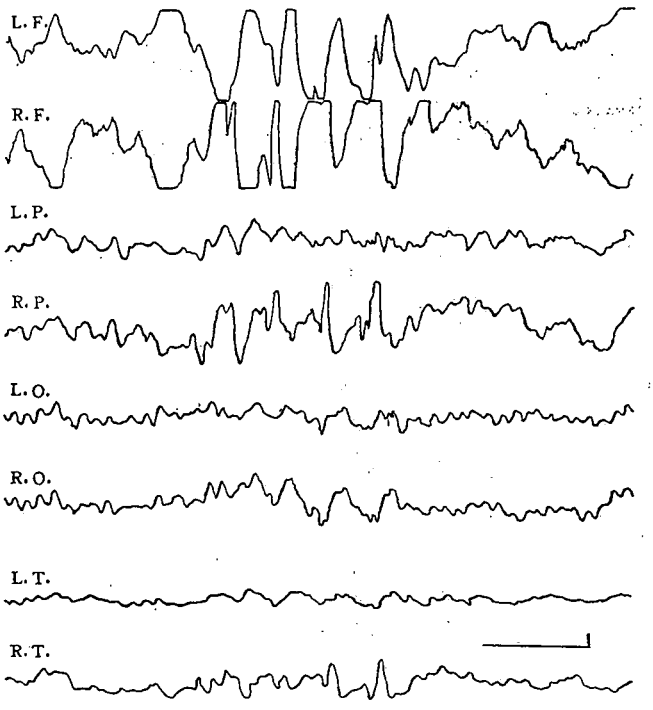

Fig. 2(3) Special-class boy, age 11 years. Hpsarhythmia.

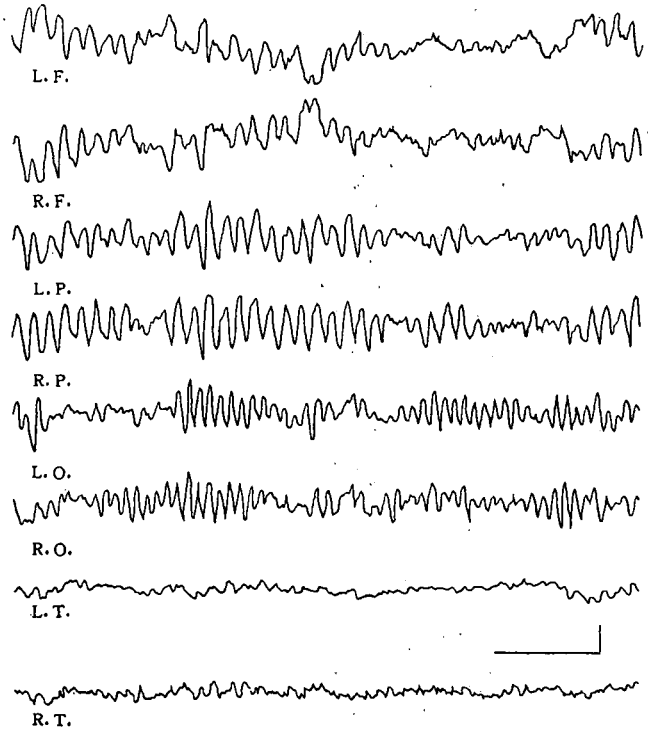

Fig. 2(4) Special-class boy, age 11 years. High voltage *θ* waves (7 c/s) in the frontal and parietal areas.

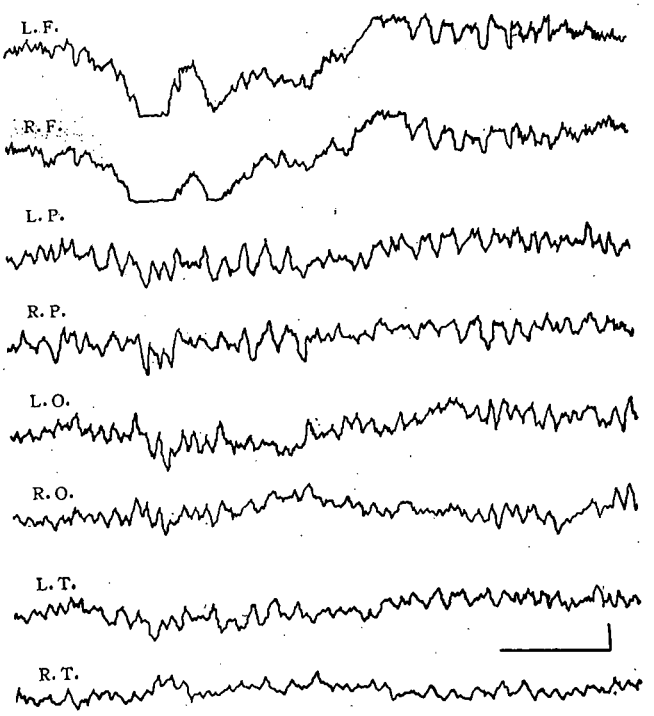

Fig. 2(8) Special-class boy, age 11 years-awake. Burst of θ waves (5 c/s) in the frontal and parietal areas with low voltage fast waves (25 c/s) in the frontal area.

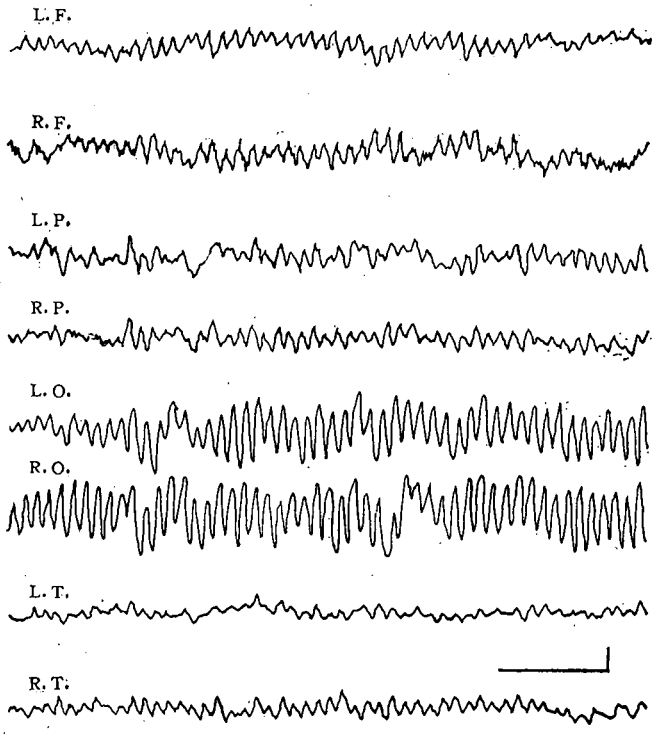

Fig. 2(10) Special-class boy, age 11 years-awake. Diffuse-α waves.

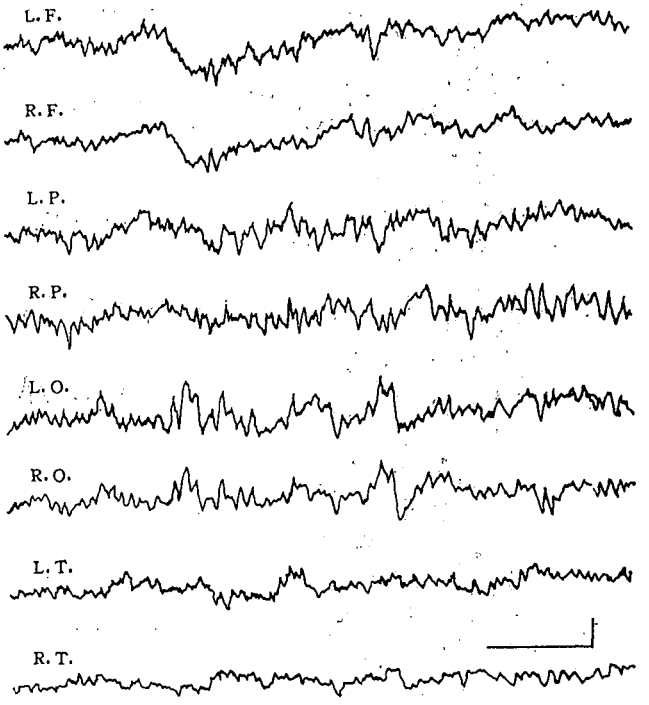

Fig. 2(11) Special-class boy, age 7 years-awake. Adult form EEG, unusual for his age.

hereditary factor and 4 had no etiology of brain damage with negative hereditary factor.

Positive activation effects were induced in 50 % by hyperventilation and in 50 % by photic stimulation which showed an easiness of its activation rather than the control.

(c) Special-class children's EEGs in the B primary school (Table 3B)

Abnormal EEGs were showed in 3 out of 19 children at B: (1) petit mal (spike and wave) type (No. 1, Table 3B) (2) asymmetry (No. 2, Table 3B) (3) diffuse paroxysmal slow waves (No. 3, Table 3B) (Fig. 3(1)~(3)).

Furthermore, 9 expressed mentally retarded type or border line EEGs. Their characteristics were as follows: (1) anterior dominancy with bursts of θ waves (6, 7 c/s) (No. 4, 5, 6, Table 3B Fig. 3(4)) (2) bursts of θ waves in all areas (No. 7, Table 3B) (3) mixture of slow (6 c/s) and intermediate fast (18−22 c/s) waves (No. 8, Table 3B, Fig. 3(8)) (4) bursts of intermediate fast waves (18−20 c/s) in the frontal area (No. 9, Table 3B, Fig. 3(9)) (5) slightly abnormal fast (F−1) (No. 10, Table 3B, Fig. 3(10)) (6) diffuse α waves (No. 11, 12, Table 3B).

The seven remainders were normal EEGs.

The rate of abnormal, mentally retarded or border line type EEGs to the whole EEGs was 63 % (12 to 19), about 5 times that of control. In those with abnormal, mentally retarded or border line type EEGs, 10 of 12 had etiologies of some kinds of brain damage, 3 had positive hereditary factor. Thus there were large numbers of EEG abnormalities

Table 3B: EEG finding of special-class children in the B primary school with etiologies, symptoms, effects of hyperventilation (H. V.) or flicker stimulation (Fl.) and hereditary relation (Her.).

	No.	Male or Female	Age y. :m.	I. Q.	EEG Finding	Etiology	Her.	Symptom, Problem	H. V.	Fl.
Abnormal	1	F	9 : 11	85	Petit mal (spike and wave) type	Encephalitis		Nocturnal enuresis, Speech disorder		
	2	F	10 : 2	50	Asymmetry (isoelectric in the left hemisphere)	autointoxi-cation		Hemiplegia (right half)	?	?
	3	M	12 : 6	89	Diffuse paroxysmal slow waves	Bruise	+	Itch	+(M)	+(B)
Mentally retarded type	4	M	10 : 2	65	Frontal dominancy, Burst of θ waves (6, 7 c/s) in the F & P	Febrile disease	+	Seizure, Speeeh disorder	+(M)	+(M)
	5	M	11 : 11	70	Frontal dominancy, Bursts of θ waves (7 c/s) in the F & P	Jaundice		Head-ache, impulsive	+(M)	+(M)
	6	M	11 : 7	74	〃 〃				+(S)	+(S)
	7	M	13 : 8		Bursts of θ waves (6, 7 c/s) in all areas	autoin-toxication		Speech disorder	+(M)	
	8	F	13 : 1	50	Mixture of slow (6 c/s) and intermediate fast waves	Encephalitis			?	?
	9	F	9 : 9	78	Bursts of intermediate fast waves (18−20 c/s) in the F	pneumonia		Clumsiness, hyperactive	?	?
	10	F	12 : 5	68	Slightly abnormal fast waves (F−1)		+	Introversive		
	11	F	12 : 7	65	Diffuse α waves with bursts of θ waves (6 c/s) in the F	Difficult birth				+(M)
	12	M	11 : 2	53	Diffuse α waves	colitis			+(θ)	
Normal	13	F	10 : 7	70	Normal EEG	pneumonia		Inferior physi-cal strength, stammer		+(M)
	14	M	13 : 2	65	Normal EEG(many 18 c/s in the F & P)	Autointoxi-cation		Chronic hives, stammer		
	15	M	12 : 9	76	Normal EEG	Bruise		Head-ache, restless		
	16	F	11 : 9		〃 〃			Nocturnal enure-sis wear glasses after Neuritis optics		+(M)
	17	F	12 : 5		〃 〃			Active, noninter-ference		
	18	F	10 : 10		〃 〃			Tonsilar hyper-trophy	+(B)	
	19	F	12 : 6		〃 〃		+			

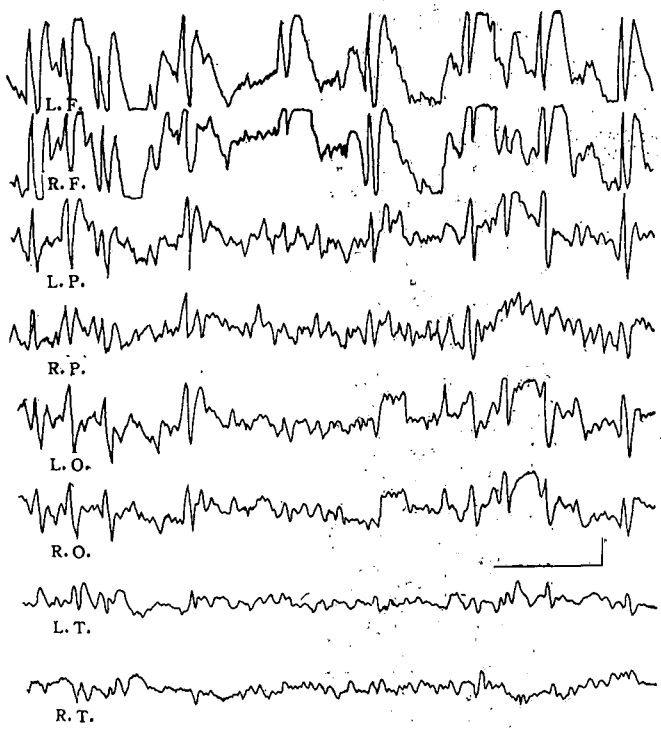

Fig. 3(1) Special-class girl, age 9 years-awake. Petit mal (spike-wave) type.

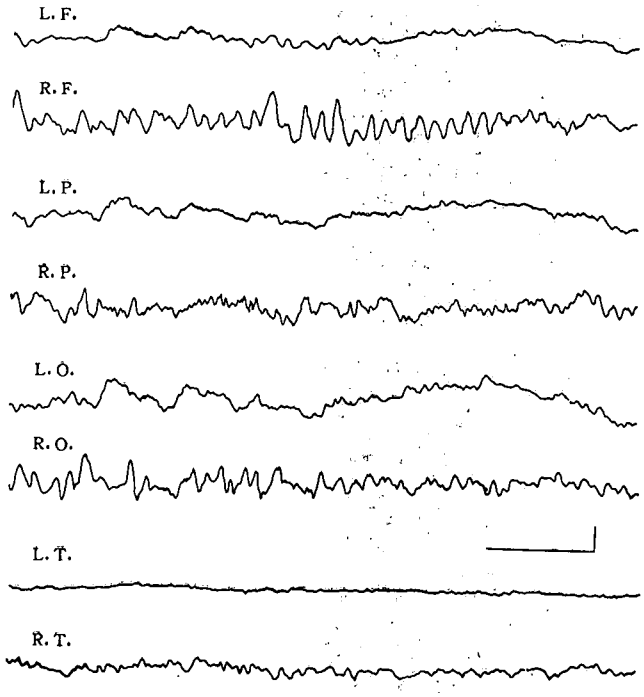

Fig. 3(2) Special-class girl, age 10 years-awake. Asymmetry (iso-electric in the left cerebral hemisphere).

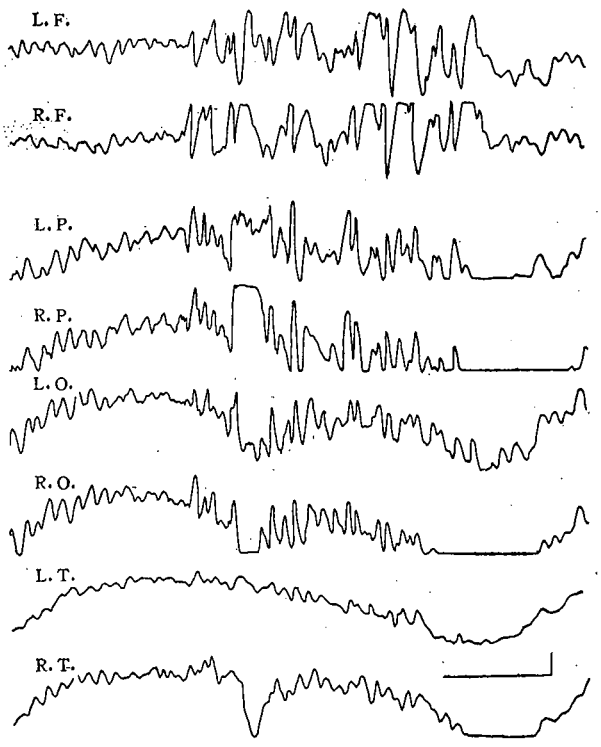

Fig. 3(3) Special-class boy, age 12 years-awake. Diffuse paroxysmal slow waves in a quiet condition.

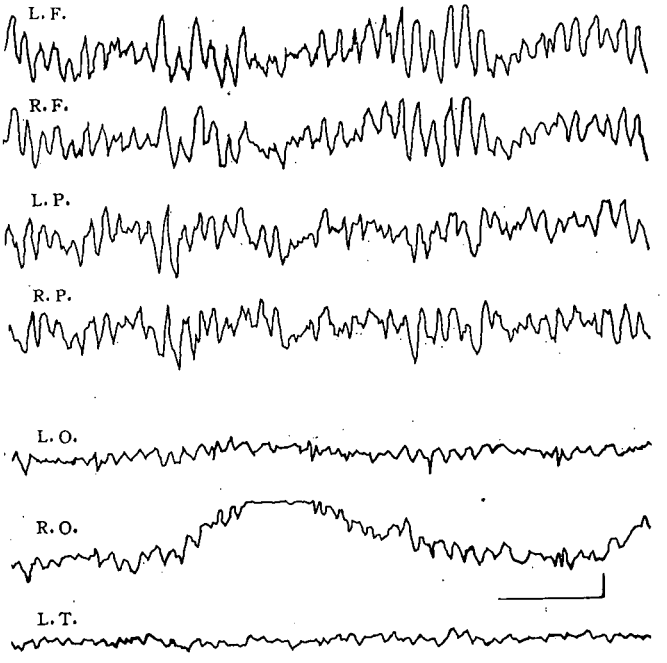

Fig. 3(4) Special-class boy, age 10 years-awake. Anterior dominancy with bursts of high voltage θ waves in the frontal and parietal areas.

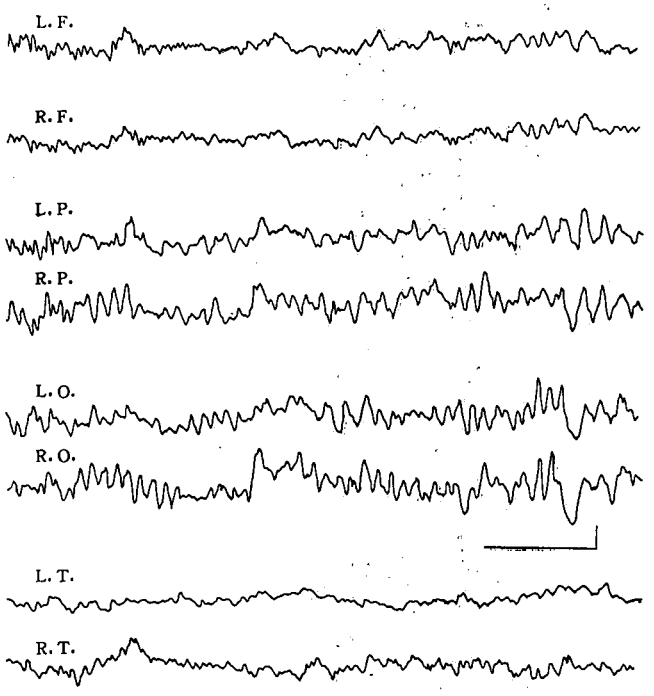

Fig. 3(8) Special-class girl, age 13 years-awake. Mixture of slow and intermediate fast waves.

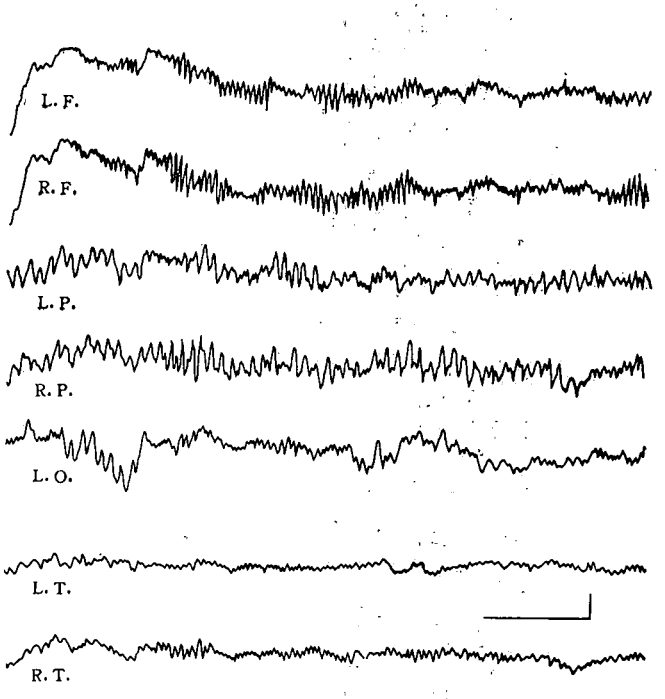

Fig. 3(9) Special-class girl, age 9 years-awake. Intermediate fast waves (18−22 c/s) remarkable in the frontal area.

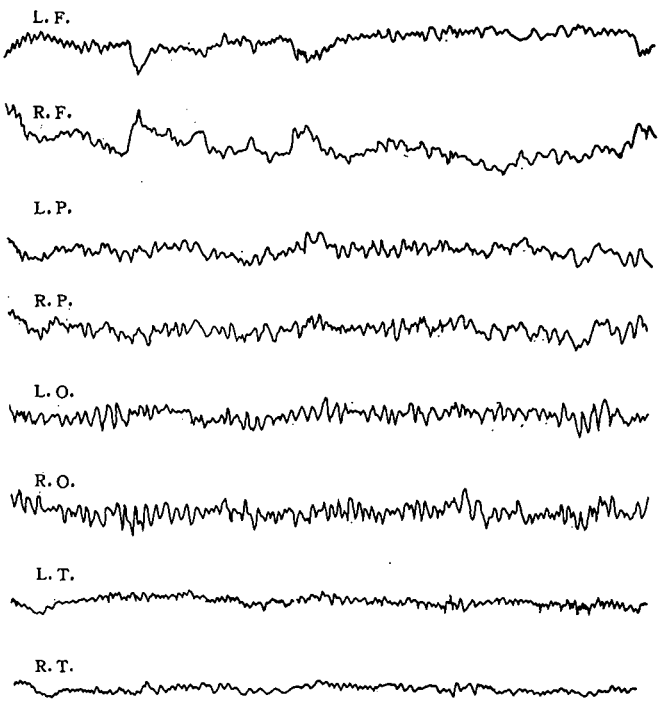

Fig. 3(10) Special-class girl, age 12 years-awake. Slightly abnormal fast waves (F−1).

caused by secondary brain obstacles in their infancies. There were 7 positive activation effects by hyperventilation or photic stimulation. They corresponded to 44 % exclusive of undecided ones interfered by EMG.

(d) Brain activities of special-class children — information obtained by key reaction time.

There was almost parallel relationship between EEG abnormality and I. Q. inferiority in special-class children. Then whether there is any difference in the activity of neuronal tract inducing motor reaction between the special-class and the common primary school children was studied quantitatively by measuring key reaction time. By applying a method described in previous report, key reaction times induced by single flicker or head light were obtained with eyes closed or open (Table 4 (a), 4 (b), 5).

As controls, 38 of 8, 9 and 10 year old children were used. As indicated in Table 4 (a), 4 (b) there was a difference of speed, to some extent, according to their ages when a 5 % level of significance was used. With their eyes open children of 10 years old could respond faster to the head light than those of 9 years old. Also children of 10 years old were quicker to respond to single flicker than those of 8 years old with their eyes closed. In the key reaction time there was a tendency of the older child to respond faster than the younger child.

However, in the present experiment, one group of 8, 9 and 10 year old children was used to compare with the special-class children. Even though the latter was composed of

older children than the control, as shown in Table 5, they were slower to respond to the head light than the control. They could respond to the supramaximal stimulation of flicker as quickly as the control, but were slow, 1.3−1.5 times the control, to respond to mild stimulation of head light.

Table 4(a): Mean values of key reaction time (msec.) in controls, 8, 9 and 10 years of age.

Age Y.	n.	Eyes Open		Eyes Closed	
		Flicker	Head Light	Flicker	Head Light
10	8	298 ± 163	476 ± 93	229 ± 71	579 ± 156
9	19	279 ± 69	603 ± 126	302 ± 127	646 ± 180
8	11	323 ± 64	625 ± 205	354 ± 99	719 ± 307
8−10	38	296 ± 94	582 ± 155	301 ± 116	653 ± 223

Table 4(b): t for testing difference of the mean value of key reaction time with ages in the control.

Comparison	Level of significance 5 %	Eyes Open		Eyes Closed	
		Flicker	Hed Light	Flicker	Head Light
8y. : 9y.	± 2.048	1.328	0.364	1.160	0.855
8 : 10	± 2.110	0.463	1.886	3.005	1.167
9 : 10	± 2.060	− 0.434	2.562	1.524	0.918

With a level of significance, 5 % there was a significant difference in those of 9 and 10 years of age in which the key reaction was induced by head light with their eyes open. Also, a significant difference in 8 and 10 years of age was observed when the key reaction was induced by a flicker with their eyes closed.

Table 5: Key reaction time (msec.) of special-class children in A and B primary school with those of controls. A, B and control were composed of number of children, 8−13 years of age indicated. When experimental values were compared with controls using level of significance, 5% (± 1.95996), those induced by head light with both eyes open or closed had significant differences judging from their t values.

	Eyes Closed				Eyes Open			
	Flicker		Head Light		Flicker		Head Light	
Control	301±116	t	653±223	t	296± 94	t	582 ± 155	t
A	324±130	− 0.611	850±165	− 3.002	387±363	− 1.435	789 ± 191	− 3.996
B	371±252	− 1.483	963±241[1)]	− 4.66	317±149	− 0.666	737 ± 453	− 1.972

1) Total number was 17 without one, 10 year old.

	No. in each age						Total No.
	8	9	10	11	12	13	
Control	11	19	8				38
A	3	1	3	6	1		14
B		3	4	4	5	2	18

IV. Discussion

1) On the normal and abnormal EEGs

It is a question how many abnormal EEGs would be found in the control primary school children as compared with those in special-class children. In the present study, 7 of 55 controls had the following kinds of EEG abnormalities : (1) moderately slow (2) slightly abnormal fast (F-1) (3) diffuse paroxysmal slow. Moderately slow activity is a minimal form of EEG abnormality, usually reversible and creating no presumption of structural damage. Also slightly abnormal fast waves do not correlate highly with any symptomatology. Number of persons with moderately slow or slightly abnormal fast waves was both 1 in 55 control subjects (1.8 %), which was a low proportion. Moreover, diffuse paroxysmal slow activity is rarely an exclusive abnormality in childhood. It is not highly epileptic unless it is associated with epileptic or epileptiform symptoms. The rate of appearance in controls was 9.1 %, which was rather high contrasted with about 4.2 % for ages up to 9 years old[2]. Long-term follow-up studies accompanied serial EEGs of children, who showed diffuse paroxysmal slow activity, may be able to predict a seizure and to avoid a production of symptoms provided that suitable medical, nutritional or environmental precautions were taken.

In controls, the remaining 48 showed normal EEGs in which 6 had background activities suggestive of mentally retarded type. On the other hand, 10 out of 33 special-class children had normal EEGs (Table 6). Generally, normal EEGs are a presumptive but not positive evidence of normality. In undifferentiated mental retardation, Gibbs found normal EEGs about 50 % for 5−9 year old children and 39 % for 10−19 year old children. In the present study, the number of normal EEGs in undifferentiated type of special-class children was 5 out of 12 (42 %) (Table 6).

Anyhow, normal EEG would not guarantee normal brain function, for EEG does not indicate all types of brain function nor does it view all parts of the brain. However, it suggests that there is no acute or progressive damage in the accessible cortex, no highly active epileptic process and no disturbance of those types of neuronal metabolism that are concerned in electrogenesis.

2) On the hyperventilation and photic stimulation

A big build-up activated by hyperventilation is not definitely abnormal at any age. In general, the younger the child, the greater will be the build-up. Thus, big build-up was observed in 50 % of 6−10 year old normal controls and in 87 % of epileptics of the same ages[1]. Below 10 years of age, therefore, it is normal. It is not very significant by itself and is classified as slightly abnormal in adults. It may reinforce a judgment of abnormality which is based on other features of EEG. In the present study, 25 % of control showed a sign of build-up as compared with 43−44 % of those in the children of special-class. The build-up is considered to be a result of failure in homeostatic mechanism, which ordinarily abjusts the arterial carbon dioxide tension in the brain. Present results suggest that the special-class children's competence of the homeostatic mechanisms was feeble and sensitive to acapnia in the cerebral neurons.

The effect of photic stimulation, photoactivation, in which increases in voltage of normal

activity or slow waves occur, has not been clearly established in its diagnostic value. The photoconvulsive, in which seizure discharges appear even after discontinuation of photic stimulation, commonly associates with clinical manifestations of an epileptic character. It is also able, however, to induce in a small percentage of normal persons. They, therefore, were concerned as a reference data attached to children's EEG. In the present result, the children in the special-class showed 1.7 times positive effects of control with photic stimulation.

3) EEG abnormality in the special-class children

EEG abnormality observed in the special-class children was classified into two groups: (a) EEGs accompanied by epileptic or other disease and (b) mentally retarded type or border line EEGs. They included EEGs with the following characteristics:

(a) 1. Petit mal type 2. Asymmetry 3. Diffuse paroxysmal slow wave 4. Generalized slow wave* 5. Hypsarhythmia.

(b) 1. Bursts of θ waves (in all areas, in the frontal and parietal areas with or without frontal dominancy).
2. Mixture of slow and low voltage fast (25−30 c/s) waves.
3. Mixture of slow and intermediate fast (18−22 c/s) waves.
4. Intermediate fast (18−20 c/s) waves in the frontal area.
5. Frontal dominancy with low voltage fast (25−30 c/s) waves in all areas.
6. Slightly abnormal fast (F-1) wave.
7. Diffuse α waves.

It is already well known that, hypsarhythmia, epileptic EEG, especially appears often in the children with mental retardation and others in (a) also tend to accompany mental retardation. Medical treatment may be given to the children with epileptic EEGs, petit mal, diffuse paroxysmal slow activity and hypsarhythmia. A child with EEG asymmetry caused after cerebral palsy should be rehabilitated to prevent further invasion of paralysis.

Kasamatsu[6] and Izawa et al[3] have reported characteristics of EEG in the mentally retarded as follows: (1) asymmetry (2) intermediate fast waves (18−22 c/s) (3) anterior dominancy (4) bursts of high voltage θ waves (5) mixture of fast and irregular slow waves. After analytical study Katada also reported that dominant components of EEG in the mentally retarded exist in the lower frequency range than that in the normal. "The lower range component becomes more dominant on from the occipital to the frontal region"[8]. EEG characteristics written in (b) roughly coincided with these observations. On the other hand, diffuse α wave has been observed often in the mentally retarded. In the present study this pattern was found in the children with I. Q. inferiorities associated with quiet temper in 11−12 years of age, i.e. in rather grown-up stage electroencepharographically.

After all, children with EEGs of mentally retarded type may be directed to develop their own potentialities in order to join in social life.

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* A child (No. 1, Table 3A) suffered from Diabetes insipidus, which suggests a EEG disorder caused by a infectious disease of hypothalamus.

4) On the brain activity of special-clss children

Parallel with EEG recording, key reaction time induced by photic stimulation was measured as a study of their physiological activity. This is a time from a moment of illumination untill a response of pushing a key made after a sensation of light is received by impulses passing through the occipital visual cortex. This is a simple reaction, rather close to a reflex phenomenon, which connects two conditions "feel the light" then "push the key", transmitted through brain activity. There were no differences observed in the key reaction times induced by supramaximal stimulation between experimental subjects and controls. This means both have equal activities to respond reflexively. Delays in key reaction for a weak photic stimulation observed in the experimental subjects suggests a delay in central nervous activity which would affect a skill of physical exercise.

5) On the etiology of EEG abnormality

In the medical histories of special-class children, there were several etiologies which might cause cerebral obstacles or EEG abnormalities: difficult birth, prematurity, nephritis during the period of pregnancy, cerebral palsy, encephalitis, febrile disease (scarlet fever, pneumonia) etc. in A, and difficult birth, encephalitis, fever caused by autointoxication, pneumonia, jaundice, colitis, bruise etc. in B. These facts suggest that the disturbances around birth, cerebral disease and infant's disease with fever tend to accompany mental retardation secondarily.

Table 6: EEGs and their medical histories.

Groups with pos. med. history

EEG Finding \ Etiology	Perinatal disturbances	Brain[1] disease	Febrile[2] disease	Jaundice	Bruise	Febrile disease or Bruise + Pos. Her.[3]	Total
Abnormal	1	3	1			2	7
Mentally retarded or border line type	2	2	2	1		2	9
Normal			3		2		5
Total	3	3	6	1	2	4	21

Groups with undifferentiated med. history

EEG Finding \ History	Pos. Her.[3]	Neg. Med. history, Noninterference	Total
Abnormal			
Mentally retarded or border line type	2	5	7
Normal	2	3	5
Total	4	8	12

The rate of abnormal, mentally retarded or borderline EEGs to total EEGs,

23/33 : 70 %

1) Brain disease : Encephalitis and cerebral palsy.
2) Febrile disease: Pneumonia, autointoxication, scarlet fever, colitis etc.
3) Pos. Her. : Positive hereditary factor.

Table 6 indicates a summary of EEGs and their etiologies. Experimental subjects with positive medical history were 21 of 33 in total (64 %). The remainders were composed of those with negative medical history of cerebral obstacles, unfavorable environment and positive hereditary factor. Those with negative medical history accompanied with EEGs of mentally retarded or border line type had commonly poor, noninterference homes. This might mean normal cerebral development occurs in those with certain nutrition and careful environment. Those with positive hereditary factor had also poor, noninterference homes in which effects of hereditary and environmental factor could not be discriminated with each other. In conclusion, most of EEG abnormalities observed in the special-class children occurred secondarily from their medical history during the embryo or the infant. However, lack of suitable precautions, educational or developmental, might be another important factor of EEG abnormality.

V. Summary

Thirty-three EEGs of special-class children (7 to 13 years of age) at A and B primary schools in Kochi city were compared with 55 of those of common primary school children (8 to 10 years of age). The following facts were observed:

1. Abnormal, mentally retarded or border line EEGs in the special-class children occupied 70 % of all, 5−6 times that of control. Percentage of experimental subjects with positive medical history of cerebral obstacles within them corresponded to 70 %. The rest was composed of those with positive hereditary factor, unfavorable environment or negative medical history, in which a lack of educational or developmental precautions was a common factor, and which suggested its effect upon the cerebral development.

2. Their main etiologies were encephalitis, cerebral palsy, febrile disease, obstacles around birth and bruise.

3. EEG abnormalities included petit mal type, asymmetry, generalized slow waves, diffuse paroxysmal slow waves and hypsarhythmia. EEGs of mentally retarded or border line type were observed as follows:

(1) Bursts of θ waves (in all areas, in the frontal and parietal areas with or without anterior dominancy).

(2) Mixture of slow and low voltage fast waves.

(3) Mixture of slow and intermediate fast waves.

(4) Intermediate fast waves in the frontal area.

(5) Mixture or low voltage fast waves in the all areas with anterior dominancy.

(6) Slightly abnormal fast wave (F-1).

(7) Diffuse α waves.

Children would be taught in consideration of their EEG findings: especially children with subclinical seizure discharges would need to take care of their symptoms.

4. Hyperventilation and photic stimulation produced more build-up, photoactivation and photoconvulsive in the experimental subjects than controls.

5. Key reaction times induced by photic stimulation were measured as an index of brain activity. Compared with the control, there were no differences in the key reaction

times induced almost reflexively by supramaximal flicker stimulation. However, there were generally delays in these induced by weak photic stimulation which might suggest delays in the central nervous activity.

Acknowledgements

The auther wishes to express her sincerest appreciation to American Association of University Women for a study grant in 1963 -64, which made this work possible. The kind cooperation of teachers, parents and children in the attached primary school, Kochi University or the Showa primary school are heartily appreciated. My grateful thanks are also due to Dr. Mikinao Mimoto for his generous guidance; to Dr. Takaharu Yamada for his efficient advice; to Mrs. Yoshi Ujihara for her help especially in providing information about special-class children; to Mrs. Michie Abo, Mrs. Yasuko Moriguchi, Miss Noriko Matsui and Miss Sumiko Ichikawa for their assistance in recording EEG.

References:

1. Gibbs, F. A. and Gibbs, E. L. : Atlas of electroencephalography, Addison Wesley Press, Inc. I (1958).
2. Gibbs, F. A. and Gibbs, E. L. : ibid. III (1964).
3. Izawa, S., Hirai, T., Kurisu, E., Mabuchi, M. & Iwata, T.: EEG and mental development -1. EEGs of mental defectives-Proc. VIIth Ann. Meet. Jap. EEG Soc. 8, (1958).
4. Izawa, S., Hirai, T., Kamide, H., Mabuchi, M. & Kurisu, E. : EEG and mental development (3) Proc. IXth Ann. Meet. Jap. EEG Soc. 49, (1960).
5. Kasamatsu, A., Hirai, T., Izawa, S. & Mabuchi, M. : On the hypersynchronous θ rhythm in normal infancy. Proc. Xth Ann. Meet. Jap. EEG Soc. 9, (1961).
6. 笠松 章・伊沢秀而：知能及び精神薄弱と脳波. 精神医学最近の進歩 第II集 (1960).
7. 梶谷 喬・大田原俊輔・志茂 実・向井幸生・高畠美人：問題行動児の脳波学的研究. 小児の精神と神経. 第4巻 第3号 (1964).
8. Katada, A. : Analytical studies on the EEG of mentally retarded children by the multi-channel magnetic recording. 東京教育大学部紀要 11 (1965).
9. Oikawa, I. : A physiological study of brain activity. —An investigation concerned with the latency of α—block and key reaction time. (Proceedings) Bull. Fac. Education, Kochi Univ. No. 18, (1966).
10. 大田原俊輔・梶谷 喬・三崎 功：小児てんかんにおける知能障害. 小児科診療, 第25巻 第7号 (1962).
11. 下田又季雄：広義異常児の医学的研究 —脳波の立場から— "異常児" 診断と治療社 (1958).
12. 高畠美人：脳性小児麻痺の脳波に関する研究. 臨床脳波 第7巻, 第2号 (1965).