SEVERAL NOTES ON PETIT MAL ABSENCE

—— A CONTRIBUTION FROM CLINICO-EEG STANDPOINT OF VIEW ——

By

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Introduction

Since the eighteenth century, the term "petit mal" has been used to distinguish it from grand mal or convulsive type seizure in the clinical behaviour—in the intensity and duration of attack (Lennox 1960). However, the attack regarded as petit mal in those days, appears to have included seizures that are classified as psychomotor epilepsy, minor motor seizure and autonomic epilepsy in these days, until Gibbs, Davis and Lennox (1935) reported that a group among the so-called petit mal shows special electroencephalograph pattern, and Gibbs, Gibbs and Lennox (1943) suggested that the term "petit mal" should be used in restricted meaning. Nowadays, generally, the definition of petit mal absence is admitted as follows: Petit mal absence is the spell of brief disturbance of consciousness, either with or without minor motor symptoms and autonomic symptoms, accompanying 3 c/s. spike and wave complex in the EEG during the spell.

Since 1935, a lot of work on petit mal has been reported and the works of Gibbs and Gibbs (1952), of Penfield and Jasper (1954) and of Lennox (1960) impress us as if they exhausted the information as to petit mal. Of course, however, the pathophysiology of petit mal has still a lot of problem, and many works as to petit mal have been reported. Anyway, clinical and electroencephalographical natunre of petit mal appears attractive as if it is a code book to resolve problems of pathogenesis of epilepsy and of pathophysiology of consciousness.

The author has observed petit mal patients, since 4 years and found several interesting cases. In present work, the author mentions common characteristics of clinical symptoms and EEG's of petit mal absence, and discusses pathophysiological nautre of it.

Subjects

Among 1000 epiletics who have EEG examinations at our department (Hirosaki Univ. Hospital) between July 1956 and December 1961, diagnoses of 24 patients were established as petit mal absence by observing clinically attacks with which paroxysmal bursts of rhythmic 3 c/s. spike-and-wave complex were shown in EEG simultaneously. In most of them, EEG's were recorded and their clinical attacks were observed by the author himself. Sixteen cases had serial tracings taken over a period of years. Additional clinical information was acquired by observing document films of attacks and by reviewing clinical records.

Results

1) Sex and Age : Among the total subjects, sixteen were female and the number was just twice as many as of males. Most of the past reports have shown an incidence of petit mal absence is more on female (Allen et al 1949, Gibbs and Gibbs 1952, Paal 1957, O'Brien et al 1959, Lennox 1960). The age of onset of petit mal absence ranged from 4 years to 14 years. The average is around 8 years 3 months. (Male: 9 yeas 7 months, female: 7 years 6 months.) In these subjects,

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Table 1 Clinical picture

Case No.	Name	Age	Sex	Onset yrs.	Seizure Type (the others)	Most Effective Drug	Clinical Picture	
							Eyes	Behavior
		y m		y m				
1	T. F.	12 : 3	♂	11 : 5	Grand mal#	Tridione	Staring	Immobility
2	M. T.	7 : 11	♀	5 : 6	—	Tridione**	roll upwards	Automatism
3	T. K.	9 : 0	♂	8 : 8	Focal #	Tridione	Blinking roll upwards	Asymmetrical twitching
4	H. K.	10 : 2	♀	7 : 10	—	Tridione	Staring	Automatism
5	H. Y.	15 : 9	♂	14 : 7	—	Tridione*	Blinking Staring	Nodding Mastication
6	M. I.	10 : 0	♂	8 : 0?	#Focal	Tridione*	roll upwards	roll head to left
7	H. M.	13 : 5	♂	8 : 0?	Grand mal#	Tridione	Staring	Nodding
8	Y. N.	6 : 2	♀	5 : 0?	#Grand mal	Tridione**	roll rightup-wards Blinking	Automatism
9	M. I.	7 : 0	♀	6 : 5	—	Tridione** Quinazolone	roll upwards	Immobility Micturition
10	R. I.	7 : 4	♀	6 : 4	—	Tridione**	Staring	Immobility Micturition
11	A. N.	9 : 6	♀	8 : 8	Grand mal#	Tridione	Staring	Immobility
12	H. K.	4 : 9	♀	4 : 6	—	Tridione* Diamox	roll up. Blinking	Automatism
13	R. T.	8 : 6	♀	4 : 2	—	Tridione**	Staring	Immobility Micturition
14	T. T.	11 : 3	♀	10 : 3	Focal #	Tridione Diamox	Staring	Immobility
15	N. N.	6 : 9	♂	6 : 6	—	Tridione**	Staring	Immobility
16	M. Y.	16 : 9	♀	10 : 0?	—	Tridione	Staring	Automatism
17	T. S.	15 : 3	♂	8 : 0?	#Psychomotor	?	roll upwards	Immobility
18	K. N.	11 : 4	♀	11 : 0	—	Tridione	Staring	Immobility
19	H. T.	15 : 0	♀	12 : 0?	#Grand mal	?	roll to right	roll head to right
20	T. M.	12 : 8	♂	12 : 0	Grand mal	?	Staring	Immobility
21	F. O.	7 : 9	♀	7 : 7	—	Tridione	Staring	Immobility
22	K. T.	12 : 11	♀	8 : 0?	Focal #	Tridione	Staring	Immobility
23	T. N.	8 : 4	♀	5 : 0?	—	none effective	Staring	Immobility
24	M. S.	11 : 3	♀	8 : 0?	—	?	Blinking Staring	Immobility

#: Its position shows the time of onset of the other seizures. If the mark is followed by the term of seizure, the onset of petit mal followed the other seizures, vice versa.

**: Their attacks have disappeared during treatment, but whether the disappearance might be caused by medication, or not, is questionable.

*: Their attacks have been completely controlled by medication and it was proved that medication was effective.

? : Questionable or unknown.

the highest age when petit mal absence attacks were recognized not only clinically, but electroencephalographically, was 18 years 10 months (case 16).

Generally, it has been accepted that the onset of absence is most frequently in the age between 3 years and 10 (Gibbs and Gibbs 1954, Janz 1955, Chao et al 1958, Lennox 1960). Although Allen et al reported an epileptic patient whose attacks joined petit mal absence at 43 years of the age and in Gibbses' atlas an EEG of petit mal of the age, 64 years old, has been shown. Generally, only few petit mal patients are seen in the age over 20 years.

6 patients had positive history as to heredity one of them has a mother with a history of petit mal (case 2) and the other one has a brother of petit mal absence (case 13).

Table 2 Correlation between seizure type and clinical picture

Symptom \ Seizure Type	Petit mal only	+Grand mal	+Focal	+Psycho-motor	Total
Heredity	3	2	1	0	6
Mental Defeciency	3	0	1	0	4
Automatism	4	1	0	0	5
Twitching, Blinking and Nodding	3	2	1	0	6
Immobility	8	3	2	1	14
Micturition	3	0	0	0	3
Rolling of eye-ball	3	2	2	1	8
Staring	10	4	2	0	16
Focal sign in the attack*	0	2	2	0	4

* : indicates asymmetrical symptoms of clinical picture; for instances, 'roll head to side', 'roll eyes to side' or 'asymmetrical twitching'.

2) Other Seizure Types: Grand mal, focal seizure and psychomotor seizure were found in 11 cases of the total, grand mal 6, focal seizure 4 and psychomotor seizure 1. Of these 11 patients, one patient with grand mal has recurrent paroxysmal headaches independently of petit mal and grand mal. In 4 cases the initiation of petit mal absence followed the other seizures, one patient had both types of attacks at about the same time (case 20) and in 6, petit mal preceded the others.

3) Mentality: In our series, 4 patients of mental deficiency were found. While 3 of them are patients of pure petit mal, it is only one patient who has mental deficiency with another seizure (case 6). This patient who has focal seizure in addition to petit mal has a history of encephalitis in the time of infancy. Gibbs and Gibbs (1952) has showed 5% of pure petit mal was of mental dificiency. Lennox (1960) has stated, some children may have both petit mal and serious mental defects, but the latter may be due to brain injury or to congenital brain defects not necessarily related to the seizures. However, each of these pure petit mal patients with mental deficiency have no positive past history and no family history of mental deficiency.

4) Clinical Pictures in Petit Mal Attacks: The author observed them dividing into symptoms of eyes and of behaviours. In each of both, only the prominent symptoms were registered. When the position of pupils exist in the center of, or near to center of eye during attack, the patient may objectively look vacant in the eyes. The state of eyes have been called "staring". However, in some patients their eye-balls may roll upwards or to side. Of these 24 subjects, 16 showed staring, ang 6 upward rolling, on right-upwards and one to the right. The patients who have other seizures in addition to petit mal show rather more (5/11) the rolling of eyes, than the patients of pure petit absence (3/13).

Immobility, nodding of head, twitching of face muscles and blinking of eye-lids have been recognized as common motor symptoms of petit mal absence. In this series, immobility were seen on 14 patients, nodding and twitching on 3, and blinking on 6. And, as the other manifestation, 5 patients showed petit mal automatism (Penfield and Jasper) and 3 have had sometimes micturition during attacks. However, asymmetrical motor symptoms, such as asymmetrical twitching of lips (case 3), lateralized rolling of eyes and rolling of head that are observed in some patients during petit mal attacks, are peculiar in comparison with common features of the attack. These asymmetrical symptoms were seen only among the patients with other seizures, while incontinence was observed only among the patients of pure petit mal. The micturition during a petit mal is more common in girls than in boys (Lennox 1960).

5) Response upon Treatment: 20 patients of the subject had medication for 6 or more months and the author considered the periods as the time enough to evaluate the effectivity of the medication. The kinds of drugs given these 20 patients were as follows: Tridione, Diphenylhydantoin,

Mysoline, Phenobarbital, Diamox and Chloroquine derivative (Wada, 1961). Diphenyl-hydantoin and Mysoline were used only for the patients with convulsive seizures and psychomotor seizure. Tridione was the most effective to petit mal absence among these drugs, although not always sufficiently effective. There were 3 cases for whom Diamox or Chloroquine derivative were as effective as Tridione. Generally, petit mal absence was resistant to medication and it was hard to control completely. Of patients whose attacks were completely controlled after some periods of medication, in 7 cases it is apparently of question to regard the disappearance of the attacks as only effectiveness of medication, because the discontinuity of medication or the administration of placebo after the disappearance of the attacks showed no recurrence or aggravation of petit mal: the attack of these patients might have disappeared spontaneously since around the time when the medication appeared to show a definite effect. For instance, attacks of case 9 had been quite resistant to medication for 3 and onehalf years and then her attacks disappeared in spite of similar quality and quantity of medication to the previous one, showing grandual decrease of attacks for 3 months. In another 3 cases the medication was proved to be really effective to their attacks. Lennox (1945, 1947) reported, Tridione tended to increase convulsions or even precipitate these in persons not

Table 3 EEG-findings

cases No.	Maximum potential	Laterali-zation	Frequency (c/sec) beginning-end	Appearrance	Other findings
1	—	—	mult. 2.5—2.0	MM	bilateral spikes
2	frontal	—	3.5—3.0	spontan.	—
3	frontal	rt.	mult. 4.0—3.0	MM	sp-w-c in rt. temporal area
4	posterior	—	3.0—2.5	OB	—
5	frontal	lt.	3.5—3.0	OB	—
6	frontal	rt.	mult. 3.5—2.5	OB	mult. spikes, predominant in rt. hemisphere
7	central	—	4.0—2.5	OB	—
8	frontal	—	mult. 3.5—2.5	spontan.	bilateral mult. spikes.
9	frontal	—	3.0—2.0	OB	—
10	—	—	3.5—3.0	spontan.	4 to 6 c/sec high voltage slow wave burst
11	frontal	—	mult. 3.5—2.5	photic	—
12	frontal	—	3.0—2.5	spontan.	—
13	frontal	lt.	3.5—3.0	OB	sp-w-c, predominant in lt.
14	frontal	rt.	4.0—3.0	spontan.	spikes in rt. frontal area
15	—	—	3.5—3.0	OB	spikes in lt. frontal area
16	anterior	—	4.0—3.0	OB	—
17	frontal	—	4.0—3.0	MM	bilateral spikes (spikes in rt. temporal area)
18	—	—	3.5—3.0	OB	—
19	central	—	4.0—2.5	OB	—
20	centro-parietal	rt.	4.0—2.5	spontan.	bilateral spikes
21	anterior	—	3.5—3.0	OB	—
22	frontal	lt.	3.5—3.0	MM	(spikes in lt. temporal area)
23	—	—	3.0—2.0	MM	—
24	frontal	—	mult. 4.0—2.0	spontan.	spikes in frontal area

mult. : spike components of 3 c/sec. spike and wave burst tend to be multiple spikes. OB : overbre athing. MM : megimide-metrazol activation (Wada et al 1962). Spontan. means that the compex patterns occur spontaneously without any activating procedures, during recording; OB shows the patient, responding to overbreathing, 'photic' shows the patient whose attacks are produced specifically by photic stimulation and MM shows the patient whose attacks are not produced by another activation, by MM.

before subjects to the major attacks. However, the fact was not ascertained in this subject: Tridione did not show reasonable increase of convulsive attacks in any cases.

6) Electroencephalograms (s. Table 3)

The EEG's of subjects were recorded 76 times for 5 and one half years. Among them, 54 EEG's which have been recorded at the intervals of 6 months or more in each patients were selected to analyses.

a) *Rhythmic 3 c/s. spike and wave bursts in the first EEG. (Table 4)*

There are 2 patients (case 3 and 23) in the first EEG of whom the rhythmic 3 c/s. spike and wave bursts in not observed even by activating procedures. Their EEG's were analysed in the first one that manifested the complex patterns. Generally, the features of 3 c/s. bursts of the complex were constant in an EEG and common in some EEG's recorded at the short intervals, of each individual person.

Finding \ Seizure	Petit mal only	+Grand mal	+Focal	+Psycho-motor	Total
3 c/sec. sp-w-c pattern					
Maximum potential					
anterior	8	2	4	1	15
centro-parietal	0	3	0	0	3
posterior	1	0	0	0	1
lateralization	1	1	4	0	6
Multiple spikes components	1	3	2	0	6
Activation					
none	4	2	1	0	7
overbreathing	7	2	1	0	10
photic stimulation	0	1	0	0	1
MM*	1	1	2	1	5
Frequency (c/sec)					
at the beginning					
4	2	3	2	1	8
3.5	7	2	2	0	11
3	4	0	0	0	4
2.5	0	1	0	0	1
at the end					
3.5	0	0	0	0	0
3	8	0	3	1	12
2.5	2	5	1	0	8
2	3	1	0	0	4
The other findings					
Spike : bilateral	1	3	0	1	5
localized	1	0	2(1)	0(1)	3(2)
Sp-w-c : bilateral	0	0	0	0	0
localized	1	0	1	0	2

Table 4 Correlation between seizure type and EEG findings

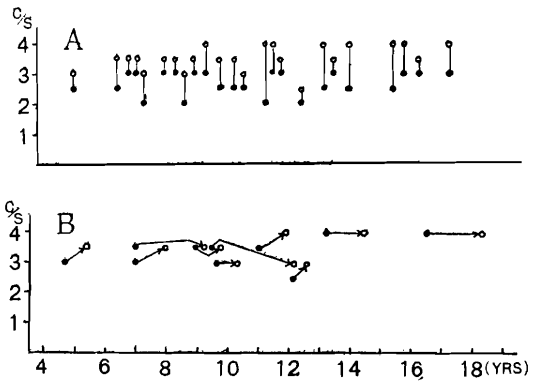
*Megimide-metrazol activation (Wada et al 1962).
( )indicates discharges provoked by action.

Maximum Potential: Regardless of lateralization of the complex burst, the area of maximum amplitude of it was analysed. Excepting 5 EEG's without clearly distinguishable emphasis, 15 EEG 's showed it in the anterior area (frontal or central area) of the head, 3 in centro-parietal area and one in the posterior area (parieto-occipital area). It is widely accepted that 3 c/s. complex burst of petit mal absence shows commonly maximum potential in anterior or frontal area of the head.

The lateralizations of the pattern were seen in 6 EEG's in the total, and it is to be stressed that all of the patients with focal seizure showed the lateralization of the pattern.

Multipe spikes component of 3 c/s. complex burst: Some patients had the rhythmic 3 c/s. multiple spikes and wave bursts, at the beginning of which usually the multiple spikes component was the most evident and the component became gradually less evident and consecutively was transformed into the usual spike and wave bursts. Of 6 cases with the pattern, 5 were the patients suffering from convulsive seizures in addition to petit mal attacks.

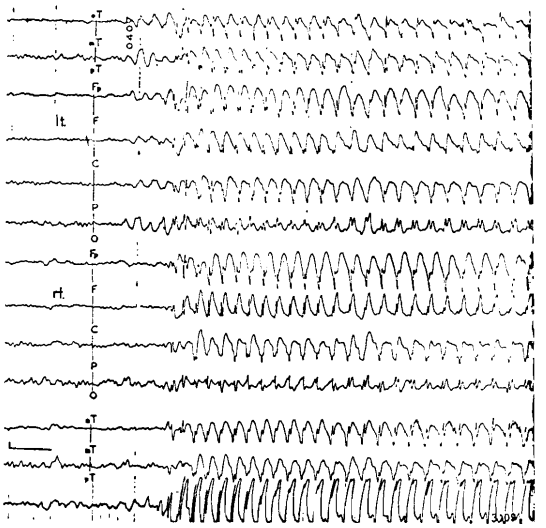
Occurance of 3 c/s. Spike and Wave Burst: While some patients showed petit mal attacks frequently during recording of the EEG of resting state, some patients scarcely do in the restricted time of recording, unless some activating procedures are given them. In this series 7 cases showed the seizure patterns spontaneously in resting EEG's but the others manifested the pattern only in the activated EEG's: 10 patients with overbreathing, 1 with photic stimulation and 5 with MM-activation (Wada et al 1962). Only one of the patients of the pure petit mal responded to neither overbreathing nor photic stimulation, but to the MM. This is the special patient who did not respond to any medication (case 23). Generally speaking, a tendency that the patients with the other seizures in addition to petit mal show hardly the patterns either spontaneously or by means of overbreathing, appears to be observed.



A) Correlation between cycles of the spike wave complex patterns and age when the patterns were recorded for the first time. ◦ show the frequency at the beginning of the pattern; • at the end. The frequencies of the beginning, generally, increase with the advance of the age.

B) As to the frequency of the beginning of the pattern, alteration of frequency with the advance of the age in individual cases (10 patients). The frequencies were compared in EEGs, recorded in the interval of 6 months or more, with each other. Four cases show the shortening of the frequency. However, one patient had slower pattern for 2 years and 6 months after the first EEG.

Fig 1 Cycle of spike-wave complex.



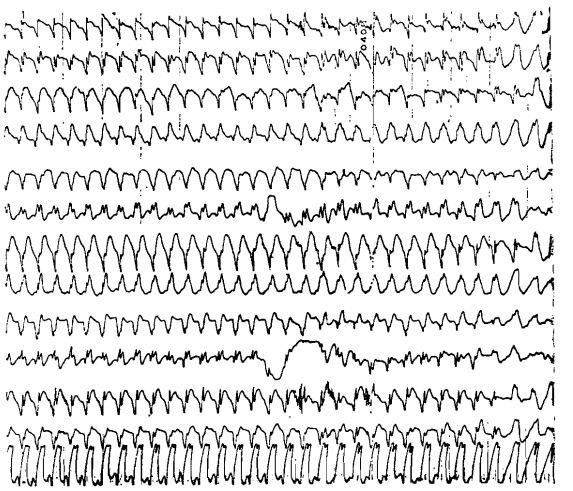


Fig. 2 (a) EEG of Case 9

Fig. 2 (b) EEG of Case 9

At the beginning of the attack, as in "a" the complex pattern shows around 3 cycle per second in frequency, but the frequency gradually decreases and finally comes to around 2 cycle per second at the end of the attack, as in "b", which continued for 22 seconds.

Frequency of Rhythmic Spike and Wave Bursts: Generally, the frequency of the 3 c/s. spike and wave in the bursts is variable with the advance of course: at the beginning of them it is faster and at the end slower.

By the way, since in an EEG the terminal frequency of the complex bursts, generally, appears to relate to the duration of the burst, the author observed the longest burst, among the complex

bursts in an EEG.

As seen in Table 3, there is individually some different frequencies and different ranges between frequency at the beginning and at the end of the burst. As to the correlation between the age and the frequency at the beginning of the pattern as observed in Fig.1, there is a tendency that the frequency increase with the advance of the age, but the frequency at the end appears to be unrelated with the age. In this series, frequencies above 4.0 c/s. and below 2.0 c/s. (between 4 years and 19 years of age) were not found. The author could not find any report that petit mal absence could show slower frequency of rhythmic complex bursts than 2 c/s..

b) *The other findings--spikes and random complexes. and the localization.*

In the resting EEG's 8 patients had random spike, multiple spikes or random irregular spike and wave complex, in addition to rhythmic spike and wave bursts. Although 3 patients of the pure petit mal showed them 3 of 6 patients with grand mal showed the bilateral spikes and 3 of 4 patients with focal seizure the localized spike or spike and wave. One with psychomotor seizure showed bilateral spike in the resting EEG, but the activation revealed evidently localized spike in the temporal area. And, in addition the activation provoked the localized spikes in the EEG of a patient with focal seizure who had no spike in the resting EEG, and so the lateralized epileptic discharges were demonstrated in all of the patients with focal seizure. To be interesting, among 3 cases of the pure petit mal that showed spike or complex, 2 (case 13 & 15) have the positive hereditary history and the other one is the only patient that has had no convulsive seizures, showing multiple spikes and wave complex bursts in the EEG.

To summarize the EEG findings, the signs of lateralization or localization were observed in 2 cases of the pure petit mal patients (13 cases), in one case of the patients with grand mal (6 cases) and in all of the patients with focal seizures (4 cases) and with psychomotor seizure (one case).

Case Report

The further information about pathophysiology of epilepsy may be obtained by analysing each individual cases and their EEG's. The author describes case histories of 3 cases of petit mal.

1. **Case 3 : Male**. He was first seen at the age of 9 years. Petit mals had begun one year before. His attacks were so peculiar that his parents noticed them and regarded them not as peculiar habits but as epileptic attacks. His parents mentioned, "he produced the black spells accompanied by the twitching of eyelids and of the left corner of lips, in frequency around 10 a day". And at the time he was suspected as focal epilepsy, because the mentioned features suggested focal epilepsy and his EEG at the time showed no characteristic paroxysmal discharges, but he produced a focal beginning, generalized convulsion by a small amount of megimide-metrazol mixture. However, 2 years later, a real beginning generalized convulsions had occurred and he was sent to

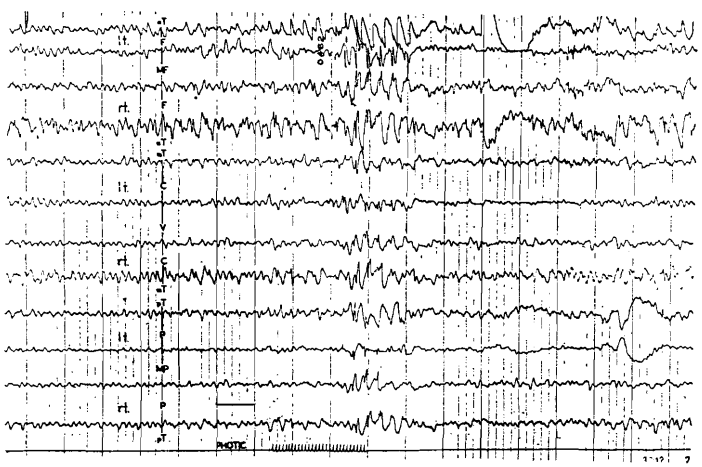


Fig. 3 (a) EEG of Case 3

In the first recorded EEG, only a short burst of spike and wave complex was showed by megimide-metrazol photic activation. This figure shows slow waves and spike forms, localised in the right anterior tempral, and the start of focal beginning convulsive seizure following the spike-and-wave complex pattern.

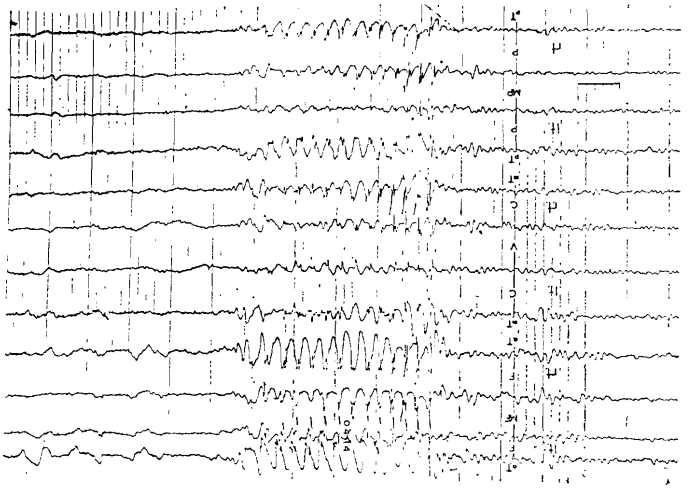


Fig. 3 (b). EEG of Case 3

In this EEG, with megimide-metrazol activation, he showed repetitively the complex pattern, combined with clinical petit mal attack in which the author notices the patient not to be in complete unconsciousness in the last half of it. Spike components of this complex pattern are predominant in the right side. Signals show 50 μv and 1 second.

our hospital. Then, it was found that his blank spells manifested always simultaneous occurrance with the bilateral asymmetrical rhythmic spike and wave complex burst in the EEG. And, the author observed him to twitch and left corner of the lips and to blink rhythmically, during the attack. In his EEG, random localized spike and wave complexes in the right temporal area were observed, too. In this case, it is interesting that not only a lateralized EEG pattern, but a lateralized motor symptom were seen in the petit mal attack.

2. **Case 14: Female.** This had been a healthy girl until 10 years when petit mal attacks occurred. Her attacks were noticed by her mother without difficulty, because they tended to appear in the form of status epileptics. She had the first convulsion-focal beginning generalized seizure at 3 months after the beginning of the petit mal. Since the convulsive seizure began, her emotionality has appeared to be distorted. And, the correlation between the state of emotion and the attack of petit mal was observed, especially getting a scolding provoked the attacks. Her status would last for 5 to 10 minutes, during when her actions were resolved into intermittent movement, because the attacks each of which lasted for around 15 seconds at the interval of around one second, interrupted her actions. The EEG pattern of her attack was characteristic always beginning at the right frontal area and showing highest amplitude in the area during attack. However, in spite of these localized emphasis, her attacks manifested no lateralized signs.

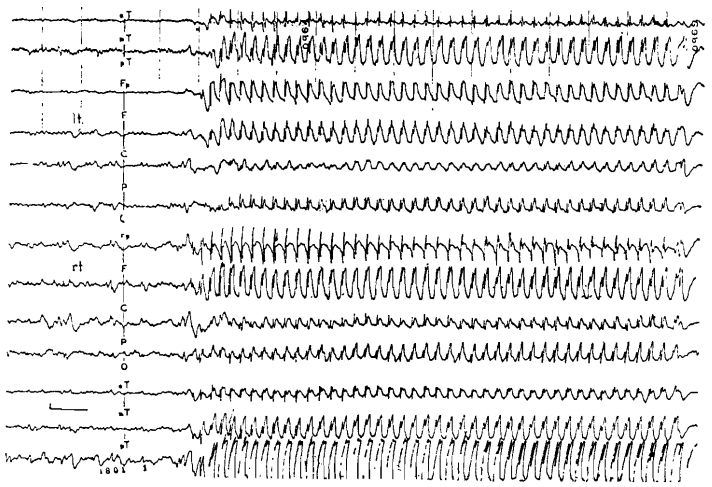


Fig. 4 EEG of Case 14

While her attacks ars symmetrical in behavior, the complex patterns begin always on the right frontal area and show predominant pattern in this area continuously during the attack.

3. **Case 11: Female.** This was a bright girl of 9 years who had neither positive past history nor heredity. Her attack first noticed at the age of 8 years when she would have blank spells

provoked by television. She would be often immobilized with staring eyes, following lightening-like jerking, when she switched on television. Her spells lasted usually for 30 seconds, during which she would show some responses, changing somewhat her posture or occasionally vocalizing, to her parents' calling. At the time of 9 years she was first examined and treated. To be characteristic in the EEG examination, her attacks would be well provoked by repetitive photic stimulation, but hard by the overbreathing. They responded to the repetitive flashes at the range of frequency between 5 to 50 c/s. (unknown over 50 c/s.) and always her attack began at the myoclonic movement. Her petit mal attacks were resistant to medication, and controlled only by 80% of the attacks, at the best. However, some changes were seen in the features of the attacks after medication: a) decrease of sensitivity to photic stimulation, b) disappearance of myoclonic jerk at the beginning of the attack, and c) improvement in the level of disturbance of consciousness. The latest was shown as

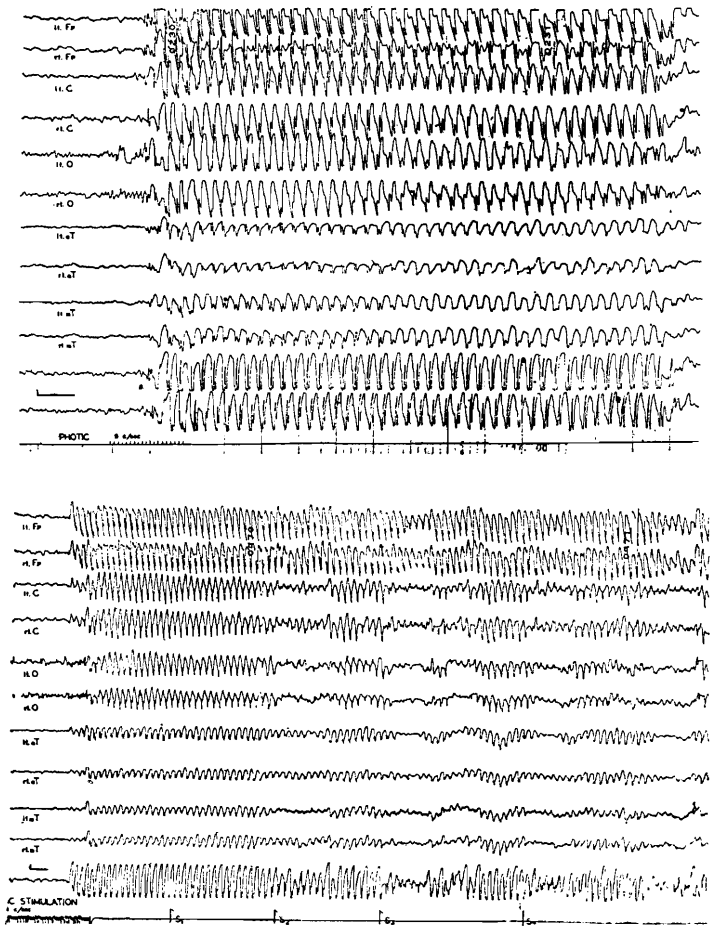


Fig. 5 (a and b)EEG of Case 11

Her attac ks would be provoked by photic stimulation and showed myoclonic movement at the beginning, combined with multiple spikes in the EEG, prior to medication. The monopolar recording (reference electrode in ear,) signal shows 50 μv, 1 second.

After medication, changes in her attack and in the EEG were recognized; the myoclonic movement and the multiple spikes at the beginning of it disappeared, the sensitivity to photic stimulation decreased and photic stimulation does not always provoked attacks even in high frequency, and finally the level of consciousness disturbance apparently was improved.
To calling her name at S_1, no res ponse in behavior nor in EEG. To calling hern ame at S_2, yes and remarkable suppression of slow waves, especially in occipital area. To '3', question, '1+2'at S_3, the answer with suppression of the patteren. To asking her name, at S_4 the answer her surname, with supperesion of the pattern.

follows: the responses that her parents noticed during attacks, became more evident and when examiner asked her name, her age or the addition of 2 signular numbers, she stared at him and could often answer clearly to the given brief questions, and always at the time of these responses were observed the EEG patterns changed evidently, manifested distinctly the transient deminution of amplitude of slow components (but, not evident in the frontal area) and emphasis of spike components that appeared to be covered by slow components. These transformations of the EEG pattern never occurred in the first several minutes, but after the period the sensitivity to the stimulations appears to go higher and higher with the progress of time. However, it is correct to

mention that she was not alert until the complex burst pattern transformed into the pattern of the interictal state, because although she remembered the contents of what she was asked, she did not remember exactly whether she was able to answer these question and how many questions were given to her, in spite of only 3 or 4 questions, and besides she could not answer the complicated questions. Before the treatment, her attacks were provoked always even by 5 c/s. flashes, but with the progress of treatment the sensitivity to the photic stimulation decreased, narrowing the range of frequencies to respond; at last the EEG responded only to higher frequency (to over 40 c/s.) and not always.

At the age of 10 years when she discontinued the administration of drugs for a few days, she occurred a grand mal seizure when she switched on the television. Since the accident, she has never occurred the grand mal seizure under the regular medications. The occurrance of convulsive seizure in this case is interesting to the report of Gastaut and Hunder (1950) who reported Tridione was effective to decrease photo-convulsive response.

Discussion

As above mentioned, it is widely accepted that petit mal absence is the episode of brief disturbance of consciousness, either with or without minor motor symptoms and/or autonomic symptoms, accompanying around 3 c/s. rhythmic spike and wave complex burst in EEG during the episode (Penfield & Jasper 1954, Lennox 1960). However, these motor or autonomic symptoms are not always manifested, as seen in this study and in the other reports. There, it should be resonable to regard the disturbance of consciousness as the essential symptom of petit mal absence. And the various manifestations of these motor or autonomic symptoms that are accompanied incidentally by the essential symptom (disturbance of consciousness) may suggest complexity of pathophysiology of petit mal absence. It is established that patients of petit mal absence have often the other type of seizures, mainly convulsive seizure, and so the prognoses of their attacks are not always fable. Gibbs & Gibbs (1952) mentioned that cases with both grand mal and petit mal epilepsy are about twice as common as those with petit mal only and Janz (1955) reported petit mal patients with the other seizures as over 50% in the number, Paal (1957) as 66% in this catamnestic study and O'Brien et al (1959) as 49%. In this study 54% of petit mal patients has the other seizures. Therefore, it may be correctly memtioned that around a half or more of them has and will have the other seizures catamnestically. The author showed that the multiple spikes, seen in 3 c/s. complex burst, and spike components in back ground activity relate to convulsive seizures of petit mal patients. Gibbs & Gibbs (1952) called multiple spikes of the burst as grand mal component by reason that "this indicates a tendency toward grand mal seizures, and the greater the chance that the patient has had or will have convulsive seizure". Okuma et al (1958) stated the same conclusion in their study on petit mal by metrazol activation. Besides, Gibbs & Gibbs suggested a close relation between convulsive seizure of petit mal patients and spike forms in their EEGs, and Paal (1957) reported the possiblity that the patients with spikes in addition to the complex bursts in their EEG, may have convulsive seizures in several years.

Although most of text books state that the frequency of the spike and wave complex burst of petit mal is around 3 c/s., the frequency is variable: faster at the beginning of the attack and slower at the end (Gibbs & Gibbs 1952, Chao et al 1958, Lennox 1960, Kiloh et al 1961). However, there are found few studies on the frequency of the complex burst of petit mal. In the result of present study, all cases shows variable frequency, and the highest frequency at the beginning of the burst is 4 c/s. and the slowest at the end is 2 c/s. As to the correlation between the age and the frequency of the complex pattern, Calderon & Paal (1957) reported that petit mal patients in the age below 10 years had frequently the complex pattern of 2 c/s. and (1957) showed that the slower frequency (2 c/s.) in the younger age changed into 3 c/s. in the frequency. And, Lennox (1960) and Gänshirt (1961) state that, 'generally, the frequency of the dominant rhythm is much faster in the older patients than young children. In the author's the frequency at the beginning tends, generally, to be the faster, the older; however, there is no significant correlation between them on the frequency at the end, because the frequency tends to

depend on the duration of the attack. The hypothesis that petit mal attack is prevented from growth into convulsive seizure by a mechanism of inhibition (mid-brain), has been widely accepted (Gastaut 1954). The tendency of slowing of frequency and of disappearance of multiple spike component in the later stage of the attack, appears to express the gradual increase of the mechanism, which brings an abrupt cease of attack without postictal change in clinical state and in EEG. And the mechanism in petit mal undoubtedly relates to the speciality of the juvenile brain.

Jasper & Penfield (1954) stated, "consciousness is invaraibly lost when the EEG shows higher voltage (over 1000 microvolt) of discharges involving both frontal and parieto-occipital region of the brain bilaterally". And it is well known, when the 3c/s. complex pattern is localized in one area or even in more areas, the pattern does not accompany clinical attack — and it has been called electroencephalographically the subclinical pattern. However, the relation between the amplitude of the pattern and the consciousness disturbance is complicated. EEGs of Fig. 5 (b) appears to show evidently corresponding relation between the amplitude slow wave component of the pattern and the consciousness: in any time when the patint can respond to some questions, the slow waves of the bursts are suppressed transiently. If the pattern of the time when the patient responds was observed continuously, it might be called the subclinical pattern. The response to stimuli could be regarded at the expression of deminuition of the depth of disturbance of consciousness. "Stupor component" which Gibbs and Gibbs called the slow component of the pattern, implies that the slow waves relate to the consciousness. However, there is no notable difference in the amplitude of the basic complex pattern between when the patient can respond to stimuli and when cannot. This means, in the complex pattern the amplitude itself does not express exactly the level of the consciousness. Shimazono et al (1953) have demonstrated that the amplitude of the pattern sometimes is unchangeable, even if the patient responds to stimuli. Therefore, as to the relation between the amplitude of the pattern and the consciousness, it may be correctly mentioned that usually the amplitude itself of the complex burst does not express adequately the level of consciousness, although the amplitude of the slow waves of the pattern might be relate to the level when the amplitude deminishes evidently in comparison with one of full size of the pattern, in an EEG.

A lot of experimental study on 3 c/s. spike and wave complex and on petit mal absence have been reported since Jasper and Droogleever-Fortuyn (1947). Among them, studies of Hunter and Jasper (1949), of Lennox and Robinson (1951) and of Ralston and Ajmone-Marsan (1956) are interesting and well known. However, the facts that petit mal absence shows the sharp age-selectivity and so tends to improve spontaneously in adolescence, may relate to the maturation or stabilization of the brain. It may mean, petit mal has some speciality, combined with the process of maturation of the brain. On the view point of this, man should be cautious, to explain or to supp osethe mechanism of this type of epilepsy on the results of animal experiments.

Against the animal experiments, experimental [or therapeutical studies on petit mal patients, reported by Hayne et al (1949), by Spiegel et al (1950, 1951), by Williams (1952), Kirikae et al(1952), Penfield and Jasper (1954) and Bickford (1956), appear to be much more of value, witout doubt. By the way, Jasper and Gastaut appear to believe "subcortical origin" and Gibbses "cortical origin". And, the Freiburg school imagines that petit mal is a reaction of the infantile cerebrum to any epileptic irritationwhich can act on various places within the brain.

Penfield and Jasper (1954) have postulated that diencephalon and upper brain stem are involved principally in petit mal attack and established the concept of "centrencephalic epilepsy". In actully, brief cons- ciousness disturbance combined with bilaterally symmetrical and synchronized discharges and the features of simultaneous course in clinical pictures and EEG, observeed in petit mal attacks, may give us a reason enough to believe that originally subcortical region may be involved and the discharges originating in the region may be projected through thalamocortical pathway (Jasper and Hunter 1949). Cohn (1954) observed asymmetry between homologious areas of both hemispheres in the pattern, by a detailed analysis of DC potential and course of it, and suggested that the pattern does not originate in common nuclei in both hemispheres, even if the firing was in the thalamus. Due to Torii et al (1962), convulsive discharges of the neuron in non-specific nucleus of the thalamus are projected to ipsilateral cortex and then to non-specific nucleus of contralateral

cortex from the latter. These results suggest the possibility of asymmetrical involvement in the subcortical nuclei, even if the discharge may fire in the region. On the other hand, against the asymmetrical pattern of the complex burst, Howell (1955) reported asymmetrical motor symptoms, observed during petit mal attack combined with typical 3 c/s. spike and wave complex burst. In the present study, the asymmetrical motor symptoms were described in 4 cases, all of them had the other types of seizure, too. Although 2 children with focal seizure showed lateralization in EEGs, 2 with grand mal had not apparently lateralized pattern, espeically case 19 had no abnormality ocurred more easily when Metrazol was injected into carotid artery which supplies cortex, putamen, caudate nucleus and the pre-optic area of the hypothalamus, than when into vertebral artery which supplies thalamus. And Shimizu et al (1952) reported the similar results to Bennett, by their animal experiment. These results show not only a possibility of cortical onset of the pattern, but do not deny a possibility of onset in subcortical structure except thalamus. In present study, focal beginning pattern of the complex and localized, asymmetrical predominance in it were expressed. And Paal (1957) and O'Brien (1959) have reported similar EEGs, too. These findings may suggest localized dysfuncion of cortex of these cases, in the following hypothetical interpretation: 1) a cortical focus is primarily activated and its firing trigger to involve the whole brain, 2) dysfunction or hypersensitivity of a localized area of cortex produces exaggerated response to diffuse bombardment from subcortical region. The supposition of 1) is supported by the report of Bickford (1956) who proved that petit mal attack can be triggered by localized sharp wave and is produced by cortical stimulation.

Conclusion

Consequently, the following supposition as to mechanism of petit mal may be reasonable at the present stage of study: both cortex and subcortical system are necessary for maintenance of the complex pattern and they make up a linkage in that subcortical firing produce cortical spike and the latter cause the former, and so on. And, cortex is capable of triggering the complex pattern, as well as subcortical structure. Something which regulates this chain reaction may be mechanism of inhibition, characterized by maturation process of the brain.

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