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Diencephalic Disorders *

With a Review and Case Presentation of "Periodic Somnolence" (Kleist & Kleine)

Suckjin Yoo **, M.D.

Captain, MC, ROK-Army, Neuropsychiatrist, Fitzsimons Army Hospital; Assistant Prof. of Neuropsychiatry, Seoul University, R.O.K.

It was as early as about four and half decades ago that Reichardt[1] has, on the basis of his vast clinical materials, clearly demonstrated the important role of the brain stem as the centre of the vegetative and somatic as well as psychic functions, while people could not yet experience the studies of encephalitis[51], which contributed to show the role of the brain stem, and especially concerning the seat of the psychic phenomena people believed only in the brain cortex at that time.

Since Reichardt, clinical literature relating to the diencephalon and the disorders arising from its changes has rapidly increased more and more, and has been summarized recently by Störring[2], Alpers[3] and many other authors. It is possible to recognize the following five distinctive groups of syndromes of the diencephalon described in the previous literature: I.Disturbances of Somatic Functions, II. Disturbances of Vegetative Functions,III. Emotional and Personality Disturbances, IV. Epileptic Disorders, V. Sleep Disorders.

I. Disturbances of Somatic Functions

All levels of central nervous integration so far studied exhibited a dovetailing of autonomic and somatic representation, and the diencephalon is no exception. Especially the thalamus, the dorsal part of the diencephalon, is the great subcortical sensory station; it receives all sensory stimuli with possible exception of taste, before sensation passes on to the cerebral cortex. There is, however, little data concerning somatic integration and primate hypothalamus which is separated from the thalamus by a well defined sulcus. Lesions of this diencephalon, especially of the thalamus, are supposed by many authors to produce characteristic somatic syndromes as follows:

1) Thalamic Dysesthesias

a) Diminution or loss of sensation of the opposite half of the body; the most frequent defect in the sphere of deep sensibility, localization and stereognosis (Bonhoffer[4]).

b) Thalamic hemihyperpathia: hemihyperesthesia or hemianesthesia to touch, pain, and temperature (Bonhoeffer[4]).

c) Spontaneous, often intractable, pain sense; for example, intestinal pains in a case of encephalitis (Bonhoeffer[4], Schuster[5]).

d) Characteristic paresthesia and autotopagnosis Bonhoeffer[4], Pap[6], Nielson[7]).

2) Transitory flaccid hemiparesis followed by thalamic sensory disturbances (Brock[8]).

3) Thalamic involuntary movements

a) Hemiataxia dependent on sensory loss or disruption of cerebello-rubro-thalamic connection (Brock[8]).

b) Lower mimetic facial paralysis on the opposite side with perseveration of the voluntary innervation (Schuster[5]).

c) Choreo-athetoid movements which are usually marked in the upper extremity (Bonhoeffer[4]).

d) Tremor (intention tremor) which is likely dependent upon disruption of rubro-thalamic connection (Brock[8]).

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** Present Address: Director of the Petrus NP Hospital; Chairman of the Professional Committee of the Seoul Child Guidance Clinic; Secretary General of the Korean Association for Mental Health; President of the Korean Neuropsychiatric Association.

4) **A choreo-athetoid posture** of the opposite upper limb due to disturbances in thalamo-hypothalamic connections in case of vascular lesions in the thalamo-perforating arteries (Walker[9]) : the fingers and hand and other parts of the affected limb are held as if frozen in a choreo-athetoid posture, i.e. the hand is cupped, the wrist flexed and the fingers overextended; the subcutaneous tissue may be swollen and the skin warm and glossy; the hand and fingers are usually painful; there are no sensory defect.

5) **Visual defects** : homonymous quadrantic and hemianoptic defects in the opposite visual fields due to lesions affecting the pulvinar and especially the lateral genicurate body (Brock[8]); or bilateral hemianopsia due to lesions of hypothalamic region especially due to involvement of the hereby optic chiasm (McDonald[10]).

Ⅱ. Disturbances of Vegetative Functions

Thalamo-hypothalamus serves as a central station in which various sensory stimuli, so necessary to the maintenanae of emotional activity, are able to play upon the centres concerned with instnctive drives. From this sensory effect organ, impulses are sent to vegetative centres in the hypothalamus, so that the organism may respond with appropriate visceral reflex to the dominant emotional state. In one who suffers "chronic unexpressed rage" because of the inability either to satisfy his "oral" demands or his ambitions, independent strivings, the emotions can act on autonomic centers as to lead to psychosomatic disease as diverse as essential hypertension, peptic ulcer, asthma, arthritis etc. (MacLean [11]).

Not only by emotional states, but also by organic, toxic humoral, and other pathological changes, the diencephalon may show many evidences of disturbed vegetative functions, especially when these changes are localized in the hypothalamus, which contains the hypophysis as one of its parts and is considered to be the head ganglion of the autonomic nervous system. According to Gagel[12], following classification of vegetative disturbances may be possible in cases of lesions of the autonomic centre in the diencephalon.

1) Disturbances of temperature regulation : hyper- or rarely hypothermia.

2) Disturbances in metabolisms;

a) Disturbances in water metabolism and diabetes insipidus,

b) Disturbances in fat and lipoid metabolism, adiposity or progressive lipoidystrophy,

c) Disturbances in sugar metabolism etc..

3) Vasmotor disturbances.

4) Disturbances of secretions of sweat, lacrimal, salivary, and mucous glands.

5) Disturbed functions of smooth muscles of bladder, uterus, and of rectum: tenesmus or incontinentia of bladder, obsitpation, and diarrhea etc..

6) Disturbances of gastrointestinal mucous membrane: gastric ulcer etc..

7) Changes of the heart activity: bradycardia, arrhythmia and etc..

8) Changes of the blood components.

9) Influences in growth and sexual development.

Ⅲ. Emotional and Personality Disturbances

The diencephalon was clearly proved by experimental works of Bard[13], Cannon[14], and others, as a region of the brain in which highly organized emotional reaction patterns as well as visceral one are integrated. Papez[15] has proposed that the thalamus and the hypothalamus, the cingulate gyrus, the hippocampus and their connections may serve as a structural and functional unit for emotion: in his formulation the diencephalon plays a very important role in emotional integration of personality.

A number of observations attest to the fact that disorders of the diencephalon are sometimes associated with disturbances of emotion and personality. The cerebral cortex particularly its frontal portion, is held as responible for such disturbances. While it is true in general, there remains the fact the personality and emotional disturbances are prominent in a small, both irritative and destructive lesion of the diencephalon, with no evidence of disease of cerebral cortex.

A. Thalamic syndromes in the personality and emotional sphers

1) Hypersensitivity to sensation and feeling which may be parallel with peripheral hyperpathia (Bonhoeffer[4]).

Foerster[16] belived that the thalamus is responsible

not only for the organic conditioned spontaneous pains and others, but also for psychogenic pains and hypersensitivity to pains in neuropathies. He explained the latter as the result of the weakness of the cortical or strial inhibitory apparatus which supervise the thalamic functions.

Schuster[5] observed a thalamus patient who showed depressive dejection and terror provoked merely by touching or talking.

2) Hallucinations as brain stem's syndrome: optic hallucination, or delirious hallucination which is mostly of optic nature (Kleist[17]).

3) Somatic delusions and ideas of referrence which are considered to be parallel with thalamic dysesthesias (Störring[2]).

Störring's[2] case-3 who was a postencephalitic Parkinsonism and showed evident thalamic syndromes, developed schizophrenic-like delusions. The author assumed that schizophrenic delusions may develop possibly from thalamic lesions. He cited other similar cases, encephalitic cases with schizophrenic-like states described by Bürger and and Mayer-Gross,[18] and Stern, F[19].

Reichardt[1] also earliest recognized that many clinical features of the schizophrenic reaction, as catatonic excitements, stuporous states, hallucinatory syndromes etc., could be produced by disturbances of the brain stem. But in the true schizophrenia, he thought, that process changes of this region are as finer as to be found uneasily, than in coarser schizophrenic-like syndromes in cases of general paresis, encephalitis, and other coarse organic brain disorders.

4) Dementia in a remarkable instances of progressive degeneration of both thalami(Stern,K[19]).

In this case, Stern described severe dementia, marked hypersomnia, iridoplegia and grasping and sucking movements. Stern emphasized the "isolation of vast areas of the cerebral cortex on both sides" resulting from the loss of thalamic impulses.

B. Hypothalamic syndromes in the personality and emotional spheres

Kleist's observations[17]

1) Time-amnesia.

2) Disturbances of recollection.

a) Pathological light recollection: logorrhea, and others.

b) Pathological difficult recollection: perseveration.

3) Disturbances of intuition and meaning: déja vu, misinterpretation, delusion, ideas of referrence.

4) Disturbances of diencephalic attention which control the thought progress.

a) Pathological stimulated states: flight of ideas, incoherence.

b) Pathological inhibited states: inhibition of thoughts.

5) Hysteric syndromes or suggestibility due to disturbances of diencephalic "body-ego".

6) "Primitive-ego (instinctive-ego)" disturbances seen frequently in brain injury cases.

a) Excited instinctive behaviors:

i. agressive or explosive reaction, similiar to epileptic excitement.

ii. immature and hysteric-like reaction or labile psychomotor reaction similiar to delirium.

iii. non suppressed sexual behavior.

b) Diminished instinctive behaviors:

iv. Stuporous and negativistic behavior.

7) "Affective-ego" disturbances: temperament changes (melancholic or hypomanic), labile affectivity.

Sterts's interpretation[20]

1) Inhibition or hypofunction of the "general psychic energy-level".

a) Thought disturbances: dementia.

b) Memory disturbances: Korsakoff syndrome.

c) Disturbances of feeling and affect: flat euphoria, or spontaneous and reactive apathy.

d) Disturbances of activity: stupor, negativism.

2) Hyperfunction of the "general psychic energy-level": hallucination, delirium.

Alpers's analysis[3] **of clinical data**

1) **Emotional changes.** There are no constant changes in the emotional reactions of patients with hypothalamic lesions (Alpers[3]). But following types of changes were reported by many authors.

a) Excitement: a common and almost constant response; manic-like reactions with push of speech and ideas, and excitement (Foerster[21], Fulton and Bailey[22], Cushing[23], Cox[24], Stern, K. and Dancy[25], Urechia[26], and others).

Characteristics are:

i. From the descriptions of the reactions it is

difficult to see how these can be differentiated from the manic excitements seen in brain disorders such as general paresis, elpepsy, and other disorders.

ii. They are episodic in nature, lasting only as long as the stimulus in many instances, but in others the excitement continues for days following operation.

iii. They resemble them by being undirected and blind.

b) Swings of mood: Alternating moods of depression and excitement (Alpers[3]), or emotional lability, with a trigger response of excessive emotional reaction such as uncontrollable laughing (Dott[27]).

c) Anxiety feelings: not uncommon (Guttmann and Hermann[28], and Grinker[29]).

d) Apathy and negativism: sometimes seen (Cushing[23]).

2) Personality changes. The number of recorded instances is not yet great, but additional evidence is accruing constantly. Following clincial features were portlayed clearly in the case of Alpers[3], in a case of Cox[24], and in a Dott's case[27] of the openly amorous advances of a very proper old gentleman:

i. A reversal of the customary personality trends,

ii. a lack of inhibitions with the subsequent development of coarse traits,

iii. a failure of appreciation of many of niceties of life,

iv. carelessness in habits, indifference to surroundings and to obvious anti-social tendencies,

v. and a partial or complete loss of appreciation of personality changes in the patient himself.

Same featrures has also been seen in a Stern, K. and Dancy's case[25] of a young woman who showed a lack of inhibitions when her case was presented before a group of students. In reported cases[17,53,55~79] and my new cases of "periodic somnolence" which will be discussed later in this paper, there are also many of these features portlayed.

These changes, like the emotional disorders in hypothalamic lesions, are transient in nature in cases which servive. They disappear completely when the pressure on the hypothalamus is relieved by operation (Cox[24], Dott[27]), they persist and become intensifield when the lesion persists and spreads (Alpers[3]). In these cases there is no evidence of cortical damage like as in cases of emotional, intellectual, or other disturbances.

3) Intellectual deficits.

a) Loss of memory (Alpers[2], Cox[24], Schilder and Weissmann[30], and Fulton and Bailey[22]).

b) Inability to concentrate (Cox[24]).

In our present state of knowledge there is no way of differentiating these constantly reported intellectual difficulties from those of frontal lobe disease (Alpers[3]).

4) Pyschotic manifesattions in other mental spheres.

a) Korsakoff psychosis: not uncommon (Foerster[21]).

b) Confusion, disorientation (Alpers[3], Schilder and Weissmann[30], Cox[24]).

c) Hallucination and delusion: Scattered observations of these experiences have been recorded in cases with hypothalamic lesions of various sorts, usually tumors (Schilder and Weissmann[30], Dott[27], Lhermitte and others[31]). It may be pertinent to point out that the hallucinations recorded have all been of visual type; only few instances of auditory hallucinations have been found. This probably has some relation to the adjacent centres in the diencephalon, which is conceivably irritated by the hypothalamic lesions, whatever its nature may be.

Ⅳ. Epileptic Disorders

1) Petit mal, absence, highest level seizures (Penfield and Jasper[32])

Penfield and Jasper[32] proved the diencephalon and perphaps mesncephalon as the highest level of gray matter the function of which is more intimately related to consciousness process than other area of the brain; and assumned this area as the source of origin of the bilaterally synchronous electrical discharges which are characteristic of petit mal, and they called the petit mal as the highest level seizures. Jasper and Fortuyn[33] could experimentally produce all of the characteristc EEG of patients with petit mal, in cats by rhythmic electrical stimuli by brief shocks to a small area, in the medial intralaminal region of the thalamus.

"**Grand mal**": This is, according to Penfield and Jasper[32], nothing but a symmetrical generalized

convulsion which follows the initial loss of consciousness and begins without significant turning, when the discharge of petit mal is a severe one. Once this generalized attack is under way, it resembles a major cerebral seizure, which has followed any other type of minor seizure, both electroencephalographically and objectively. The term "grand mal" is a time-honored one which contributes nothing at all to our understanding.

2) Epileptic automatism, "dreamy states" (Jackson, H.[34,35]).

Jackson[34,35] associated this state with discharges of regions in the uncinate region; but in some of his autopsy cases, the lesions extended into the tip of thalamic lobe.

Since the introduction of the EEG it has been shown that in this type of epilepsy there are abnormal electrical discharges arising in the region of one or both temporal lobes; and this type of epilepsy has been called as temporal lobe seizures. But Jasper, Penfield and others[36], from their clinical and EEG studies, concluded that the regions primarily involved in these attacks may be in subcortical structures related to the temporal lobe. The same possibility was also pointed out by Lennox and Brody[37].

MacLean, in association with Arellano[38], related to the basilar part of the rhinencephalon. On the other hand Walter, Dovey and Cobb[39] related to deep-seated lesions in the general vicinity of the third ventricle. Marsan and Stoll[40], recently from their experimental and clinical studies, suggested that in at least some cases bilaterally synchronous discharges and unilateral abnormalities recorded in the EEG of patients with temporal lobe seizures may be of subcortical origin.

3) Thalamic and hypothalamic epilepsy (Gibbs and Gibbs[41]: 14 & 6 per second positive spike discharges)

Gibbs and Gibbs[41] recently reported that the clinical correlates of 14 & 6 per second positive spike discharges suggest epileptic disorder in the thalamus and hypothalamus; attacks of pain, rage, vegetative symptoms were common. In the patients who were reported by Stephenson[41], neoplasms were verified at operation and anatomic location was as such as to afford some supports to the thesis that the source of the 14 & 6 per second positive spikes is in the diencephalon.

4) Autonomic epilepsy (Penfield[42], Cushing[23]).

Symptoms of diffuse discharge of the autonomic nervous system in otherwise normal individuals have been reported both for the sympathetic and the parasympathetic divisions of the system. Generally, however, there is some overlapping between the two divisions. Predominantly sympathetic seizures were described by Penfield[42] in the case of small ball-valve tumors of the third ventricle, and Cushing [23] noted that parasympathetic outbursts occur in response to intraventricular injections of pituitrin and pilocarpine.

Ⅴ. Sleep Disorders

Apparently opposed to Pavlov's view[43] that sleep is primarily a cortical inhibitory process, Dubois[44], Demole[45], Hess[46], Ranson[47] and many others have experimentally demonstrated that there exists a localized area which is intimately concerned with the regulation of sleep, at a lower level of the nervous system especially in the diencephalon. Many clinical and pathological observations on various clinical features of sleep disorders classified as follows will also support the experimental evidence.

A. Prolonged sleep states:

1) Non periodical lethargy as the result of following conditions:

i. Cerebral tumor. Righetti[48] found hypersomnia in 115 out of 775 verified cases of cerebral tumor, its frequency varying according to the distribution of the lesion. Thus in tumors of the thalamus and third ventricle it was present in 15.8% of cases, in medullary tumors 27.9%, in tumors of the hypophysis and vicinity 36.5%, of the cerebellum 15.8%, of the central gyri 10%, of frontal tumors 6%, etc. Lhermitte and Tournay[49] quoted another series of cases in which sleepiness was present with tumors involving the parietal, occipital and temporal lobes, the corpus callosum, optic thalamus, pineal body, corpora quadrigemina, peduncle, pons, cerebellum, medulla and hypophysis. The frequent occurrence of sleepiness in patients with tumors in or about the pituitary is well known. Lhermitte and Tournay

[49] draw the conclusion that the common factor in all the cases is direct or indirect pressure upon the third ventricle, direct pressure being especially common with pituitary tumors, and indirect pressure being often the result of distension of the third ventricle by hydrocephalus, to which a tumor remote ly situated may give rise. Walter, Criffiths and Nevin[50] reported a case of pathological somnolence caused by a tumor of the hypothalamus in which the electro-encephalogram resembled that found in deep natural sleep and could be differentiated from the electroencephalogram both of ether anaesthesia and of coma due to increased intra cranial pressure.

ii. Epidemic encephalitis. The sleep disturbance of encephalitis lethargica can be correlated with the predilection of virus of this disease for the gray matter around the anterior end of the aquaeduct of Sylvius, which led Von Economo[51] to posstulate a sleep centre in this region.

iii. Other infective conditions of the nervous system, especially syphilis and trypanosomiasis (Brain and others[52]).

iv. Cerebral arterio-sclerosis (Brain andothers[52],)

v. Head injury (Brain and othars[52]).

vi. Exhaustive and febrile diseases (Kleine[53]).

vii. Autotoxic states such as diabetes and uraemia, and soporific drugs (Brain and others[52]).

viii: Neuropathic or psychopathic personality (Laudenheimer[54], Daniel[55]).

2) Periodic somnolence (Kleist & Kleine[53]): In this disease, there are attacks of sleepiness occurring periodically and lasting from several days to several weeks. The longest record was three months. During the attack the patient sleeps excessively day and night, in extreme instances walking only to eat and go to toilet. He can always be roused. When roused he usually is irritable and wants to be let alone so that he can go back to sleep. These attacks are separated by intervals of normal health.

This disease was very rarely reported. In the literature that is available to me, I find reports of only about 30 cases or less of "periodic somnolence". These were reported by the following authors in the chronologic order given.

i) **Anfimoff**[56] (1889): 1 case, cited by Kanabich[61] in 1923, reported again by Kaplinsky and Schulmann [69]-I. (Case 4), 1935.

ii) **Rhode**[57] (1912): 5 cases (case 60-64), mentioned by Kleine[53] as doubtful, 1925.

iii) **Stoecker**[58] (1913): 1 case (case 5), cited by Kleine[53], 1625.

iv) **Schröder**[59] (1918); 1 case (case 7), cited by Kleist[17] in 1921, rep orted again by Kleine[53], 1925

v) **Krüger**[60] (1920): 2 cases, cited by Kleine[53] in 1925.

vi) **Kleist**[17] (1921): 2 cases,reported again by Kleine[53] (case 2 and 4).

vii) **Kanabich**[61] (1923): 1 case, cited by Kaplinsky and Schulmann[69]-1. (case 3), 1935.

viii) **Goldflam**[62] (1924): Few cases, referred by Stadler[70], 1938.

ix) **Kleine**[53] (1925): 3 cases (case 1, 3 and 5).

x) **Lewis**[63] (1926): 1 case (observation iv), cited by Levin[66], 1936.

xi) **Stiefler**[64] (1927): 1 case (postencephalitic case cited by Stadler[70] in 1938.

xii) **Campbell**[65] (1927): 1 case (postencephalitic case), mentioned by Daniel[55], 1934

xiii) **Levin**[66] (1929): 1 case (case 7), referred by Daniel[66], 1934. and reported again by Levin[66] in 1936.

xiv) **Tsiminakis**[67] (1930): 1 case (post-dengue-fever case), mentioned by Daniel[55], 1934.

xv) **Redlich**[68] (1931): None of new cases.

xvi) **Daniel**[55] (1934): 1 case

xvii) **Kaplinsky and Schulmann**[69]

I. (1935): 2 cases (cass 1 and 2).

II. (1935): 3 cases (case 1, 2 and 3).

xviii) **Levin**[66] (1936): None of new cases.

xix) **Stadler**[70] (1938): 1 case (case 1), with other cases of hypersomnia.

It is possible, in these previous reported cases, to find that the "periodic somnolence" syndrome may be associated with following varied pathological states:

i) Chronic encephalitis (Stiefler[64], Campbell[65], Levin[66]).

ii) Hydrocephalus (Schröder[59], Kaplinsky and Schulmann[69], Stadler[70]).

iii) Post exhaustive or febrile-disease (Rhode[57], Stoecker[58], Schröder[59], Krüger[60], Kleist and Kleine[53], Tsiminakis[67], Daniel[55], Stadler[70]).

iv) Endocrine or metabolic disturbances.

a. Disturbances of sexual and thyroid gland (Krüger[60], Kleist and Kleine[53], Goldflam[62], Kaplin-

sky and Sculmann[69], Stadler[70]).

b. Acromegalia as the result of hypophyseal disturbances (Stadler[70]).

v. Neuro- or psychopathic personality (Rhode[57], Krüger[60], Kleist and Kleine[53], Goldflam[62], Lewis[63], Daniel[55], Kaplinsky and Schulmann[69], Stadler[70]).

These cases should not be confused with the shorter lasting sleep attacks, Gelineau's syndrome[71]) (Kleist [17], Kleine[53], Redlich[68], and others), or with other types of sleep disturbances. But **Kleist and Kleine**[53], who have first tried to give the nosologic nomenclature of this disease-"periodic somnolence"(**Periodische Schlafsucht**), interpreted this disorder as a constitutional disease, which is supposed to belong to a certain larger type of disease group, with epilepsy, pyknolepsy, narcolepsy, episodic terror, dipsomania, migraine-psychosis(Ranzow[72]), poriomania, and episodic twilight states(Kleist[17]). Kaplinsky and Schulmann[69], and Stadler[70] had also similiar nosologic concepts. Daniel[55], who in his long paper concluded that the malady narcolepsy was attributable to a disturbance in the floor of the third ventricle, discussed the periodic somnolence in the same paper as the neighbour disease of narcolepsy. I want also to think assumably that the periodic somnolence could be interpreted as one of diencephalic disorders, as well as narcolepsy or other types of somnolence, on the basis of following clinical observations of characteristic symptoms of reported cases.

i) Somnolence lasting long.

ii) Transient personality and emotional changes (apathy, indifference, lack of inhibition, swings of mood, excitement, irritability, restlessness, depression, anxiety, stupor, negativism etc).

iii) Transient hallucination, confusion, loss of memory, and difficulty of thinking etc.

iv) Hypersensitivity, Paresthesias and hyperactivity of reflexes.

v) Vasomotor changes, slowing of pulse, and cyanosis.

vi) Hunger and polyphagia, and polyuria.

Levin[66] thoght that this "hunger and polyphagia" might be the main and commonest symptom of this disease as well as somnolence itself, when he reviewed in 1936, previous 10 cases of periodic somnolence including his own case which had been reported in 1829 merely as a non-narcoleptic morbid somnolence with possible encephalitic process, but referred by Daniel[55] in 1934 as a periodic somnolence case. Among 10 cases which he reviewed, 7 cases were cited as good cases of what may be called "periodic somnolence hunger" according to himself. He cited Fulton's theory[73] to explain the hungry syndrome and offered his hypothesis that the morbid somnolence-hunger is due to excessive inhibitability of the highest centres, frontal lobe. But Reichardt[1], and Kaplinsky and Schulmann[69] have already earlier pointed out that "hungry and polyphagia" could be considered as a diencephalic syndrom.

vii) Acromegalia, struma, or hypogenitalismus.

viii) Headache, dizziness etc.

ix) Dilatation of third ventricle, or changes of sella turcica.

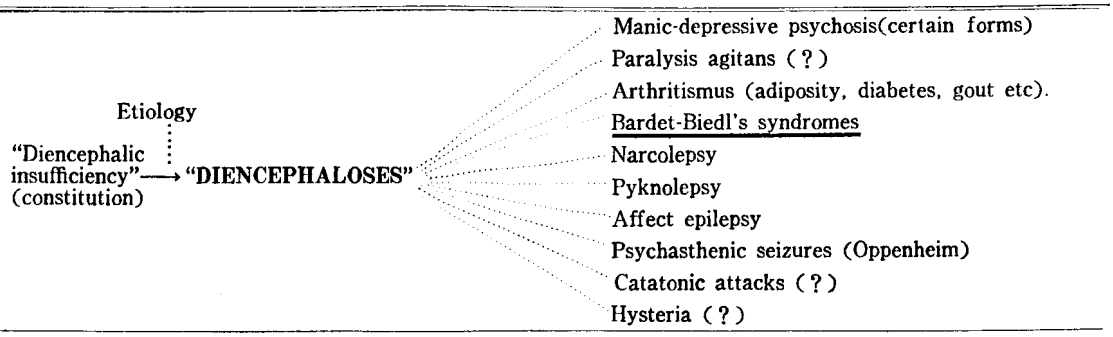
x) Onset at puberty, periodical occurrence, and reversibility.

xi) Usually good prognosis and curability by spinal puncture in certain cases.

These syndromes are not always constant in each individual case, but as a whole it seems, as Kaplinsky and Schulmann[69] concluded, that these syndromes are seemingly combinations of organic lesional processes, with organic process-like insufficiency which appears periodically, possibly under the influence of an occurring changes of metabolism and endocrine equilibrium. **Ratner**[74] raised, under the concept of "diencephalic insufficiency", certain phenomenon(Bardet-Biedl's syndromes[75]) as characteristics of a disease group (**diencephaloses**), which is associated with the constitutional weakness of vegetative and trophic centre in the diencephalon, and he counted narco- and pyknolepsy and many other syndromes or disorders for diencephaloses as shown in the following table 1. The "periodic somnolence" might also be assumably counted as one of these diencephaloses.

Because of the rare cases reported in the literature, as described above, it will be surely of importance to observe more cases carefully in the future and to add more evidsnces which may prove the true nature of this disease. And now few more additional cases will be reported in this paper.

Table 1



Case 1 : P.Y., a 17-year-old school boy, admitted for consulting on the indefinite psychiatric problem which was later found as sleeping attacks. This was seen in 1948 as the first case of periodic somnolence in our university clinic. During the attacks in this case, the personality changes were dominated among the clinical features, while the patient was usually sleeping not deep and roused so easily that one could not realize exactly about his sleep. And he was first thought as schizophrenia or some other psychopathic disorder by both his family and doctors.

But by the past history taken carefully, it became quite evident that since 2 years before the patient had sleep attacks which occurred periodically 2 or 3 times in a year and lasted a few days to 2 or 3 weeks each time; and that during the between periods the patient came back to quite normal healthy conditions. He didn't go to school by the attacks and stayed at home to got to bed. When the attack eventually began in the day time, he had to stay at school on the first day of attacks; but he was thought by his teacher just as lazy at such time. He was otherwise amenable and docile, but was very difficult to manage during the attacks, like as Daniel's case[55]. He did sleep not so deep enough that he could be easily aroused only by knocking the door. He was irritable, sensitive and restless. He was also immature and indifferent, and got angry very easily toward the parent or nurses. When admitted, he spit at the people who came in to his room, or shouted loudly to let the people leave the room. Sometimes it was found in the ward that he kept some big bottle or pillow among the bed. He also used to enter deep into the bed and never let the visitors see and talk to him, and refused to answer. He was entirely naked in th bed, and he was also found masturbating. He ate excessively, and asked grotesque to the nurses for bringing more and more food. While he ate foods, he sometimes became suddenly 'not 'interested in the nursing care, and the nurses left him alone; then the patient ate in the bed sleeping on and off. During the attacks, he didn't come out from the room except when he went to the toilet. But when he came out, although he seemed quite drowsy, he usually found something unnecessary like as some bottle, can etc. to take those into his room, bed.

He was admitted through 4 weeks. From the 3rd week end his sleep became little deeper. He was rather quiet at that time, because he slept by day and straight through the night. After the coarse sleep states was over, he began to come out from his room oftener and tried to talk to the people, but during few days then he looked still drowsy and tired, and used to go back to his bed minutes or hours. Personality changes became improved gradually to normal, but there remained for a while some difficulties in thinking processes.

The patient was developed normally in height and weight. Throughout the attack he had no fever at all. There had been no polyuria. Exophthalmus and struma existed both in slight degree. The patient complained headaches and dizziness, and was hypersensitive to the physical examination and made marked defensive motions against sensation or reflex tests. He seemed sometimes rather demonstrable. Reflexes were hyperactive, but no pathological reflexes were found. Chest and abdominal diseases couldn't be found. Results of labortory examinations were negative. There was nothing particular of the physical and laboratery finding except few syndromes

found as described above. Past history: nothing particular to describe. Family history : Uncle had headaches, brothers (elder) had same. Catanamnesis: uncertain.

Case 2 : T.K., a house wife, aged 23 was admitted to the same Seoul University Clinic in January 1950, with the complaint that she had already 9 attacks of somnolence occurring every month. She had always been well except that she was once told by the school doctor about her lung conditions little badly during the high school days. She was always adequate and average in her school, family and marriage life. No evidence of hereditary diseases was found in her blood relation.

One day in April, 1949, about 2 months after the birth of her first baby, she got the first attack of somnolence. She didn't get up in the morning. But she hadn't any good reason why she was too sleepy on that particular morning. She slept all the day and didn't care her baby. The patient was never so otherwise, but on that day she looked extremely drowsy and tired, and she was not interested in every thing. But she was quite all right on the next morning. And so her family didn't think about the attack seriously.

But in the next month, 30 days after, she got another similiar attack. At this time the attack lasted 2 days, and her husband began to think about curiously and brought her to docters. During the attack she was easily roused but quite restless and talked little more than usual when roused. She was thought by a doctor as having an equivalent syndrome of epilepsy, and was recommended to take antiepileptic medications and general hygienic cares.

Any kind of medications and treatments were ineffective for her disease, and she had had every month the same disease which repeated with regular 30 days interval of onset and seemed to become worse each time. The duration of attacks had usually prolonged each time one or half day more than that of the former attack, and sleep had become more deeper at the terminal period of the attack each time. The last attack before coming to our clinic lasted 7 days. Between attacks the patient was comfortable and there was nothing wrong except that the patient began to feel certain anxiety for the next attack to come. She could herself feel the new attack is occurring, from 2 or 3 days before the attack of somnolenae every month; she felt herself her swings of mood. Family around her also could recognize her initial changes in mood and facial apperance in which exophthalmic and cyanotic changes were characteristic. When admitted, the patient told that the periodical occurrence of the dissase was alike that of her menstruation which was also usually accompanied by swings of mood.

After admission, she had 2 attacks in February and March, which both appeared after 30 days interval of onset and lasted 9 days. About 2 days before the attack the patient could tell her changes in mood. She began to talk little more than usual at that time. But the patient herself didn't feel much comfortable and just couldn't express her feelings,— she just knew there were some changes. Exophthalmus and struma(not too big enough cough) could be recognized since that time to the end of the attack period. She used to go to urinate quite often during few days before the attack. During the attacks she slept on day and straight through the night. Eyes were closed, and the patient showed characteristic defensive motions, having still definite contact with her surroundings, and could be roused at anytime when she needed to go to the toilet. She complained her tiredness, sleepiness, vague headache and dizziness. She became indifferent and lost interets in every things. She feeded the baby just passively falling into sleep and didn't know when her baby was taken away after feeding. When she was requested to rouse to talk to the docter, she didn't care her cloths and hair etc., and talked little more hypomanic than usual seemingly having lost inhibition. Although she never got excitement, she got little confused states from the 7 th day of the attacks, and became restless, delirious and disoriented. This later part of periods of the attacks were followed by amnesia for events during the attacks. After this confused states passed during 2 days the patient became clear minded more and began to try to come out from the bed. This patient didn't show any big appetite during the attacks. She didn't refuse to eat, but didn't ask for foods herself. She just adopted to the nurse's care. She didn't

show any changes in sexual attitudes. During the interval period, she felt very healthy feeling and took care of her baby as usual or did everything just normally. She had no insomnia and difficulties in thinking, which some of reported cases had so on after the attack.

During the attacks, careful physical examinations were made. Exophthalmus and struma existde, and cyanotic changes of the lips and slowing of the pulse were characteristic. Dermographismus was [positive.] There was a coarse tremor of the tongue and slight fine tremor of the fingers. Tendon reflexes were hyerapctive, but there were no pathological reflexes. There were no marked dysesthesias. The patient was normally developed, and showed no signs of acromegalia. Gynecological findings were normal. The patient had no chest and abdominal diseases, and her tonsils or teeth were intact. Temperature was always normal. And no other physical syndromes could be found except some positive syndromes described above, which were trasinent during the attacks.

Laboratory examination : The urine was normal. Parasitological examination of the feces was negative. The blood was normal; the wassermann reaction of the blood serum was negative. The spinal fluid (withdrawn during and after the attacks) was clear and revealed always about 30 to 50 cells; the globulin (Pandy) test was positive or negative; the Wassermann reaction was negative, and the result of the colloidal mastic (Takada-Ara) test was negative, and the pressure of the fluid was increased. Pilocarpin, atropin-, and adrenalin test showed negative results during and between the attacks. Water tests showed also negative results.

Pneumoencephalogram showed the dilation of the third ventricle. Due to the lack of facilities no EEG results were obtained.

Rorschach test and Kraepelin and Bourdon's tests showed marked differences between the results of the attacks periods and those of the between periods. During the attacks periods, less co-operating attitude and more organic signs were observed. No particular signs of epilepsy or other main psychoses were demonstrated in the Rorschach test.

Lumbal punctures were made twice after the attack in February and once during the attack in March, and the patient showed seemingly the deminution of the severe syndromes during the latter attack. The patient discharged the hospital soon after the last attack was over, and came to the clinic regularly as an out-patient, while she received endolumbal injections of streptomycin every other day for two weeks. But since after she had once severe complications (headache, vommiting, dizziness etc.), she did not come to the hospital anymore. But later the patient visited our clinic again to report that she had no more somnolence attack. About one year later from her discharge, she was found still healthy and never shown any sign of the somnolence attack.

I have seen **another case** of periodic somnolence at the same out-patient clinic in November, 1950. He was a 21-year-old youth who was referred for psychiatric evaluation to determine whether he was responsible for military duty or not. He was at that time quite normal, however, he told that he had, since 8 years prior to that time, had mental syndromes which had used to occur 3 or 4 times in a year. On the first occurrence of the syndrome he had been admitted to the same hospital (during the Japanese occupation of Korea). He was observed during about 4 weeks and finally diagnosed as hysteria or beginning schizophrenia. But through the interview with the patient and his father, it was found that he had periodic somnolence which occurred less, gradually while he was getting older. I have imagined at that time that there might had been quite a number of patients of periodic somn olence who had not been diagnosed as such because of dominant clinical features of personality disturbances in these cases. In the "periodic somnolence", not only somnolence itself, but alsthese personality and emotional changes, which seem apparently to be of diencephalic nature, will be important to determine the nature of the disease.

B. Short lasting Sleep States

1) Narcolepsy (Gelineau[71]): The essential feature of this disease is repeated and apparently irresistible attacks of sleeping which last very short, several minutes or hours, and occur 3 to 6 times, or 100 (Jelliffe[76]) to 200 times (Gelineau[71]) daily. Wilson[77]

has called attention to the fact that some of these patients may be in trance like states resembling sleep, in which consciousness is retained. In others, the sleep is deep and indistinguishable from normal sleep. According to recent EEG studies, it is suggested that in some group the disorder is limited to the normal sleep mechanism (Dynes and Finely[78], Murphy[79], or or others), where as in the other group it is wide spread (Dynes and Finley[78]) and abnormal wave-forms similiar to those commonly observed in epilepsy are present. In some patients suffering from narcolepsy, there is an associated condition called catalepsy which is usually brought on by strong emotion, anger, hearty laughter, or pleasurable excitement etc. Or there is sometimes sleep paralysis occurring either while the subject is falling asleep (predormital or hypnagogic paralysis), or immediately upon his waking from sleep (postdormital or hypnopompic).

This syndromwas first brought by Caffe before the medical public of Paris and it was named as nacrolepsy first by Gelineau[71] in a published description. After Gelineau, it was studied and reported by many authors; especially the World War I., according to the legal problem, was the great moment which made many docters interested. Redlich[68], Stiefler[64], Singer[80], and many others have contri buted in the study of this disease. Thus already by 1961 the reported cases numbered 481 (Nothin and Jelliffe[81]) and more and more cases were added subsequently.

The list of pathological conditions giving rise to prolonged somnolence may be taken to apply equally to narcolepsy. That this disorder may be of diencephalic nature has been earlier postulated by Ratner [74] and been also assumed later by Daniel[55], as already described above. Redlich[68] had the assumption that the narcolepsy is one of the functional disturbances of the sleep centre which had been considered by Economo[51] and Mauthner[82] as to exist at the boundary of di- and mesencephalon, in the wall of the third ventricle. Foerster[21] observed narcoleptic and other sleep disturbances in his case which hypothalamus was occasionally interfered with on operation. Von Benedek and Jubu[83] have, by autopsy studies of a narcoleptic case, proved that in the narcolepticcase there were anatomic changes at the post erior part of the wall of the third ventricle, the beginning portion of aquaeductus Sylvii, which has been thought as the waking centre by Mauthner[82], Economo[51], Kleist[17] etc.

Mental symptoms in narcolepsy have not been accorded the attention they deserve, partly because they have been cast into the shade by other and more spectacular manifestations of this disorder. As Levin[66] pointed out, they are not merely incidental phenomena of only trivial significance, but constitute a true element of the narcoleptic picture, being a direct result of the neurophysiological disturbance that underlies this disorder. Levin[66] thought that certain mental symptoms of narcolepsy, such as thinking difficulties and lapse of memory, are direct results of excessive inhibition in the brain. Kleitmann [84] reviewed earlier that just as these higher centers are susceptible to the action of poison, they are more susceptible to fatigue. I would rather like to assume, as I did in cases of periodic somnolence, that mental syndromes of narcolepsy may most likely prove the diencephalic nature of the disease. Various clinical features of mental and personality disturbances in narcolepsy cases were well reviewed in Daniel's article[55] : sensitiveness, irritability, nervousness, lazyness or indifference, shyness, demoralizing, emotional instability, argumentativeness and indolence, temper, personality changes like as postencephalitic states, avoidance of social intercourse, depression, lack of interest, difficulties of concentration, impairment of memory, a prey to any phantastic idea, feeling as though "mental machinery would not budge", delusional and hallucinatory experiences (similiar to peduncular haluucinosis described by Lhermitte) etc.

Doyle and Daniel[85] reported that ephedrine, which acts on subcortical centres, is effective in preventing the sleep attacks : this could be one reason for believing that diencephalic sleep center is affected in narcolepsy. In a case which I have seen, there was a remarkable deminution of sleep attacks while the patient was receiving EST treatments, which were made on the probable assumption of electrical stimuli to the awaking centre.

app. "Pyknolepsy"

In this disease, the patient suffers from frequent seizures which resemble the epileptic petit mal with the following characteristics: the attacks appear in otherwise healthy children as frequent, short, incomplete cloudings of consciousness; running a fairly monotonous course, with little response to therapy; no deterioration; and generally a favorable prognosis. Friedmann thought this disease as related narcolepsy (Gelineau[71]) and described often as non epileptic absences or brief narcoleptic attacks, or narcoleptic absences etc. Nearly every one, however, now agrees that two clinical features are probably unrelated, and the disorder described by Friedmann[86] is now generally referred to as pyknolepsy, a term proposed by Stoecker[58]. Ratner[74], however, assumed that this disorder is of diencephalic nature as well as narcolepsy. And recent EEG studies (Owen and Berlinrood [87]) proved that pyknolepsy is a form of petit mal epilepsy, which is apparently a diencephalic disorder as described above; though some characteristics, especially the lack of deterioration after years of attacks sometimes numbering as high as a day, and the favourable prognosis, have made such casses stand out from epilepsy and other disease and have led many to the belief that they constitute a separate disease.

2) Epileptic sleep states

i. Sleep states following epileptic seizures.

ii. Sleep attacks as equivalents of epilepsy: Redlich [68] observed a case of this attack which used to occur after unusual phenomenon, nausea or vomiting, and lasted usually 2 to 3 hours.

iii. Mixed occurrence of narcoleptic and epileptic attacks: Redlich[68] observed also a case of narcolepsy which developed later epileptic attacks and another case of epilepsy which developed narcoleptic attacks. Wilson [77] stressed the close relationship between narcolepsy and epilepsy. Cohn and Cruvant[88] stress both the familial incidence of the disorder and its familial relationship to epilepsy in their series. The EEG observations of Dynes and Finley[78] seem to indicate that narcolepsy is only a symptom, the underlying cause of which may be related to epilepsy in some cases but not in others.

C. Inversion of the Sleep Rhythm

The individual sleeps during the day and is awake during the night; the cause of this reversal is unknown. This phenomena is sometimes seen in cases of epidemic encephalitis (Brock[8]). And this was observed in a Foerstser's case[21], the hypothalamus of thish was occasionally interfered with on operation.

D. Insomnia

This may occur as the result of following conditions (Muncie[89]): .

1) pain, fever, or other somatic discomforts (cardiac distress, gastroentestinal conditions, itching etc.);

2) metabolic disturbances as hyperthyroidism;

3) mental factors as brooding, rumination, anxiety, melancholia, hysteric or schizophrenic breakdown, or delirium;

4) plain overconcern for the sleep itself.

There are also following additional conditions to result insomnia:

5) narcolepsy;

6) periodic somnolence;

7) constitution related to cyclothymic disorders.

Kaplinsky and Schulmann[69] reported, in their second article on periodic somnolence,2 cases of **"periodic insomnia"** which occurs periodically and lasts few weeks without any complication. They cited Gaupp's assumption[90] on the relation between cyclothymic group and periodic insomnia, and Merklin's observation[91] on direct heredity of periodic somnolence. And they have, citing also Ratner's hypothesis[74], concluded that this syndrom might be a polar state of periodic somnolence and there exist cycloid disorder and epilepsy between 2 poles, periodic insomnia and somnolence.

Comments and Considerations

Because of these observations reviewed descriptively as above, one may no longer deny to recognize the fact that the diencephalon in conditions of disease is capable of responding with a series of symptoms in the realm of the emotional, intellectual, personality, and mental spheres, as well as in the realm of the vegetative and somatic functional spheres.

Nevertheless the question remains why many pathological changes in those regions fail to produce such syndromes (Spatz and Wittermann[92], Massermann[93] etc.). In order to answer to such question,

however, Foerster[16,21] has already pointed out that the different part of the brain can easily be mistaken as the diencephalon by the reporters. He also postulated that the appearance of the diencephalic syndrome is very dependent upon the tempo of the process of the disease. Bonhoeffer[4] and Schuster[5] recognized again that not only the tempo but also the specific kind of the disease will play a role in the genesis of the clinical features. On the other hand, Ratner[74] postulated the important role of constitutional insufficiency of the diencephalon in producing various syndromes or disorders, as described above. K. Stern and Dancy[25] also argued that it appears from their case that in addition to the lesion and its particular localization, there must be an endogeneous predisposition. Analogous observations have been made in the case of traumatic epilepsy (Lennox, Gibbs and Gibbs[94]).

Another view will be then aroused here. Some of the of the diencephalic disorders cannot be apparently recognized as such, because of the insufficient evidence of symptoms which are dependent upon the tempo and specific kind of the disease. And it will eventually be considered as the different disease from the diencephalic disorder, sometimes merely as the constitutional disease, when an endogeneous predisposition is predominent in the clinical feature of the case. Now it seems to be probably well understood why the "periodic somnolence" or so-called "idiopathic narcolepsy" etc. have not been apparently recognized as diencephalic discorders, but have been often considered as to belong to a certain independent disease group. Many clinical syndromes or disorders, epilepsy, pyknolepsy, narcolepsy, periodic somnolence, episodic terror, dipsomania, migraine psychosis, poriomania, and episodic twilight states, which have been by Kleist[17] and his co-workers considered to be independent from each other but constitute a larger type of constitutional disease group, or epileptoid disorders, may better be considered as diencephalic disorders, or as **"diencephaloses" (Ratner[74])** in certain meaning.

Kleist[17] thought that the above described diseases' group, the group of episodic twilight states and periodic somnolence etc., is the neighbour group of his "autochthonous degeneration pyschoses", but they are to be separately considered as independent from each other. Any how, those two groups could be considered as related very closely by his formulation. Then, what is his conception of **"autochthonous degeneration psychoses"?**

Kleist[17] described following characteristics of this group of psychoses:

1) Each individual psychosis under this group will have following common conditions:

i) the constitutional basis,

ii) the autochthonous appearance,

iii) episodic or periodical repeating of the same disease attack,

iv) good prognosis;

2) But each psychosis may have various independent nature in

i) symptomatology, and

ii) pathogenesis.

And he counted following syndromes or disorder "as autochthonous degeneration psychoses":

a) periodic melancholia and mania, as well as mixed states of both,

b) certain kinds of paranoid disorder (Ewald,[95] Thomson[96], Kleist[17]),

c) Wernicke's expansive autopsychosis[97] with autochthonous ideas (Boström[98]),

d) motility psychosis (Wernicke[87])without early deteriorations,

e) Hypochondriac psychosis and obsessive-compulsive psychoses which autochthonously and sometimes periodically occur, and

f) atypical excitements, depression, or anxiety etc..

Finally he assumed following conditions as pathological factors of this group of psychoses.

1) Heredity.

2) Various somatic processes:

i) Extracerebral processes: hormonal and metabolic processes, infectious diseases, injury, or strong emotions.

ii) Intracerebral processes: constitutional weakness of the brain.

And he further thought that the above extracerebral processes will never act alone but it will act together with the constitutional weakness of the brain to cause the psychoses. He assumed the weakness of the brain especially at the lower or deeper

part of the brain, the part to be considered as the centre of sleep and awake functions and consciousness; such a part is, of course, regarded to be apparently in the diencephalon nowadays.

In some occasions when psychopathological syndromes are predominent in clinical pictures, the case of diencephalic disorders may also be disregarded as such because of the same reason why diencephalic disorders were often considered merely as constitutional disorders; and it may only be viewed and treated psychologically. Often in our daily clinical practice, there are such periodic cases as that of a person who seemed first to belong to a certain category of the definite classification of psychiatric disorders from the psychological and psychodynamic view points, but changes its predictable course suddenly and atypically with often benign prognosis and repeat it again or several times without any known environmental and psychogenic factor. Or there are sometimes such problematic psychiatric conditions as that of a patient who was being "successfully" treated for anxiety, depression and other nervous symptoms and then died from tumor suddenly (Stern, K. and Dancy[25]) or shortly after the end of the treatment. Those cases will bring the necessity and encouragement to attempt to find some correlation of structure and function, instead of trying only to credit to the emphasis on the psychogenesis of emotional and personality disorders. As the result of the flowering of psychodynamic concepts nowadays, the possibility of finding structural bases for personality disorders seems to be sometimes disregarded. Howevar, many organic conditions, as Dr. Adolf Meyer[99] argued, influence a person's mind precisely because he is a personality; i.e., a patient is not only a body or a mind, but a personality composed of both (Vander Veldt and Odenwald[100]).

Summary

1. Clinical literatures, relating to the diencephalon and disorders arising from its changes, were briefly reviewed with a review and case presentation of "periodic somnolence".

2. Observations of both reported cases and my cases of "periodic somnolence" made the author to assume these cases probably as of diencephalic nature not only because of the existence of somnolence itself but also bacause of many other diencephalic symptoms in the realm of emotional and personality spheres, as well as in the realm of visceral and somatic functional spheres.

3. After the review, the remained question, why many pathological changes in the diencephalic region fail to produce the syndrome, was discussed; and constitutional concepts as well as psychodynamic concepts on certain disorders' group relating to the diencephalon were commented.

4. However, no claim was yet clearly made for the thesis, that in view of the diencephalic evidence, the cortical concept of these disorders must be abandoned. On the contrary the observations may tend rather to indicate more clearly the importance of the cortex and its relations with so called lower centres (Alpers[3]).

국 문 초 록

間 腦 部 疾 患

—周期性睡眠病의 症例報告와 아울러—

유 석 진

1) 周期性睡眠病의 症例報告와 아울러 이 疾患을 包含한 間腦部疾患群 全般에 對한 文獻을 綜合的으로 考察하였다.

2) 周期性睡眠病은 著者例로나 文獻例로나 그 觀察 結果가 많은 點에서 아마도 이것이 間腦部에서 起因하여 發生하는 疾患이 아닌가 推證하였다. 그것은 固有한 睡眠症候뿐만 아니라 臟器및 身體機能의 領域과 아울러 情動및 人性領域에 있어서의 여러가지로 많은 間腦部症候群을 이 疾患이 가지고 있음이 引證되었기 때문이다.

3) 間腦部에 各種 病理學的變化가 發生하였을때 반드시 一定한 症候群이 나타나는 것이라고는 할수 없는데 이런 問題의 未解決點에 關하여는 따로 考察을 加하였다. 體質論에 立脚한 概念 또는 精神力動學的概念을 特히 間腦部疾患群에 關하여 어떻게 볼것인가에 對하여도 加評을 하였다.

4) 上記 諸考察에서 間腦部症候群의 證左로만 各種 疾患의 皮質과의 關係를 無視한다는 結論을 내린것은 勿論 아니다. 그보다는 오히려 大腦皮質및 그것과 下部中樞와의 連絡關係等의 重要性이 强調되어야 함을 諸觀察 結果는 어느點 뒷바침하는 것도 있을 것이다다.

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