5 Popoviciu, L.; Aşgîan, B.; Pascu, I., and Szabo, L.: Somnul normal şi patologic, Editura Medicala, Bucureşti (1972).

6 Schirardin, H. et Metais, P.: Determination de l'acide 5-hydroxy-indol-acétique, metabolite de la serotonine. Ann. Biol. clin. 18: 677-681 (1966).

7 Schwartz, B. A.; Segny, M. et Escaude, J. P.: Correlations EEG, respiratoires, oculaires et myographiques dans le ' syndrome Pickwickien ' et autres affections paraissant apparentées. Proposition d'une hypothèse. Rev. neurol. 117: 145-162 (1967).

8 Woïwod, A. J. and Knight, R.: Determination of the urinary catabolites of catecholamines. J. clin. Path. 14: 502 (1961).

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Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications. 1st Europ. Congr. Sleep Res., Basel 1972, pp. 550-554 (Karger, Basel 1973)

Clinical and Polygraphic Study of Cataplectic Attacks

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Materials and Methods

By means of clinical, electroencephalographic, and polygraphic evaluation we have examined 8 narcoleptic patients (7 males and 1 female), who, in addition to the attacks of sleep, also showed cataplectic episodes. We have investigated these crises, their duration and frequency, as well as the conditions of their appearance. EEG recordings were made during spontaneous activity, as well as after hyperpnea and following activation with Ahypnon, and Baytinal photocardiazol stimulation. Polygraphic recordings of the pneumogram, of the eye movements (EM), of the EMG and EEG were done around the clock. In three patients, acoustic and photic stimuli were applied during the various phases of sleep.

Results

The frequency of the attacks varied between 1 and 10 per year. In none of the cases did the duration of crises exceed 12-30 sec; none of the patients lost consciousness during these attacks. The crises involving the loss of postural tonus were usually set off by pleasant or unpleasant emotions; occasionally they occurred spontaneously. All our patients have shown total cataplectic episodes.

The standard EEG examinations, as well as those under activation with Ahypon or photocardiazol stimulation were found to be normal.

The poly graphic examinations revealed that in the majority of instances the patients fell asleep by directly moving to rapid eye movement (REM) sleep, both in daytime and during the night. Furthermore, they showed an increase in the percentage of the REM phase of sleep (34.4-48.2%), with an increase in REM percentage during the day of up to 58.3% of total sleep.

The qualitative analysis demonstrated the following: (1) in narcoleptics, during waking, the muscular tone tended to be decreased. During the occurrence of attacks of atonia, the EEG pattern was manifestly similar

Fig. 1. a Daytime recording in patient T.I., aged 41. Diagnosis: narcolepsy with cataplectic crises. Repeated tendencies towards decrease of the muscle tone (with atony). EEG pattern of wakefulness, b Daytime recording in patient C.I., aged 34. Diagnosis: narcolepsy with cataplectic crises. During an attack of muscular atonia, persistence of the eye movements, which are nevertheless stopped upon application of the acoustic stimuli, to which the patient fails to respond with motor activity. EEG pattern of wakefulness. c Nocturnal recording in same patient as in figure 1 b. Sleep paralysis. EEG pattern of wakefulness. EM (REM). Muscular atonia. The patient had perceived the acoustic stimuli, but failed to respond, d REM in same patient as in figures 1 b and c. The patient perceived all the acoustic stimuli, responded only during the REM phases of sleep with reappearance of the muscle tone, but failed to respond during the periods of REM with muscular atonia. P = pneumogram.

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to that observed during wakefulness; (2) eye movements, like those noted during the paradoxical phase of sleep, may or may not appear during the attacks of muscular atonia, although the EEG pattern remained constantly that of wakefulness; (3) during these conditions, there were occasional short periods (10-15 sec) during which the EEG pattern reverted to that observed during the paradoxical phase; (4) the transition from normal muscular tone to atonia and vice versa occurred gradually, usually within 3-7 sec, and (5) during the period of atonia, with eye movements similar to those noted in paradoxical sleep, the patients sometimes showed hypnagogic hallucinations.

These phenomena not only appeared while falling asleep or upon awakening, but also during the recording of a complete 24-hour period,

demonstrating the excessive tendency towards the dissociation of the various parameters of sleep (EEG, muscular tone, eye movements, hypnagogic hallucinations).

During the experiments with acoustic and photic stimuli, the following observations were made: (1) during the atonic crisis in which the patient showed eye movements similar to those noted in the paradoxical phase of sleep, but with an EEG pattern of wakefulness, there was no motor response to the acoustic stimuli, although they were perceived. The patients declared that they were unable to execute any movements; (2) during the phase of paradoxical sleep, the patients were able at times to respond to the acoustic stimuli, as revealed by the appearance of phasic or tonic activity in the EMG, and (3) during the muscular atonia with eye movements or during paradoxical sleep, the photic stimuli stopped the eye movements for a period of 10-15 sec; thereafter ocular motor activity reappeared. During these periods both the EEG activity and the muscular tone remained unchanged (fig. 1 a-d).

Discussion

According to Roth [8] cataplectic attacks (so-called spontaneous attacks) may occur without the intervention of any eliciting factors. This we have observed also in our material. Passouant et al. [5] reported a case with an attack of cataplexy (telemetric recording) with loss of the muscular tonus and an EEG activity of wakefulness which lasted 48 sec. All our patients have shown total cataplectic attacks, but their consciousness remained unaltered throughout the duration of the episode.

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Janzen and Behnsen, quoted by Roth [8], have noted a cataplectic attack while the EEG tracing showed a pattern of sleep.

Our investigations confirmed the observations by Okuma et al. [4], Nan'no et al. [3], Rechtschaffen et al. [7] and Shimizu et al. [9]. In addition, we found that in such patients one may observe the development of atonic attacks, during which the patients are incapable of any motor activity even during wakefulness. At times, the attacks are accompanied by eye movements similar to those noted in paradoxical sleep. We propose to label such episodes as true cataplectic attacks.

Our observations indicate the existence in narcoleptic patients of descending inhibitory influences impinging on somato-motor pathways, which are very resistant to the effect of the stimuli. Furthermore, they suggest a dissociation of the various components of sleep (behavior, EEG

activity, motor activity).

From our observations we conclude that there are, in all probability, no qualitative or quantitative differences between the cataplectic attack and the sleep paralysis. Both phenomena probably depend on the same physiopathological mechanisms and constitute at least similar, if not entirely identical, entities.

References

- 1 Devic, M.; Aimard, P.; Michel, F. et Masquoin, M. O.: Etude clinique des narcolepsies-cataplexies essentielles. *Rev. neurol.* 116: 471-490 (1967).
- 2 Kubota, K.; Iwamura, Y., and Nishimi, Y.: Monosynaptic reflex and natural sleep. *J. Neurophysiol.* 28: 125-138 (1965).
- 3 Nishino, H.; Hishikawa, Y.; Koida, H.; Takahashi, H., and Kaneko, Z.: A neurophysiological study of sleep paralysis in narcoleptic patients. *Electroenceph. clin. Neurophysiol.* 28: 382-396 (1970).
- 4 Okuma, T.; Nakamura, K.; Hayashi, A., and Fujikori, A.: Psychophysiological study on the depth of sleep in normal human subjects. *Electroenceph. clin. Neurophysiol.* 21: 140-147 (1966).
- 5 Passouant, P.; Popoviciu, L.; Velok, G. et Baldy-Moulinier, M.: Etude polygraphique des narcolepsies au cours du nycthemere. 118: 431-441 (1968).
- 6 Pompeiano, O.: The neurophysiological mechanisms of the postural and motor events during desynchronized sleep. *Res. Publ. Ass. nerv. ment. Dis* 45: 351-423 (1967).
- 7 Rechtschaffen, A.; Wolpert, E.; Dement, W.; Mitchell, S., and Fischer, C.: Nocturnal sleep of narcoleptics. *Electroenceph. clin. Neurophysiol.* 15: 599-609 (1963).
- 8 Roth, B.: *Narkolepsie und hypersomnie*, pp. 156-163 (Veb Verlag Volk und Gesundheit, Berlin 1962).

Free Communications: I. Clinical Investigations 554

- 9 Shimizu, A.; Yamada, Y.; Yamamoto, J.; Fujiki, A., and Kaneko, Z.: Pathways of descending influence on H reflex during sleep. *Electroenceph. clin. Neurophysiol.* 26: 337-347 (1966).
- 10 Vizioli, R.: Les bases neurophysiologiques de la cataplexie. *Electroenceph. clin. Neurophysiol.* 16: 191-193 (1964).

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Sleep: Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications.

1st Europ. Congr. Sleep Res., Basel 1972, pp. 554-559 (Karger, Basel 1973)

Hypersomnia with Rapid Eye Movements

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Hypersomnia—that is, excessively long or deep sleep—must be distinguished from narcolepsy, which consists of inappropriate attacks of sleep or dissociative sleep states with no necessary increase in time asleep per 24 h. All hypersomnias studied to date have involved non-rapid eye movement (NREM) sleep mechanisms or consisted of extended sleep in toto [cf. reviews of Broughton, 1971, 1972]. A recent case demonstrates the existence of hypersomnia involving rapid eye movement (REM) sleep mechanisms.

An 18-year-old male was hospitalized in May, 1972, with a 4-month history of diurnal fatigue and drowsiness, and 5 weeks of increased sleep (up to 16-18 h/day), difficulty in awakening (but no true 'sleep drunkenness' once awake), diurnal sleepiness and sleep, and severe ocular discomfort. The symptoms greatly interfered with schooling and homework. There were no irresistible sleep attacks, daytime sleep coming on slowly over minutes or dozens of minutes and allowing the subject to lie down. Daytime sleep episodes lasted 20 min to several hours. Once he tried to avoid sleep while on his motorcycle, and eventually drove into a snowbank. During diurnal drowsiness, he noted involuntary flicking of his

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