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Hypersomnia

A Discussion of Psychiatric Implications Based on Three Cases

By ALEXANDER BONKALO

Epimenides, a Cretan poet of the 6th century B.C., withdrew into a cave where he fell asleep; he awoke 57 years later (7). This anecdote is probably the first recorded case of hypersomnia. Sleeping Beauty, Rip Van Winkle and other personalities of folklore and literature followed. Clinically valid contributions, however, first appeared around the turn of this century.

Hypersomnia has been defined by Kleitman (15) as "uncontrollable somnolence and pathologically prolonged sleep, from which it is sometimes difficult to arouse the sleeper, or to keep him awake for any length of time after he has been awakened". The reduction of consciousness in hypersomnia is what appears to be *normal* sleep. It should therefore be differentiated from conditions of *impaired* consciousness, such as coma, stupor, ictal or postictal confusion, and hypnotic or hysterical dissociation.

Narcolepsy and hypersomnia are two separate and distinct categories among the sleep disorders. Narcoleptic attacks are sudden and imperative; the patient falls asleep on the spot, and his sleep is of short duration, with immediate alertness when awakened. In hypersomnia, sleep is irresistible as in narcolepsy, but its onset is more gradual. The patient has time to withdraw, even to go to bed in his accustomed manner. His sleep is prolonged, lasting hours, days or weeks. He can only with difficulty be aroused; on awakening he is drowsy, usually irritable, slow in mentation, even confused. Essentially, though this is somewhat over-simplified, the salient feature in narcolepsy is the need to *fall* asleep; in hypersomnia, to *keep on* sleeping. Phenomena typically associated with narcolepsy (cataplexy, sleep paralysis, hypnagogic hallucinations) are not present in hypersomnia.

Hypersomnia is a syndrome which may be brought about by different conditions. Among common causes are tumours, destructive lesions or inflammations affecting the brain-stem, as reviewed by Kleitman (15), Michaelis (20) and Roth (22). Excessive abdominal girth may also lead to somnolence, as seen in the "Pickwickian syndrome" identified by Burwell, Robin, Whaley and Bickelmann (1) and elaborated further by Drachman and Gumnit (5). The menarche or the menstrual cycle may be linked with hypersomnia, as shown by Jochims (13) and Lhermitte and Dubois (18) respectively. Sleep states can develop on a psychogenic basis, e.g. after sudden intensive negative emotional experiences, according to Roth (22), or as an avenue for passive retreat from conflict and a means of withdrawing from action, as reported by Goldstein and Giffin (11). Michaelis (19) observed hypersomnia, rather than insomnia, in cases of endogenous depression.

A clinical pattern has emerged from the diversity of forms, characterized by "periodic somnolence and morbid hunger", first described by Kleine (14), then more specifically defined by Levin (16), and eventually named the "Kleine-Levin syndrome" by Critchley and Hoffman (4). In a comprehensive study, Critchley (3) has pointed out that in all "genuine" cases the patients involved have been adolescent males. He also suggests that excessive eating in this condition was compulsive and not due to morbid hunger. His criteria narrow the syndrome to a clinical entity referred to as "periodic hypersomnia and megaphagia in adolescent males". It becomes a matter of opinion whether this restricted category constitutes the Kleine-Levin syndrome, or whether this term should be used in a broader sense to include every case in which periodic somnolence is associated with excessive eating. Recent reports by Gallinek (9), Gilbert (10) and Earle (6) have extended the limits of this syndrome, and have also added a few female patients to the very small, and in

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part controversial, roster of such cases.

A special form of hypersomnia was called the syndrome of sleep drunkenness (*Schlaftrunkenheit*) by Roth (22), and considered to be a separate entity. Essentially it consists of protracted drowsiness in the morning after a complete and deep night's sleep. Linked with this condition is a tendency toward dissociative states due to incomplete wakefulness on premature arousal.

Three cases of hypersomnia admitted to the Toronto Psychiatric Hospital are here presented.

Case 1

This 16-year-old boy had four sleep attacks between October 1959 and April 1960; the last one was observed in hospital. Each sleep episode lasted 5 to 6 days, separated by normal intervals of 49, 87 and 62 days respectively. Several weeks before the first attack he suffered a mild "flu-like" illness.

Description of a Sleep Episode. The typical attack was heralded by reluctance to get up in the morning. During the day the patient was slow, uncommunicative, appeared sleepy or "in a daze". His responses were monosyllabic, often revealed anger or irritation. The following day he remained in bed, lying flat or curled up on his side, in a state which appeared to be normal sleep, no different from his regular night sleep. Occasionally he became restless, banging his heels, turning and tossing in bed. During the early phase he perspired excessively, his hands and feet were cold and clammy; there was also moderate acrocyanosis. He could be awakened for meals, he did go to the washroom for eliminations; he was, however, drowsy, mute or monosyllabic, abrupt and irritable on such occasions. He ate and drank somewhat less than his usual diet the first day or two, but during the later days of his period of somnolence he ate large amounts. He was resistive and angry when awakened for clinical examinations, though fully oriented and rational. The condition of almost continuous sleep lasted for five days. On the sixth, he was more spontaneous and spent some time out of bed, walking up and down and watching TV, though he remained dazed and poorly communicative until towards evening, when he became more alert and active. After one more day of gradual improvement he became fully alert and somewhat elated; he ate excessively at meals, with sweets and candies in between; he was pleasant, friendly, co-operative and well organized. In a day or two his mood, eating habits and general behaviour were back to his usual standards.

Investigations. Personal and family history were uneventful, except for manic-depressive illness in a maternal aunt. His mother, now domineering and over-protective, attempted abortion early in her pregnancy by taking quinine. He was a "model" child. There was no history of enuresis or other sleep disturbance, though he was a day-wetter until age 10. All his life he had been overweight. At 16 his height was 5 ft. 8 in. and he weighed 151 pounds. His body contours were softly moulded, giving him an effeminate appearance which was accentuated by bilateral gynaecomastia. Skeletal proportions and hair distribution were masculine, and his genitalia were of normal size. He had normal heterosexual phantasies and interests. Clinical investigations, including EEG, lumbar CSF, air encephalogram, glucose tolerance tests, electrolyte study and serum protein assessments, gave normal results. Examinations during a sleep attack also resulted in essentially normal findings, except for massive defence reactions bilaterally and upgoing toes at the left side on plantar testing, as well as eosinophilia (12 per cent.) and episodic or continuous high amplitude 5–6 cycles per second slow waves occurring in both frontal and temporal leads in the EEG. From May to September 1960, detailed observations and laboratory studies were carried out in hospital. No sleep attack occurred during this period. Serial EEG examinations resulted in poorly organized but essentially normal patterns. Daily BMR findings were consistently low, ranging between −4 and −25. Remaining data were deemed to be within normal limits; they showed no periodic or episodic variations, beyond regular daily rhythms.

Mental State. Between attacks, this boy's behaviour and personality did not differ essentially from others of his age. He was co-operative and friendly; though there was some underlying hostility, he was able to establish good rapport, with considerable warmth.

Psychological testing presented no evidence of pathology. Intelligence (WAIS) was average (verbal 108, performance 102, full scale 106). Personality profile (Cattell 16 PF) indicated submissiveness, timidity and lack of concern. Projective tests suggested introversion, as well as anxiety about the environment generally and about female authority specifically. Dynamically, the test results were interpreted as suggestive of inadequate defences in relation to an aggressive, hostile mother.

Follow-up examination was carried out in June, 1966. In the interim the patient had had four or five similar episodes, about one a year. All occurred in late spring or early summer. During this six-year period he functioned normally. He married and is well adjusted. He is now grossly overweight; 5 ft. 8 in. tall, he weighs 205 pounds. His gynaecomastia is less apparent; there are striae over his abdomen. Routine physical and laboratory tests gave normal results. He is generally satisfied, has no complaints except for his sleep attacks, which occur about once a year and which he accepts as unavoidable.

Case 2

This unmarried female office worker suffered eight sleep attacks between the ages of 16 and 21 years. Their dates and their duration were as follows: September, 1936, 4 days; May, 1937, 3 days; September, 1937, 8 days; December, 1937, 8 days; March, 1939, 10 days; April, 1939, 8 days; October, 1939, 11 days, and a "mild one" in 1940 for which details are not available. All episodes followed the same general pattern: for no reason that could be ascertained the patient would become listless, indifferent, and careless about her personal appearance; her appetite would diminish, she would complain of nausea

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and occasional vomiting. Within a few days she would become very drowsy, progressing into sleep from which she could be roused with difficulty for meals and toilet. Profuse sweating was noted during the sleep attacks. On arousal she had a muddled, dull look, often appeared irritable or depressed, mildly confused at times, but not grossly disoriented or irrational. Her food intake was classed from "poor" to "good" at different times, apparently improving as the spell progressed. After a variable period of days, she would suddenly "snap out of it" and be perfectly well.

Neurological testing during the sleep attack recorded sluggishness of the right deep reflexes, but no essential abnormalities. Repeated intensive clinical and laboratory examinations between 1937 and 1939, including an air study and early versions of EEG testing (in and out of attacks) resulted in normal findings. It was noted that ergotamine tartrate, ephedrine, amphetamine and phenobarbital, each tested separately, had no effect in relieving attacks. No significant relationship was observed between the menstrual periods and the attacks.

This patient came from an insecure, unhappy home. Her father was a poor provider and a heavy drinker, repeatedly treated in mental hospitals. Two paternal uncles and an aunt committed suicide. The mother was stable, domineering and over-protective. The patient was the youngest of three siblings. The eldest, a sister, was emotionally stable and generally healthy. The second in the sibline, a brother, suffered drowsy episodes, and is presented below in detail (Case 3).

As a child she suffered acute nephritis and otitis media following measles, with residual deafness. Her development was otherwise uneventful. She was described as a likeable, attractive person, prone to mood swings and periods of anxiety; she was ambitious, with strong loyalties; basically, a sensible and able person, who had supported herself since youth working in stores and offices.

In 1950 and 1951 she had two more hospital admissions, at ages 30 and 31, respectively, for reactive depression precipitated by a love affair. This illness had no direct relationship to her sleep attacks, the last of which occurred in 1940. At this time, again, physical and laboratory investigations, including EEG tests, resulted in normal findings, except for bilateral conductive deafness due to previous middle ear infection. Psychological testing suggested superior general intelligence (full scale WAIS IQ 122) and no thought disorder or break with reality. Projective tests suggested very high aspirational level, with a tendency to blame her family, her mother in particular, for her own failures. Insecurity was noted, with a great need for friendship but an inability to accept or trust it. She responded quickly to supportive psychotherapy, and was discharged with the diagnosis "reactive depression". She was in good health when last heard from in 1953.

Case 3

This 32-year-old male factory worker, brother of Case 2, had considerable difficulty waking up in the morning since adolescence. Occasionally he would sleep until 5 p.m., if left alone. At the age of 21 there was an episode when for several weeks during the summer he slept continuously throughout the day in spite of efforts to wake him. When he did respond to arousal he was "bad tempered and mean in disposition". During the fall in the same year, he became drowsy while working in a factory, and went home and slept more or less continuously for three days. At the age of 26, while in military service in England during the war, after a single glass of beer in a "pub", he did not remember anything for a day or two. He was found by a policeman, apparently sleeping, on a park bench. Taken to a hospital, he was discharged a week later without abnormal findings and with no diagnosed illness. Morning drowsiness continued, with episodic worsening. After the war he worked on the assembly line in a refrigerator factory. According to a report from his job, he was an efficient worker, cheerful and co-operative; the only fault to be found with him was that periodically, for several consecutive days, he was late arriving at work because of "sleeping in"; it was also noted that he was "always hungry". Regrettably, the notes failed to clarify the time relationship between his "hunger" and the episodes of "sleeping in".

In 1948, when he was 32, he was admitted to hospital because of depression, precipitated by marital trouble (personal discord). At the hospital it was hard to wake him in the morning, and he was irritable if forcibly aroused. He remained "in a daze", and would frequently go back to sleep, with subsequent amnesia for activities carried out in the dazed state of incomplete wakefulness. His appetite varied; occasionally he refused meals, at other times notes indicate that he ate large amounts of food. Routine investigations reported normal physical findings. The EEG test noted "some sensitivity to hyperventilation" and the presence of subharmonic alpha activity in small amounts, but no definite abnormalities. Psychological examination suggested superior intelligence (full scale IQ 133) and no gross abnormalities. His developmental history was essentially uneventful; he suffered no significant illnesses. He was discharged four weeks later, improved, diagnosed "reactive depression".

Discussion

The salient feature common to these cases was the occurrence of irresistible drowsiness and sleep, linked with pathological emotions and vegetative irregularities. The leading abnormal manifestation was a condition which appeared to be true *sleep:* each patient subjectively *felt* sleepy; their appearance in the attack was very similar to that during their *regular* night sleep; they *could* be aroused from their condition; on arousal, their drowsiness, perplexity and general behaviour (e.g. yawning, stretching, etc.) were in keeping with phenomena common to *natural* awakening. Apart from hypersomnia, however, the clinical picture as a whole differed considerably in each case.

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Recurrent sleep episodes, linked with the ingestion of large amounts of food and some personality characteristics, class the first case in the nosological syndrome referred to by Critchley (3) as "periodic hypersomnia and megaphagia in adolescent males". In 1962 the same author recorded, from literature and from his own observations, a total of 26 "genuine" cases belonging to this category. From reports since 1962, the first case of Regli and Haynal (21) would also belong, and the further addition of our patient as the 28th "genuine" case appears justified. Evidently he also qualifies for the more broadly defined category of the Kleine-Levin syndrome.

It is of some interest that this patient suffered a "flu-like" illness a few weeks before the first sleep attack. Among events which seemingly act as precipitating factors, febrile illness was noted in 5 cases out of the 26 collected by Critchley.

Our patient's eosinophilia, the intensive sweating, the dysrhythmic EEG during the sleep attack, as well as his chronic obesity, gynaecomastia and abdominal striation should be noted here. These findings may implicate diencephalic dysfunction as part of the process.

The second patient showed periodic hypersomnia from adolescence to 21 years of age. The general pattern of the sleep attacks was very similar to that seen in Case 1, except that gastric phenomena other than excessive food intake were prominent, nausea and vomiting occurred regularly during the early phase of each attack, and only towards the end of the sleep attacks did she eat "good meals". Critchley (3) observed in one of his patients that the excessive eating was not due to excessive hunger. The patient did not demand food he did not see, but ate "compulsively" every foodstuff in sight and within reach. Critchley therefore suggested the use of non-specific terms like megaphagia or polyphagia instead of bulimia. He also suggested that the nature of this symptom is in need of thorough investigation, including studies on the experience of satiety, on gastrointestinal peristalsis, and on metabolic functions. Our second patient further highlights this need, as her gastric phenomena may represent a variant of the same symptom; her nausea and vomiting, regularly linked with the sleep attack, may be regarded as an equivalent of the condition which leads to excessive eating in "typical" cases. With this qualification, this patient could be included in the category of the Kleine-Levin syndrome as an "atypical" case.

The sleep disturbance followed a different pattern in the third patient. His history does include two occurrences at the age of 21 years, which appear compatible with two separate attacks of "periodic hypersomnia". More importantly, this patient suffered in varying degrees for many years from an exaggeration of the normal sleep rhythm, with excessive sleep added through grossly oversleeping in the morning. If forcibly aroused after a full night's sleep, he was irritable and carried out "in a daze" such activities as getting dressed and having breakfast; often he went back to bed and continued to sleep. On spontaneous awakening later, he had amnesia regarding the earlier activities.

The pattern of disturbed sleep in this case conforms with Roth's (22) "syndrome of sleep drunkenness". This pathological form of morning drowsiness is characterized by considerable resistance to arousal after a full night's sleep, leading to a gross prolongation of regular sleeping hours; meanwhile the 24-hour sleep-wakefulness rhythm continues, as the patient maintains his alertness for the rest of the day, following his spontaneous awakening at a late hour. Roth also includes with this syndrome the occurrence of "twilight states" due to incomplete awakening on enforced arousal.

The nosological position of pathological morning drowsiness among the varieties of hypersomnia is not yet clear. It may merely be a *forme fruste* of the Kleine-Levin syndrome; there are indications of this in our patient's personal and family history. It can also appear as an unusual sleep disorder in depression, as reported by Michaelis (19). Moreover, there may be cases where the exact sequence and causal relationship of "sleeping in", "maladjustment" and "reactive depression" are blurred or inseparably fused; the diagnostic label in such a case may merely represent observer-bias. Whether it is a symptomatic or a genuine entity, this condition is distinct enough to warrant clinical acceptance in its own right.

The term "sleep drunkenness", however, is

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ambiguous as well as awkward. It was used to denote morning drowsiness as a pathological entity (22). It was also used with reference to impulsive acts, self-destructive or criminal in nature, committed in the twilight state of semi-wakefulness brought about by premature arousal (12, 17, 23). For the sake of clarity, it is proposed herewith to apply the term *diurnal hypersomnia* to cases showing pathologically prolonged sleep with the 24-hour rhythm maintained, i.e. to cases with pathological morning drowsiness. Irresistible sleep to late hours in the morning after a full night's sleep does become a clinical problem if it seriously interferes with the requirements of everyday life, if it is symptomatic of some other disease, or if abnormal waking mechanisms are manifest.

The dissociative states due to incomplete awakening and leading to impulsive or irrational behaviour have been labelled by Carrot, Velluz and Rigal (2) "the syndrome of Elpenor" in reference to an incident in Homer's *Odyssey*.* This term is applicable to abnormal behaviour due to confusion on arousal from regular or from hypersomnic sleep; it is also applicable whether the sleeper is sober or under the influence of alcohol, as were the legendary Elpenor and some of the cases described in the literature.

Of special interest is the familial occurrence of hypersomnia in Cases 2 and 3. In addition to the sleep disorder in two of three siblings, this family was loaded with psychopathology; chronic alcoholism and psychopathy were diagnosed in the patients' father, three suicidal deaths occurred in the paternal sibling, and our hypersomnic patients themselves repeatedly required treatment for depression. Pathologically loaded family settings are not unusual with hypersomnia, as well as with narcolepsy, according to Roth (22). Among cases observed by him or collected by him from literature, the following conditions concurred with relative frequency in different members of a family: narcolepsy, hypersomnia, various forms of psychoneurosis and psychosis, suicide, alcoholism, endocrine disorders, obesity, congenital blindness and enuresis.

Psychogenic origin and the role of personality factors in hypersomnia are much debated problems. Our patients' personalities, though different on the surface, did show certain similarities at a deeper level. All three patients had strongly domineering mothers. All three had high dependency needs, and some difficulties in satisfying them because of ambivalence in their relationships. All harboured deep-seated hostilities, with also some demonstrable paranoid aggression. Denial and passive withdrawal were successful protective mechanisms in the first patient. In the second and third cases, latent hostility and aggression were probably significant factors in the development of their depressive episodes and suicidal preoccupations.

A clear-cut separation of psychological and organic elements in the causation of hypersomnia is hardly possible; if attempted, it may merely reflect the investigator's personal proclivities. Psychological motives, if they are not primarily responsible, may be present as reinforcing factors in every type of hypersomnia, though they enter most readily the diurnal form, as this appears to be mainly an accentuation of regular sleep.

A transitory elimination of consciousness, as suggested by Fenichel (8) is probably the archaic form of all repressions, and may serve as protection against the influx of too much anxiety-producing stimulation, or it may be a total protective response to a specific stress situation. Loss of consciousness, according to Fenichel, whether it is fainting or sleep, may have an unconscious meaning of its own. Such meanings could include any libidinal needs, they may represent death wishes against some other person turned against oneself, or they may serve as distorted expression of hostile impulses of many kinds.

The possibility of causative somatic elements in hypersomnia is a controversial issue, except for the grossly organic varieties with demonstrable pathology, located usually in the brain stem. Certain findings in episodic, periodic or diurnal

* Elpenor, a companion of Odysseus, while asleep on the roof of a house, was awakened by noise. He "suddenly sprang up, and forgot to go to the long ladder . . . but fell headlong from the roof and his neck was broken . . . and his spirit went down to the house of Hades".

Homer: *The Odyssey*, trans. by A. T. Murray. Loeb Classical Library (1960), 1, 385. London: William Heinemann Ltd.

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forms strongly suggest the presence of significant organic factors among the causes. Such findings include the frequent concurrence of sleep disorder and vegetative, hormonal or constitutional abnormalities in the same patient, or the frequent concentration of such symptoms among different members of his family. The striking similarity of the clinical pattern in patients showing the syndrome defined by Critchley (3) or the Kleine-Levin syndrome would also indicate significant organic pathogenic or pathoplastic factors. Moreover, this syndrome usually appears in patients with a reasonably healthy and well adjusted personality.

Beyond its inherent interest, the study of hypersomnia has also heuristic value in psychiatry. Elements which appear in clusters in various forms of hypersomnia are more or less similarly combined in certain other psychiatric syndromes. The concurrence of pathological affective states, sleep disorder and abnormal food intake, as seen in manic-depressive illness and also in periodic hypersomnia, could be a physiologically determined conjunction of functions, rather than merely an incidental analogy. Another converging clinical borderland is the rhythmic alternation of changes in consciousness and in metabolic regulation, as seen in periodic catatonia. Interestingly, stupor attacks in some periodic catatonia cases are heralded by gastric phenomena similar to those which preceded the periodic sleep attacks in our second patient.

Clearly, further observations are needed, not only to understand the clinically valid variants of the hypersomnia syndrome, but to relate the findings to bordering psychiatric conditions as well as to our expanding knowledge of limbic and reticular functions.

Summary

1. Hypersomnia is defined and differentiated from narcolepsy. The literature on various forms of hypersomnia is briefly reviewed.

2. Three patients are presented. The first is an example of the narrowly defined entity referred to by Critchley as "periodic hypersomnia and megaphagia in adolescent males", forming the core-group of the Kleine-Levin syndrome. The second patient is a qualified representative of the latter syndrome, as she manifested, linked with the sleep attacks, gastric phenomena other than megaphagia. The third patient suffered from a syndrome which is essentially a pathological exaggeration of the regular sleep rhythm; the term *diurnal hypersomnia* is proposed as the name for this form of sleep disorder.

3. The role of psychological and organic factors in hypersomnia is discussed. It is suggested that the hypersomnia syndrome, manic depressive illness and periodic catatonia have elements in common, worthy of special attention, even beyond the nosological interest in sleep disorders.

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References

1. Burwell, C. S., Robin, E. D., Whaley, R. D., and Bickelmann, A. G. (1956). "Extreme obesity associated with alveolar hypoventilation—a Pickwickian syndrome." *Amer. J. Med.*, **21**, 811–818.
2. Carrot, J., Velluz and Rigal (1947). "Syndrome d'Elpénor." *Presse méd.*, **55**, 573.
3. Critchley, M. (1962). "Periodic hypersomnia and megaphagia in adolescent males." *Brain*, **85**, 627–656.
4. —— and Hoffman, H. L. (1942). "The syndrome of periodic somnolence and morbid hunger (Kleine-Levin syndrome)." *Brit. med. J.*, *i*, 137–139.
5. Drachman, D. B., and Gumnit, R. J. (1962). "Periodic alteration of consciousness in the 'Pickwickian' syndrome." *Arch. Neurol. (Chic.)*, **6**, 471–477.
6. Earle, B. V. (1965). "Periodic hypersomnia and megaphagia." *Psychiat. Quart.*, **39**, 79–83.
7. "Epimenides" (1966). In *Encyclopaedia Britannica*, **8**, 646. Chicago: Encycl. Brit. Inc.
8. Fenichel, O. (1945). "Hysterical dream states and disturbances of consciousness." In: *The Psychoanalytic Theory of Neurosis*. New York: W. W. Norton and Co. Inc.
9. Gallinek, A. (1962). "The Kleine-Levin syndrome: hypersomnia, bulimia and abnormal mental states." *Wld. Neurol.*, **3**, 235–241.
10. Gilbert, G. J. (1964). "Periodic hypersomnia and bulimia." *Neurology (Minneap.)*, **14**, 844–850.
11. Goldstein, N. P., and Giffin, M. E. (1959). "Psychogenic hypersomnia." *Amer. J. Psychiat.*, **115**, 922–928.
12. Illing, E. (1941). "Schlaftrunkenheitsdelikte bei Soldaten." *Dtsch. Militärarzt*, **6**, 618–622.

https://doi.org/10.1192/bjp.114.506.69 Published online by Cambridge University Press

13. JOCHIMS, J. (1953). "Schlafsucht als Auftakt zur Menarche." *Arch. Kinderheilk*, **147**, 156–158, quoted by Roth (22).
14. KLEINE, W. (1925). "Periodische Schlafsucht." *Mschr. Psychiat. Neurol.*, **57**, 284–320, quoted by Critchley (3).
15. KLEITMAN, N. (1963). *Sleep and Wakefulness*. Chicago and London: Univ. of Chicago Press.
16. LEVIN, M. (1936). "Periodic somnolence and morbid hunger." *Brain*, **59**, 494–503.
17. LANGELÜDDEKE, A. (1955). "Delikte in Schlafzuständen." *Nervenarzt*, **26**, 28–30.
18. LHERMITTE, J., and DUBOIS, E. (1941). "Crises d'hypersomnie prolongée rythmées par les règles chez une jeune fille." *Rev. neurol.*, **73**, 608–609.
19. MICHAELIS, R. (1964). "Depressive Verstimmung und Schlafsucht." *Arch. Psychiat. Nervenkr.*, **206**, 345–355.
20. —— (1965). "Zur Typologie der Hypersomnien." *Fortschr. Neur. Psychiat.*, **33**, 587–599.
21. REGLI, F., and HAYNAL, A. (1965). "Zwei Fälle von 'periodischer Somnolenz junger Leute' (Kleine-Levin Syndrom)." *Arch. Psychiat. Nervenkr.*, **206**, 576–583.
22. ROTH, B. (1962). *Narkolepsie und Hypersomnie*. Berlin: VEB Verl. Volk und Gesundheit.
23. SCHMIDT, G. (1943). "Die Verbrechen in der Schlaftrunkenheit." *Z. Neur. Psychiat.*, **176**, 208–253.

NOTE: See also DUFFY, J. P., and DAVISON, K. (1968). "A female case of the Kleine-Levin syndrome." *Brit. J. Psychiat.*, **114**, 77–84.

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