

English Translations Of The First Clinical Reports On Narcolepsy And Cataplexy By Westphal And Gélinau In The Late 19th Century, With Commentary

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Study Objectives: To publish the first English translations, with commentary, of the original reports describing narcolepsy and cataplexy by Westphal in German (1877) and by Gélinau in French (1880).

Methods: A professional translation service translated the 2 reports from either German or French to English, with each translation then being slightly edited by one of the authors. All authors then provided commentary.

Results: Both Westphal and Gélinau correctly identified and described the new clinical entities of cataplexy and narcolepsy, with recurrent, self-limited sleep attacks and/or cataplectic attacks affecting 2 otherwise healthy people. Narcolepsy was named by Gélinau (and cataplexy was named by Henneberg in 1916). The evidence in both cases is sufficiently

convincing to conclude that they were likely each HLA-DQB1*0602 positive and hypocretin deficient.

Conclusions: The original descriptions of narcolepsy and cataplexy are now available in English, allowing for extensive clinical and historical commentary.

Keywords: Narcolepsy, cataplexy, JBE Gélinau, C. Westphal, late 19th century, neurology, history of medicine, sleep disorders, motor dyscontrol, excessive sleepiness/sleep attacks

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To our knowledge, there are no published English translations of the first clinical reports describing narcolepsy (in French, 1880)¹ and cataplexy (in German, 1877).² The first author herein (CHS) had a professional translation agency (Berlitz) translate these 2 reports into English, which he then edited, as described below. (A minimum of 2 language experts reviewed each translated manuscript.) These historic documents richly describe recurrent, self-limited sleep attacks and/or cataplectic attacks in 2 otherwise healthy people.

Preliminary comments on the translations are as follows: First, all punctuations and italics come from the original articles. Second, the article by Gélinau was twice as long as the article by Westphal. Third, the translations were edited slightly. Two paragraphs from the Gélinau report that had no direct bearing on the description of narcolepsy or cataplexy were eliminated from the text, as indicated, and placed in an Appendix. Fourth, the Berlitz agency translator of the Gélinau report made this

comment: “The original French of this two-part article is written in an unusually loose style for late 19th century scientific reports. It is somewhat like a slightly-edited copying of hasty notes on a physician’s note pad. Accordingly, it is difficult to render in smooth English; we have in many cases sacrificed esthetics of style for accuracy.” Nevertheless, Passouant, who wrote about Gélinau for the narcolepsy centennial, mentioned that “Throughout his life, Gélinau wrote in a clear, alert, and easy-to-read style.”³ It is evident that Gélinau astutely identified and accurately named narcolepsy; he wrote an impressive set of descriptions on narcoleptic sleep attacks and their contexts, and he provided a detailed and carefully reasoned differential diagnosis and list of treatments. Westphal also correctly described the new clinical entities of cataplexy and narcolepsy. Therefore, both authors, in our opinion, deserve equal recognition for formally identifying narcolepsy and cataplexy.

(At this point the reader may wish to consider going directly to the translations of the original texts found in the final section, before returning to the commentary that immediately follows.)

GÉLINEAU'S DESCRIPTION OF NARCOLEPSY

Dr. Jean Baptiste Edouard Gélinau (1828-1906) at the outset of his report¹ attributed the initial description of narcolepsy to a Dr. Caffé who had published a case 18 years earlier, in 1862. However, a reading of Gélinau’s quotes from Caffé’s report would instead suggest the diagnosis of obstructive sleep apnea (OSA) as being more likely than narcolepsy. The case involved a 47-year-old man with “an irresistible and incessant propensity to sleep” that had forced him to resign from his job. He was not

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reported to have cataplexy, sleep paralysis, or hypnagogic/hypnopompic hallucinations. However, he was reported to have “attitude detached; stupor; mental sluggishness; persistent stoutness; effect on overall health,” and his face was “puffy.” These descriptions are more indicative of OSA than of narcolepsy. Dr. Caffé was apparently describing an overweight or obese patient when he used the word “stoutness”, particularly in the context of “persistent stoutness.” (One of the coauthors—IA—reinforced this conclusion in regards to the French word “fort” that describes a person being “overweight” distinctly more so than “strong,” both in the 19th century and in the contemporary French language). Although various treatments did not help Dr. Caffé’s patient, a stay at a spa did improve his condition. Is it possible that he lost weight at the spa, which would have had a beneficial effect on his presumed OSA?

Gélineau presents a 38-year-old man with a 2-year history of very frequent narcoleptic sleep attacks, totaling up to 200 attacks daily. This man could not speak with Dr. Gélineau for even 30 minutes without falling asleep and constantly needed his 13-year-old son at his side to keep awakening him, so that he could attend to his successful business. A wide array of intense emotional states played a prominent role in triggering his sleep attacks. The description of his initial visit with Dr. Gélineau is a dramatic example. In reading the entire report, a question could be raised as to whether this man—besides his “volatile temperament”—had histrionic personality traits that interacted with his narcolepsy.

Gélineau briefly described cataplexy (which he termed falls or “astasia”) and sleep paralysis in his patient but did not comment on the presence of sleep-onset dreaming, dream disturbance, or hypnagogic/hypnopompic hallucinations. He mentioned that his patient had “excellent night-time sleep, waking only once,” which argues against the presence of either disruptive periodic limb movements of sleep or REM sleep behavior disorder, conditions now known to be commonly associated with narcolepsy. Cataplexy was the initial manifestation of his narcolepsy. Gélineau’s patient was a member of the “mutual aid society,” and his card bore the diagnosis, “morbis sacer,” Latin for “sacred disease” in reference to epilepsy, which during antiquity had been considered a divine disorder.

Gélineau’s male patient reported that his infant child “was conceived in a moment when the illness came over him.” Among the various explanations to account for this intriguing comment, the most likely would be either a hypnagogic hallucination or a vivid sleep-onset REM dream, which are common events with narcolepsy that may have accounted for an imagined sexual event. Another possibility is that this man indeed had coitus with his wife while awake that was immediately followed by a sleep attack, and in retrospect he incorrectly recalled the coitus to have occurred during the sleep attack. Also, he may have experienced peri-coital cataplexy.

This patient received many unsuccessful treatments, including bromides, strychnine arsenate, curare, picrotoxin, apomorphine, phosphates, amyl nitrate vapors, hydrotherapy, electricity, and cauterization of the nape of his neck. Gélineau was thus led to comment, “as we both acknowledged that these successes were not in keeping with our mutual efforts, we lost contact, leaving to time and to nature the care of healing or improving this painful neurosis.”

WESTPHAL'S DESCRIPTION OF NARCOLEPSY-CATAPLEXY

Westphal had 2 cases that he presented at a Berlin Medical and Psychological Society meeting during 1877 that were then published in the Archives of Psychiatry and Nervous Disorders (for which he was an editor).² It is of note that he first chose to speak and write about “larvate epileptic attacks” before he described a patient with cataplectic attacks, and the publication of the former topic was twice the length of the latter topic. Westphal emphasized in italics 2 aspects of his patient’s clinical history: “He did not lose consciousness during these attacks,” and “persistent night-time sleeplessness must be noted.” Westphal clearly grasped that the cataplectic attacks involved loss of muscle tone without associated loss of consciousness, and his comment about sleeplessness may have indicated the presence of disrupted nocturnal sleep that is common in narcolepsy.

In being the first investigator to describe narcolepsy with cataplexy, Westphal was also the first to describe the possible existence of familial cataplexy, as the mother of his 36-year-old male patient had also suffered from longstanding, recurrent episodes of cataplexy and/or sleep attacks that were of milder severity than her son’s cataplexy, although “she had been troubled by such attacks frequently earlier on”. The observation that some of her attacks occurred when sitting quietly, while sewing or eating, is more suggestive of sleep episodes rather than cataplexy. There was no mention, however, of daytime sleepiness or any other features of the “narcolepsy tetrad.”

Westphal also described repeated sleep attacks in his patient: “At times...these attacks [viz. cataplexy] do cause the patient to fall asleep. The falling asleep appears, as it were, to be an extension or increase of the attack.” The patient would also have sleep attacks in public while “strolling around quietly and aimlessly.” These descriptions of sleep attacks and cataplectic attacks indicate that Westphal recognized and described narcolepsy with cataplexy before Gélineau, although he did not name these conditions, as did Gélineau for narcolepsy in 1880 and Henneberg⁴ for cataplexy in 1916. Whereas Gélineau described narcoleptic sleep attacks in great detail, Westphal only briefly described sleep attacks in a circumscribed manner as an extension of a cataplectic attack or as a consequence of aimless wandering. It is noteworthy that only in 1902 a third author (Löwenfeld) confirmed Westphal’s and Gélineau’s suggestion that narcolepsy with cataplexy represents a “disease sui generis.”⁵

THE HISTORICAL CONTEXT OF THE WESTPHAL AND GÉLINEAU REPORTS

The forceful unification of Germany by Prussia’s Otto von Bismark was completed after first defeating Austria and then the French armies during the short 1870 war against Napoleon the Third. Germany was a strong but barely united country. France had lost the Alsace and the Lorraine regions and was separated from Germany both culturally and linguistically. Psychoanalysis was not formally established, as Sigmund Freud had not yet completed medical school, but there was growing interest in the unconscious and in psychological explanations for physical disorders. The pioneering work of Jean Martin Charcot’s “Leçons sur les Maladies du Système Nerveux” had just been published, introducing the notion of hysteria.⁶ Neurology and psychiatry were still virtually one discipline.



Figure 1—Portrait of Karl Friedrich Otto Westphal (1833-1890) reproduced with permission from www.mrcophth.com/ophthalmologyhalloffame/westphal.jpg

ON THE AUTHORS

Karl Friedrich Otto Westphal, born in 1833 in Berlin, was the son of a well-known and wealthy physician. After a European medical education that included studies in Germany, Switzerland, and France, he joined the smallpox clinic at the Berlin Charité hospital to rise to become full professor of psychiatry in 1874. He trained a number of well-known physicians including Arnold Pick and Carl Wernicke. His achievements were numerous and included the first descriptions of agoraphobia;⁷ the first description of periodic paralysis; the report of a relationship between tabes dorsalis and general paralysis of the insane, prefiguring the syphilis connection; work on pseudosclerosis; and the first description of the deep tendon reflex. In 1887, two years after Ludwig Edinger, he described the accessory nucleus of the 3rd nerve which bears his name. His picture is that of a well-groomed, bearded aristocratic man with a bow tie (Figure 1). Dr. Westphal died in 1890 and is not frequently credited for his report on narcolepsy-cataplexy,² which was linked to the possible forensic implications of sleep attacks.⁸

Jean Baptiste Edouard Gélinau had quite a different career that took place outside of the medical establishment. Born in 1828 close to Bordeaux in the south of France (Blaye, Gascony), he was educated as a navy physician in Rochefort and practiced medicine on ships, studying tropical disorders on his frequent and long



Figure 2—Portrait and signature of Jean Baptiste Edouard Gélinau,⁹ reproduced with permission.

travels to the Indian Ocean. He spent the war as a surgeon-major and was decorated for his services.^{3,9} With his large muttonchop beard (Figure 2), it is easy to imagine him with the flamboyant and proud character of people born in the country of Cyrano de Bergerac and of *The Three Musketeers*. Not only was Dr. Gélinau a prolific writer of medical articles and monographs, he also had a great deal of business acumen. Dr. Gélinau was known for his arsenic-bromide tablets to calm neurosis and epilepsy, was involved in coordinating a medical insurance system for older physicians, and founded a successful society of health spas and mineral waters. In 1878, he moved to Paris, to rapidly establish a successful private practice, a position he left only in 1900 to retire as a wine grower, owner of the castle of Saint-Luce-La-Tour and seller of Bordeaux wines (probably thanks to the success of his tablets). Dr. Gélinau's publications are eclectic and cover literature, the history of his native town, commercial ventures, and medical studies. His medical work includes observations on tropical diseases, postpartum psychosis, neurosis, angina pectoris, phobias, deafness, and epilepsy. He is credited for coining the term "narcolepsy" in the attached translated 1880 report,¹ and for forcefully defending it as a disease entity distinct from epilepsy.

Interestingly, Dr. Gélinau also published a monograph in 1880 on agoraphobia,¹⁰ citing Westphal's work on the topic ("agoraphobie des Allemands"). This indicates knowledge of the work of the German physician prior to his own 1880 article or discovered just after his *Gazette des Hôpitaux* publication. In 1881, Dr. Gélinau wrote a more detailed account on 14 narcolepsy cases in a monograph "De la narcolepsie,"¹¹ still not citing Westphal's 1877 narcolepsy report. A careful review of the cases reported in the monograph, however, suggests that most, if not all (except the original 1880 case) are not genuine narcolepsy-cataplexy. Whether or not Dr. Gélinau spoke German and if the 2 physicians met or corresponded is unknown, but certainly possible.

ON THE CLINICAL DESCRIPTIONS

There is no doubt that both Westphal's and Gélinau's cases have genuine narcolepsy-cataplexy. Both physicians report on the presence of sleepiness and of strange episodes of either sleep or atonia triggered by emotions, which we now call cataplexy. In both cases, onset was somewhat late in life, 34-36 years old, and abrupt, following what could be considered a psychological insult. Earlier reports of narcolepsy have been attributed to Willis (1672, in "De anima brutorum"), Schindler (1829), Bright (1836), Graves (1851), Caffé (1862), and Fischer (1878),¹² but they in fact described cases of either isolated severe, overwhelming (narcolepsy-like) sleepiness or atypical/ imprecisely described (cataplexy-like) "fits."¹²⁻¹⁴ In contrast, the cases described by Westphal and Gélinau are likely to be HLA-DQB1*0602 positive, hypocretin deficient cases.

A missing aspect in these reports is the lack of description of automatic behavior, abnormal dreaming, and sleep paralysis. Hypnagogic hallucinations in particular had been described in 1848 by Alfred Maury¹⁵ and sleep paralysis by Binns in 1842¹⁶ and by Mitchell in 1876,¹⁷ but were not reported in either Gélinau's or Westphal's case. Nevertheless, the reports of Gélinau and Westphal are remarkable for their diversity and, in both cases, by the certainty of the 2 authors reporting on a new disease entity (later authors erroneously equated "narcolepsy" with every condition associated with severe daytime sleepiness¹⁸). The descriptions are tainted by their schooling and influenced by their time. Nonetheless, nothing better would be written for many years thereafter, and it could be argued that the next major discovery in narcolepsy was on the association of narcolepsy with REM sleep onset by Vogel in 1960,¹⁹ almost a century later.

In Westphal's case, the description of the case is mostly focused on episodes of muscle weakness with persistence of consciousness, and in the discussion the author agonized at length on whether these episodes did or did not represent genuine epilepsy, and wisely summarized that it was impossible to conclude for or against this hypothesis. Westphal pointed out correctly the presence of subtle "positive" motor phenomena during cataplexy consisting of "small sporadic nostril contractions" and "slight twitching movements in the face... as were movements of the jaw." The precise observation has been confirmed by electrophysiological recordings.²⁰ Emotional triggers are also noted but are not very well described ("mental stimulation of seeing two boys fighting in the street"; "any type of excitation"). Laughter and joking, for example, are not reported as triggers. It is in this context to note that Oppenheim, in his 1902 article on "Lachschlag" (syncope with laughing), while discussing the differential diagnosis of spells associated with laughing, did not mention narcolepsy.²¹

Sleep attacks are noted to occur "especially if not engaged in some physical activity, but is sitting quietly, talking or reading" but also "while standing" and "while walking in the street." Sleep attacks while engaged in physical activity are indeed typical although not specific for narcolepsy. A relationship and an association of the muscle weakness episodes with sleepiness is emphasized by Westphal, and considered as an extension of the muscle weakness episodes ("at times, however, these attacks do cause the patient to fall asleep"). The German author did not completely differentiate the sleep attacks from cataplectic episodes, an ambiguity which may have reflected the simultaneous co-occurrence of both symptoms in his patient (as can be observed occasionally in

narcoleptics). This ambiguity may have also reflected Westphal's uncertainty about the true nature of the sleep attacks. It is of interest to note in fact that in Oppenheim's "Lehrbuch der Nervenkrankheiten," the most important German textbook of neurology at the beginning of the 19th century, such episodes were considered to represent episodes of "psychic immobility" with muscle weakness, rather than "true" sleep attacks.²² Insomnia and the absence of any response to potassium bromate were also noted by Westphal in his report.

Interestingly, Westphal noted that the patient's mother also suffered from similar episodes, following a head trauma. In this case, however, it is difficult to make a conclusion as maintenance of postural tone is reported, and the episodes may be more reminiscent of absence seizures. Similarly, in the case of Fisher (1878),¹² i.e. a younger case where cataplexy is not described with certainty, a sister is suggested to have had the same condition, to later outgrow it during adulthood.

Further discussion of Westphal's cases also attest to the rise of "pre-psychoanalytic" ideas, already evident in Westphal's prior studies on "sexual inversion" and homosexuality.²³ Detailed reference to and discussion of the case of Van Zastrow, a famous criminal pedophile evaluated by the author in prison, is made. Contrary to what was generally believed in this time, the author was surprised not to find the criminal epileptic (epilepsy was frequently considered at the time a sign of "mental degeneration"), but rather excessively sleepy, falling frequently asleep in public (the symptom was severe enough that people were laughing about it). A relationship between his sleepiness and his alleged frequent masturbation, repressed homosexuality, and associated shame is suggested, with the prisoner attributing his sleepiness to the "secret vice of masturbation" to which he had become "addicted." Whether Mr. Van Zastrow had obstructive sleep apnea or Kleine-Levin syndrome is impossible to reconstitute, but narcolepsy is not likely.

Dr. Gélinau's report is complementary to Westphal's. Its style is more descriptive, "story telling." A potential head trauma 2 years prior to onset is reported as a possible contributing factor. Whereas Westphal was more interested in the loss of muscle tone and the sleep attacks (as reflected by the title of his communication), Gélinau was fascinated by sleep attacks during active tasks such as eating and by the existence of refreshing short naps. Cataplexy is confused with sleep attacks, but its triggers are very well described, i.e., playing cards (and having a good hand), smiling at someone poorly dressed in the street, being surprised by a sudden danger, and anticipating the pleasure of a good play in the theater. Most telling is the story of this patient going to the zoo of the Jardin des Plantes and "falling asleep" in front of the monkey's cage when everyone was laughing around him. The patient had up to 200 episodes per day, suggesting a form of "status cataplecticus."

Although Gélinau did not formally distinguish cataplexy from sleep attacks, it is of interest to note that in naming narcolepsy he identified "narcolepsy's twofold analogy with drowsiness and catalepsy." Catalepsy, however, involves prolonged maintenance of body posture, which is a form of motor dyscontrol opposite to cataplexy. Gélinau's use of the word catalepsy may refer to the patient maintaining posture during some sleep attacks, which were not cataplectic attacks. Nevertheless, his use of the term "astasia", viz. falling episodes, in relation to narcolepsy prefigured the naming of cataplexy in 1916 by Henneberg, who clearly differentiated sleep attacks from episodes of muscle paralysis triggered by emotions.

A second article follows the initial report where Gélinau excludes potential differential diagnoses including vertigo, epilepsy, agoraphobia, anxiety, meningitis, and sleeping sickness, and to conclude that narcolepsy is a unique disease entity. As mentioned above, Gélinau also wrote a monograph reporting on 13 additional cases, none of whom is likely to have genuine narcolepsy. Gélinau described how decreased brain tissue oxygenation and metabolism in the pons, the “site of emotional regulation and dreams” could occur in selected predisposed patients or was caused, in 2 patients, by too much sex (“Venus’ pleasures”). Decreased oxygenation would be precipitated by emotions, considered as consuming too much oxygen and energy. Gélinau also reports on numerous therapeutic attempts. Therapies aimed at relieving a potential vasomotor abnormality, including picrotoxin and amyl nitrate to induce vasodilation, were tried without success. Further trials with apomorphine had no efficacy. Interestingly, he tried to give strychnine (which is now known to block postsynaptic glycinergic transmission, in particular at the spinal motor neuron where it could antagonize REM sleep-induced atonia), but obtained only a transitory effect. Dr. Gélinau finally suggested caffeine to treat the narcoleptic sleepiness (as originally suggested in 1672 by Willis²⁴), despite the fact it was of little benefit in his only genuine case. A more potent treatment than caffeine, i.e., ephedrine sulfate, was suggested by Janota and Daniels about 50 years later.²⁴

Gélinau considered in his monograph that the sleep of narcoleptic patients was deep and devoid of dreams, which suggests, as emphasized below, that the 13 other cases were probably not narcoleptic. Importantly however, he introduced the still-valid notion of a duality in narcolepsy, that of sleepiness and falls (also called astasia).

REFERENCES

- Gélinau JBE. De la narcolepsie. *Gazette des Hôpitaux* 1880;53:626-8;54:635-7.
- Westphal C. Eigentümliche mit Einschlafen verbundene Anfälle. *Archiv für Psychiatrie und Nervenkrankheiten* 1877;7:631-5.
- Passouant P. Doctor Gélinau (1828-1906): Narcolepsy centennial. *Sleep* 1981;4:241-6.
- Henneberg R. Ueber genuine Narkolepsie. *Neurol Zbl* 1916;30:282-90.
- Löwenfeld L. Ueber Narkolepsie. *Münchener Medizinische Wochenschrift* 1902;49:1041-5.
- Charcot JM (1877). *Leçons sur les maladies du système nerveux faites à la Salpêtrière*. Tome deuxième. Bourneville DM, Ed. Delahaye VA, Paris; 496 p.
- Westphal C (1871). Die Agoraphobie, eine neuropathische Einescheinung. *Arch. für Psychiatrie Nervenkr.* 3: 138-161.
- Holdorff B. Carl Westphal (1833-1890). *J Neurol* 2005;252:1288-9.
- Passouant P (1981). La narcolepsie du temps de Gelineau. *Histoire des Sciences Naturelles*, 2: 1-7.
- Gélinau JB (1880). De la kénophobie ou la peur des espaces (agoraphobie des Allemands). Tessier, imprimerie Surgères (Charente-Inferieure), 90 p.
- Gélinau JB (1881). De la narcolepsie. Tessier, imprimerie Surgères (Charente-Inferieure), 63 p.
- Fischer F. Epileptoide Schlafzustände. *Arch J Psychiatr* 1878;8:200-3.
- Passouant P. The history of narcolepsy. In: Guilleminault C, Dement WC, Passouant P, eds. *Narcolepsy Proceedings of the First International Symposium on Narcolepsy*. Montpellier, France: Spectrum Publications, Inc.; 1976:3-13.
- Furukawa T. Heinrich Bruno Schindler’s description of narcolepsy in 1829. *Neurology* 1987;37:146.
- Maury A. Des hallucinations hypnagogiques ou erreurs des sens dans l’état intermédiaire entre la veille et le sommeil. *Ann Med Psy* 1848;11:26-40.
- Binns E. *The anatomy of sleep; or the art of procuring a sound and refreshing slumber at will*. London: J Churchill; 1842.
- Mitchell SW. On some disorders of sleep. *Va Med Monthly* 1876;2:769-81.
- Blocq P. Semeiology of sleep. *Brain* 1891;14:112-26.
- Vogel G. Studies in psychophysiology of dreams III. The dreams of narcolepsy. *Archives of General Psychiatry* 1960;3:421-8.
- Rubboli G, d’Orsi G, Zaniboni A, et al. A video-polygraphic analysis of the cataplectic attack. *Clin Neurophysiol* 2000;111(Suppl 2): S120-8.
- Oppenheim H. Ueber Lachschlag. *Monatschrift für Psychiatrie und Neurologie* 1902;11:242-7.
- Oppenheim H. *Lehrbuch der Nervenkrankheiten*. Berlin: S.Karger; 1913.
- Westphal C. Die konträre Sexualempfindung. Symptom eines neuropathischen (psychopathischen) Zustandes. *Arch Psychiat Nervenkr* 1869;2:73-108.
- Lennox WG. Thomas Willis on Narcolepsy. *Archives of Neurology and Psychiatry* 1939;41:348-51.

ENGLISH TRANSLATIONS OF THE ORIGINAL REPORTS ON NARCOLEPSY AND CATAPLEXY BY WESTPHAL AND GÉLINEAU

Archives of Psychiatry and Nervous Disorders

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BERLIN, 1877

“TWO MEDICAL CASES”

PRESENTED AT THE BERLIN MEDICAL AND PSYCHOLOGICAL SOCIETY

BY PROF. C. WESTPHAL

I. Larvate epileptic attacks many years before the outbreak of a paralytic mental disorder. (pages 622-631).

II. Peculiar attacks associated with falling asleep. (pages 631-635).

II.

Mr. Ehlert, a bookbinder, was admitted to Charité for the first time on July 18, 1871. He has been admitted a few times since then, and is there now. He is 36, and is reported always to have been healthy. Approximately three months before his first admittance, he became ill, he says, as the result of a fit of anger. He had lost his job because of quarreling. After having a few drinks of schnapps (he is reputedly not a drinker), he went home, where he was scolded by his wife. Soon thereafter, he had a brief “fit” (1—1 ½ minutes), characterized by a loss of speech, or at least an inability to express words clearly. His whole body was trembling (the patient called it “agitation”), so that he had to sit down (he reported that he had an “involuntary compulsion to sit down”). This “agitation” is said to have continued throughout the entire evening. He slept well that night. He says that he felt completely fine the next day, but a similar condition (in which he lost his capacity to speak and experienced trembling) occurred thereafter at the least mental stimulation, e.g. once when he saw two boys fighting in the street and had, in his mind, taken sides with one of them. Headaches and other complaints never occurred in these instanc-

es. Thereafter he was employed in a workshop, which was heated even on hot days. He cites these circumstances as the reason for the increased frequency of the attacks. Approximately 10 weeks before his admittance, the attacks changed so that his teeth chattered, speaking was difficult and, if he had anything in his hands, he would have to lay it aside, because he did not have the strength to continue holding it. During these attacks, he was unable to raise his arms. If the attack came upon him while walking or standing, he had to find some means of support, although a cane was sufficient for the purpose. These attacks varied in duration, depending on whether he had exerted himself beforehand. *He did not lose consciousness during these attacks.* He understood everything when spoken to; he was simply unable to respond coherently or fluently. He always had to close his eyes when doing so.

According to the patient, his mother, who had been struck in the head by a falling brick earlier, also suffers from similar attacks. Specifically, her attacks occur while she is sitting quietly, sewing, eating, or while drinking coffee from her saucer, for example. When asked, he expressly stated that these occurrences in his 61-year-old mother were not caused by any type of senility, and that she had been troubled by such attacks frequently earlier on.

I have had the opportunity to observe the attacks in the patient himself on repeated occasions. He had one of these attacks while I was engaged in conversation with him. While he was still speaking, one could see that a certain change had occurred in his facial coloration, his upper eyelids lowered gradually like those of a person falling asleep (during which the eyes roll upward). Then they opened again once or twice, seemingly with great effort, until they finally shut completely, whereupon the patient stopped speaking after murmuring something incomprehensible. His head sank down to his chest, and his brow seemed forcefully knit. Small sporadic nostril contractions were observable, and the patient's appearance was that of a seated person asleep. After a short time (several minutes), the eyebrows relaxed, the patient raised his right arm a few times as if stretching upward, and rubbed his eyes sleepily, like one awakening from slumber. The scene then repeated itself all over again, during which one could observe that, though apparently asleep, the patient hears if one addresses him, since he nods in response to questions directed to him. Afterwards, he also knows everything that was said during the time.

He experiences many such attacks all day long, especially if he is not engaged in some physical activity, but is sitting quietly, talking or reading. However, even when occupied in a physical task he often undergoes these attacks, e.g. while helping wash the dishes. He then sits down on a bench, continues holding the objects that he had in his hand, nods off, and usually returns to his activity a few minutes later. As he says, he has noticed, as corroborated by others, that the attacks certainly usually start at a specific place in a particular situation. For example, from time to time he has to get papers and other objects from the chief attendant's office. Almost always, while standing, he nods off as described above immediately after picking up these objects; he staggers, with his head on his chest and his trunk bent forward like one intoxicated with sleep, from the office out into the corridor. He then proceeds down the corridor, and after taking a few steps the attack is over. He never drops the objects given to him, but he holds them differently. He does not carry them with outstretched arms, as before, but his arms hang down loose. He does not lose consciousness at all during these attacks. He says that when he

enters the office, his spirit becomes uneasy, that he feels a kind of anxiety, and it seems to him as though something had happened to him there before.

The attacks always come on suddenly. When he was a porter, he had such an attack when a man was giving him an order. The man thought that he was drunk, and told a policeman who happened to be there that he wanted him arrested. Meanwhile the attack passed, and the policeman was quite amazed when the patient reasonably explained to him that it was a medical condition. The patient still had time to run after the man, and to ask him for the order again. He further related that once, when he was leaning far forward over the table to get something from the other side, he experienced an attack in that position, and that he stayed in that position until it had passed.

His information about the sensations that he has during these attacks is as follows. His eyes close involuntarily, and he cannot keep them open. If he manages to open them for a moment, he sees a bright light, but cannot make anything out distinctly. At the same time, he loses all strength in his limbs and the ability to speak. He cannot move, and must sit or lean on something. He says that he does not feel tired like someone on the verge of falling asleep. In his mind, it is as though he were thinking of nothing at all, as if his thoughts were wandering completely. He could not provide a more specific description of his mental condition. He says that he does not experience any dizziness. He reports that he hears and understands what is said to him during the attack, but only pays attention to it if it interests him somewhat.

At times, however, these attacks do cause the patient to fall asleep. The falling asleep appears, as it were, to be an extension or increase of the attack. He says that if he can stretch, the attacks do not go to that extreme. During visits, one often finds the patient already asleep, and one can observe him for fairly long periods at a stretch in that condition. The image is exactly that of a person sleeping peacefully in a seated position. By simply calling his name, he can always be awakened, is aware that he had been sleeping, and notes particularly that upon awakening he is immediately lively and alert, not drowsy. He has also experienced this actual falling asleep while walking in the street. Most often, he steps into the gutter or runs into a lamppost or a person, whereby he is suddenly awakened. He has also stayed asleep in the street and a passer-by, tapping him on the shoulder, wakes him saying, "My good man, you're asleep!" Occasionally another attack occurs after he walks about another hundred paces. This falling asleep in the street, says the patient, usually does not happen if he has a specific destination, but occurs more often when he is strolling around quietly and aimlessly.

Aside from what has been described above, the patient also has attacks that he characterizes as more severe. I was witness to one, which he says falls into this category. The patient was brought into the room by an attendant walking behind him. The patient was completely limp, his eyes were closed, and he was staggering like an intoxicated person, and had difficulty in maintaining his balance. Then all support was removed, and the patient stood free, with only a slight swaying motion, but did not fall. During this time, slight twitching movements in the face were observed, as were movements of the jaw. The eyes were half shut, and the whites of the eyes, which appeared to be rolled up and to the right, remained visible. Respiration was rapid, with sighing. At times it seemed as though the patient was searching for a chair or a seat to hold himself up, but he only

made motions with his head that corresponded to such a search, and did not use his eyes. Finally, he was able to reach the edge of a bed, which he then held onto. Toward the end of the attack, he murmured, “Chair,” and then said immediately, “Professor, please excuse me while I take a seat,” with his eyes still half shut and continued rapid breathing. Although the attack had given the observers the impression that the patient had been unconscious, when asked, he said that he had been fully conscious during the entire attack, and knew exactly which attendant had brought him into the room.

No specific indication of the onset of the attack in this or any form can be determined through observation. The patient himself states quite clearly that any type of excitation, even of the most minimal kind, is very often the trigger for the attacks. He says that they often occur immediately after such excitation.

The patient’s intelligence leaves nothing to be desired, and his demeanor is generally calm and reasonable, and no particularly violent outbreaks have ever occurred, as far as we know, although he is easily roused.

Finally, persistent *night-time sleeplessness* must be noted. He says that he spends only a very small portion of the night sleeping, and that the night-time disturbances of other patients are a kind of entertainment for him, rather than making him uncomfortable.

During his first stay at Charité (July 18, 1871 to December 22, 1871), he was treated consistently with potassium bromate, but to no avail.

As is clear from the medical history, the patient attributes the onset of these attacks to a significant emotion. It is also noteworthy that his mother at times falls asleep while performing ordinary chores. However, the patient notes that there is a difference, in that his mother does not lose control of her limbs during the attacks, as he does, but that when she is drinking coffee, for example, the hand bringing the full saucer to her mouth remains in that position, whereas it would be impossible for him to maintain such a position.

One is faced with a predicament in attempting to attribute a name to the illness described above. It would be a simple matter to call these episodes “epileptoid” attacks, as well, and I cannot object to the term, if one wishes to lengthen the list of very varied conditions commonly called by that name. This does not advance our understanding at all, however, and the peculiarity of the attacks, to which I need not add any further detail given the exhaustive description above, persists nonetheless.

However, I would like to draw attention to this case not solely for its interesting pathology, great as it may be, but in the interests of forensics. A number of years ago, I collaborated with Messrs. Liman and Skrzeczka on the task of providing an opinion regarding the mental state of the well-known v. Zastrow *) [*] He had committed pederastic rape of a boy, H., and had even made attempts on the child’s life.] In the course of the medical examination, as I was attempting to uncover any prior epileptic events, but turned up absolutely nothing, as had been indicated, a chance comment of v. Zastrow struck me, namely that he had often fallen asleep on social occasions, and that he had frequently been laughed at as a result. Since I no longer have in my possession a copy of the opinion in which my relevant notes on this case are kept, I present the case here as reported in the opinion written and communicated by Liman: **) [**] Casper-Liman, *Practical Manual of Forensic Medicine*. Vol. 1, p. 497, 5th Edition, 1871.]...“he said that he had not noticed anything unusual about himself, and

he could only report that following fairly considerable mental exertion, e.g. after fairly extensive reading aloud, he found himself to be half asleep, subject to the derision of those around him. He said that this condition had come over him rather frequently while walking, so that he had to ask directions to find his way. He said that it must be the result of such a secret vice as the one to which he had become addicted (i.e. masturbation).”

In this instance, I was spontaneously reminded of the medical case reported above, and one cannot deny that if additional observations should uncover a fairly common occurrence of such “sleep attacks,” then we are in the presence of a pathological manifestation of the nervous system, which, in the exploration of the mental condition of certain categories of criminals, deserves no less consideration than epileptic or epileptoid attacks. It is evident that for the time being nothing less than a disease of the central nervous system can be concluded, and that the question of responsibility in and of itself is not involved.

GAZETTE OF THE CIVIL AND MILITARY HOSPITALS OF THE OTTOMAN EMPIRE

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“ON NARCOLEPSY”

BY DR. GÉLINEAU

I.

I am proposing the name narcolepsy (from the Greek “narcosis,” drowsiness, and “lambanein,” to seize, to take) for a rare neurosis, or at least one that has been little known until now, characterized by a sudden, brief, urgent need to sleep, which recurs at varying-spaced, close intervals. This name calls to mind narcolepsy’s twofold analogy with drowsiness and catalepsy.

Initially, I believed that the case I had observed (reported below) was the only known instance; however, in Dr. Delasiauve’s *Journal de médecine mentale*, nos. 8 and 9, vol. II, 1862, I have just read that Dr. Caffé published an initial case of this sleep neurosis in his *Journal des connaissances médicales pratiques* (August 20, 1862). I am pleased to report this case here, as undeniable proof of its existence.

CASE I. “For more than a year,” states Dr. Caffé, “I observed an employee of the Grand Cercle, 16 Boulevard Montmartre, who, because of an irresistible and incessant propensity to sleep, was forced to resign his position. This forty-seven year old man was tall and strong, married, and had always lived soberly. He had no history of illness, and the first external sign was heaviness and half-closure of the eyelids. This drowsiness, which varied in severity depending on circumstances, had affected him for more than four years, coming on while he was standing, sitting, lying down, or while walking. If he woke up, he would fall back to sleep immediately. Even the most pressing hunger did little to divert these effects; his face was somewhat pale and puffy; attitude detached; stupor, mental sluggishness; persistent stoutness; effect on overall health.

Various treatments were unsuccessful, and a stay at the spa at Brides served only to improve his condition, but not result in

complete recovery.

Later, after a terrifying emotional experience and illicit excesses (abuse of coitus, masturbation, and alcoholic beverages), he suffered hallucination and meningitic delirium, for which he was intensively treated by Dr. Semelaigne.”

CASE II (my own observation). Mr. G., age thirty-eight, a barrel seller with a nervous, volatile temperament, came to my clinic on February 15, 1879.

He had not experienced convulsions in his youth, nor syphilis at a later age. He has two children, the elder of whom, age thirteen, always accompanies him, and the second of whom is only a few months old. G.'s father was nervous, but was free from illness; his mother died of cancer, and his brother of a stomach ulcer. He drinks moderately. Five years ago, he suffered acute rheumatism in the joints and Herpes tonsurans at the same time.

Three years ago, during a heated argument, he received a violent blow of the fist from the other party, to which he responded by striking his opponent with a drill, after which he was physically apprehended by a policeman and imprisoned; it was a deeply distressing incident.

Finally, a short time later, a log fell on his head, although it did not cause any great pain; and I find no sensitivity at that spot nor any depression worthy of note.

For a long time, this individual experienced no consequential phenomena. Only in the past two years, when laughing out loud or when anticipating a good business deal in his profession, he would feel weakness in his legs, which would buckle under him. Later, when playing cards, if he was dealt a good hand he would freeze, unable to move his arms. His head would nod forward and he would fall asleep. He would wake up a minute later. Soon, the slightest emotion—the sight of his barrels, for example—would be enough to bring on sleep, and since then, this urgent need to sleep has bothered him constantly. When he eats, his meal is interrupted four or five times by the need to rest. His eyelids droop, his hands drop the fork, knife, or glass. He has trouble finishing a sentence, falls asleep. Rubbing his eyes to ward off this sensation while seated is of no avail. His hands fall inert, he is overcome, bends forward, and falls asleep. If he is standing in the street, when this need comes over him, he wavers, stumbles like a drunk, hears people accuse him of drinking and make fun of him. He cannot answer them. Their taunting overwhelms him all the more, and he collapses, instinctively avoiding passing carriages or horses by a final effort. When several people then form a circle around him which always happens in Paris, he hears them or perceives them offering sympathy, and their amiability paralyzes him, affecting him even more and preventing him from getting up.

If he experiences a deep emotion, whether painful or joyous, the need to sleep is even more urgent and sudden. Thus, for example, if he is closing a good business deal, if he sees a friend, if he speaks with a stranger for the first time, or if he receives a good hand while playing cards, he collapses and falls asleep. If he goes to the Jardin des Plantes, near the Monkey House, the place where curiosity-seekers, children's nannies, soldiers, and hecklers usually congregate, he falls asleep seeing this whole laughing crowd around him. A bolting horse, a carriage about to cross his path, or the sight of a person grotesquely dressed who causes him to smile is all he needs to suffer an attack.

At the theater, he falls asleep at the mere thought of the pleasure he is going to experience. He falls asleep again when sitting in his seat, and his son has to shake him and pinch him to pull him

out of it. Once the actors come on stage, however, the need disappears; he follows the play with great interest, not collapsing for a single instant, unless a poignant act arouses too great an emotion in him.

Bad weather, particularly the approach of a storm, increases the frequency of these sleep attacks; he has experienced up to two hundred per day.

The only way to pull him out of these attacks is to shake him strongly, or to pinch him. When he becomes violently angry, he sleeps less, but longer and deeper. When he wakes up, he walks straight and firmly, until a new sleep attack comes over him a quarter of an hour later.

I will always remember the way he entered my clinic. He was guided and supported by his son, who held him by the arm. No sooner had he passed through the door of my office and turned his eyes toward me, than, frozen, his gaze glazed over, his eyelids drooped, he staggered, stumbled, and fell asleep, onto a chair; his son spoke to him and shook him hard, after which he began to speak to me.

During his sleep, his pulse, which ranges from 66 to 68 ordinarily, immediately drops to 38 to 60. His pupils, which are highly contracted when awake, are slightly less so when he is asleep. His pupils contract once again when they are raised and brought near the light. The attacks last one to five minutes.

In addition, nothing in him gives evidence of a state of illness; he is calm, at ease. He eats well and his night-time sleep is excellent, waking only once. He has coffee once a day and is not constipated. His sexual desires have diminished considerably. I should repeat that he has just had a child; he says, however, that the child was conceived in a moment when the illness came over him.

A member of the mutual aid society, his card bears the diagnosis *morbus sacer*. He has been treated at his home and at Salpêtrière. When he was going there, he fell asleep several times at the door of the hospital, then at the door of the room, and finally, for the third time, confronting the doctor whom he was there to consult. They recommended potassium bromide, subcutaneous injections, hydrotherapy, electricity, and finally, they cauterized the nape of his neck, but, he says, none of this brought any improvement.

When asked to explain his disease and its onset as best he could, he said that he never feels any pain when he is overcome. He merely feels a deep heaviness, an intracranial emptiness, a sort of whirlwind spinning around inside his head, and a heavy weight on his forehead and in back of his eyes. His thoughts dim and fade; his eyelids close half way. He continues to hear, and he is conscious. Finally, his eyelids close completely, and he sleeps. All of this occurs very quickly, so that the normal physiological sleep phase which occurs in progressive periods of five, ten, and twenty minutes, lasts at most a few seconds for him.

If one has him close his eyes and asks him to speak and walk, as is done in cases of ataxia, his voice fades out, he falls asleep and collapses, but without disordered movements. If he enters a dark place, such as a cellar, he also has increased tendency to fall asleep. When he descends a steep street, he has difficulty remaining standing; also, when he pushes a wheelbarrow, with a small cart hitched to him from behind, he pulls it along easily behind him by means of a harness, and he does not fall asleep, probably because his will is more intense at that particular moment.

During his morbid sleep, he never releases any urine or fecal matter. At my office, he has on occasion spoken for a half hour without falling asleep.

His memory is not affected in the least. He is aware of the status of his business, and he is actively involved in taking care of it, but he is always accompanied, because he cannot go out alone without risk of danger. When he works alone, he has fewer attacks than when he is with someone; this is because he enjoys talking, becomes animated and falls asleep.

The intermittent appearance of this illness, its frequency, its lack of resulting injury would place it in the category of a neurosis. The question arises, however, as to whether it should be included under a type already known, or whether it deserves a place apart in this group that is so large and already so numerous? That is what we shall examine.

First, is this a form of *epilepsy*? I do not think so... He does not experience either tonic convulsions or clonic movements. He feels when he is pinched. He is always conscious of what is happening around him. When one shakes him, one can rouse him from his sleep. He does not stammer when he wakes up, and he recovers his intellectual faculties, his senses, and his motility immediately. Moreover, far from overwhelming him, this rest seems to be necessary for him, and appears to give him strength. Finally, his recall is perfect. In addition, potassium bromide, that touchstone of epileptic seizures and epilepsy, has had no positive effect on him. Besides, what epileptic, after one or two hundred spells of dizziness and falls per day, would keep his intelligence and memory intact after two years?

Dr. Semelaigne, however, sought to link his subject's illness to epilepsy. "One symptom," he said, "predominated and masked the others, but their occurrence together is nonetheless significant. Everyone knows that attacks of dizziness can occur for a long time without revealing their true character. Everyone is also aware that such attacks result in drowsiness, mental sluggishness, a weakening of the memory, problems with the brain, stupidity, and moral perversion. — In the absence of attacks of dizziness, or when the attacks cease, intelligence and the moral sense return. That was the situation with M.... --- Certainly, the drowsiness first attracted attention as the predominant symptom, but several times a day he experienced "blackouts", dizziness or pseudo-attacks as they are called. It is generally drowsiness and not stupor which precedes this sort of attack." With regard to the slight cerebral congestion, Dr. Semelaigne says that this is one of the most frequent complications of epilepsy. Finally, the acute meningitic delirium which struck the patient also falls within the "domain of mal comitial".

We reproduce our colleague's opinions in full, but they are not at all convincing. Here is a man who has had continual falls and dizziness for four years, and has never had a full, typical epileptic seizure. He falls, and his drowsiness ceases after the attack; he falls and the *ictus* never causes him to fall stiff, with resultant injuries of the type so common among epileptics. He falls and immediately recovers his wits, his intelligence. Ah! This is because his fall is similar to that of a drunken person or a sleeping child. It is a collapse caused and *preceded* by drowsiness, whereas in the epileptic seizure, sleep *comes after* the fall. Let us add, finally, that Dr. Semelaigne does not mention the one thing that, for us, constitutes the *criterion* of epilepsy from its mildest to its most severe manifestations: the loss of memory, of recollection of what just happened. A subject who remembers and is conscious of what is happening and what happened after an attack of dizziness, an absence, a fall, is not an epileptic.

II.

Can one confuse the affliction from which G. suffers with kenophobia (from the Greek "kenon," the void; "phobeo," I fear), or the fear of open spaces, to use Mr. Legrand du Saulle's term, or agoraphobia, as the Germans put it? Not anymore. Clearly, when crossing a fairly wide street, a square, he is frightened, upset, hesitant. But it is less the view of the open space which affects and frightens him than the fear of being surprised by a carriage, a wagon, or horses. When emotion stops him in his tracks is the moment that sleep overcomes him, and freezes him in place. Also, a person suffering from kenophobia does not fall asleep. He moans, looks about, shouts, makes gestures, calls, and backs away if no one comes to offer him a hand. G., when moved or upset, does not reason or look around; he goes to sleep and falls.

One cannot confuse this affliction with vertigo accompanied by syncope, falling, and the loss of consciousness. First, in the beginning, the objects that surround G. do not appear to him to be moving. Although his eyelids are half closed, he sees them as immobile. When he rocks, the reason is that, since he has stopped seeing, he wants to stretch out to sleep. He does not try to hold himself up by the objects surrounding him, as a dizzy person does. He yields without a struggle. For G., sleep is the rule. For a man experiencing a dizzy spell, syncope is the exception. Finally, what a difference between G., sleeping peacefully, blissfully, his face colored, in comparison to the appearance of a livid, frozen man covered with cold sweat and as pale as death, plunged into syncope!

Dr. Casse had attributed this condition of illness to a *serous and passive congestion of the meninges and of the brain*. I assert that this anatomical injury is difficult to reconcile with an intermittent symptom such as sleep that appears and disappears several times a day. Cerebral circulation does not lend itself at all to such sudden alternating flux and reflux, which are necessary to explain the main sign of the illness, the intermittence of sleep, whereas the idea of a spasm makes it quite easy to explain.

Can this affliction be linked to various degrees of morbid sleep which have been somewhat forgotten in our day, but which the ancients were careful to distinguish in theirs: cataphora, sopor, stupor, coma, carus, and lethargy? The form, duration, and idiotic insensitivity which characterize these last three types make the comparison impossible from the outset.

Perhaps one could associate it with cataphora, and, if one were to consider only the meaning of the Greek words ("kata," down; "pherein," to carry), one would actually believe in a certain analogy between these two types of sleep. But in cataphora, sleep, which is easily interrupted in the case of G., starts again as soon as one stops speaking to the patient. The sleep is continuous, of a certain duration, and does not include long intervals in which the subject thinks, acts, and works. Finally, cataphora would not be prolonged for years without ending in death or recovery.

As for *sopor* or drowsiness, an intermediate stage between cataphora and coma, the difference is even more marked. The patient, lying on his back, sleeps even more soundly, and cannot be awakened without great effort, and exhibits clearly defined cerebral symptoms, cephalalgia, dizziness, loss of memory, akinesia. However, our patient has no symptoms indicating a cerebral illness... and ultimately has more waking hours than sleeping.

Confusion with what the English call "sleeping dropsy," which Dr. Nicolas calls "somnosis" and Dr. Dangaix calls "hypnosis," is

impossible. First, this illness is only found in negroes in equatorial countries, and no cases of it have been observed in our temperate latitudes. However, since that is not in and of itself reason enough to exclude any analogy, let us recall the insistence with which Dr. Nicolas recently (reports from the Academy of Sciences, issue of May 10, 1880) outlined the progressive and fatal evolution of sleeping sickness from initial drowsiness to death. Sleeping sickness, he says, begins with drowsiness that is completely indistinguishable from normal drowsiness, and its progression is marked by increments that start with deep sleep, followed by longer and longer periods of sleep, until finally the patient does not wake up again. I might add that, being familiar with the work of my friend, Dr. Nicolas, I invited him to examine this patient with me, and that, as a qualified judge of such matters, he immediately rejected any idea of an analogy between these two afflictions.

(Paragraph deleted, placed in the Appendix^a).

I had thought of associating the illness with the particular form of nervous condition that was so well described by Morel under the name *emotive delirium*. I found this idea attractive for a short while. In fact, there is no disputing that G. does have a very obvious degree of emotionality, and that this emotionality provokes the attacks. But what a difference there is in the depth of their effects and the final outcome! Although it is true that the two illnesses appear in response to the slightest of causes, and even the most bizarre, it all adds up to just one effect for G., namely sleep, whereas the scene is quite complex and varied in emotive delirium, accompanied by agitation, anxiety, palpitations, and clouding of the senses, rapid pulse, exaggeration of ideas, and finally automatism. There is nothing of this sort with G. *He falls asleep without suffering*; a subject suffering from emotive delirium shakes at the slightest occasion, complains, and *suffers without falling asleep*.

We also do not believe that it can be considered incipient *ataxia* for short periods, because there are no flashes or jerky movements.

The reduction of strength, motility, and the will in G. also made me think of the *neurasthenic* form of *spinal irritation*. However, on the one hand, the back pain, the sense of fatigue, compression, or burning in the spinal column, is absent, and, on the other hand, he shows no sign whatsoever of the melancholia or hypochondria that accompany irritable weakness and make this type of patient extremely unhappy and quite given to complaining. This man is a happy, talkative individual, who lacks neither strength nor energy, and never worries, and shows fatigue in his limbs only on occasion. All the facts are in contrast to cases of neurasthenia.

Therefore, I feel justified in designating *narcolepsy* as a specific neurosis, little known until now, and it is good to draw the attention of observers to it.

Let us remember what happened with agoraphobia, which was long confused with vertigo. Once identified, many practitioners in every country throughout the world began to recognize it immediately. Perhaps the same will be true for narcolepsy, which we consider a specific neurosis, characterized by the twofold criterion of drowsiness and falling or *astasia*.

A few words of explanation regarding the cause, the role, and the need for physiological sleep will help us, I believe, explain the pathogenesis of this neurosis.

Whether cerebral function is dependent upon a substantial material, liquid or solid, supplied by the gray matter, or by a molecular movement of the fibers and ganglia of the brain, as soon as there is work or exercise, the result is wear, exhaustion, and loss,

and consequent absolute need for repair.

If cerebral activity is a direct function of the amount of oxygen absorbed by the brain and other tissues, and if that amount is greater when awake because the blood reaches the brain more quickly, then the more active this oxidation, the greater the wear on the cerebral substance, the elimination of materials, particularly phosphates, and an exhausting fatigue. This results in a need for a period of rest and calm, during which the brain expends less, and receives the elements needed to make repairs, to store them, as well as a need for frequent pauses during which oxidation and reabsorption are less active. What could better ensure and procure this period of rest, this necessary pause, this indispensable repair, than sleep?

Having said this, let us try to explain the intimate cause of narcolepsy in the case of G. I do not believe that the fall of a piece of wood on his head, by causing a congestive state that is continually renewed on the surface of the hemispheres, has caused it. This intermittent and so frequent state of congestion of the brain is not easier to explain than an intermittent commotion.

As I see it, G. is subject to the laws of two different types of sleep. Thus, like all of us, after the day's fatigue, he feels a need for rest during the first hours of the night, and from nervous habit, and his sleep is then the natural, normal physiological sleep. But during the day, things are quite different. Several times each hour, he is forced to obey a morbid, urgent, and sudden need for narcosis.

Probably, through a special idiosyncrasy, the amount of oxygen accumulated in the nerve centers is in too short supply there, or the oxygen is exhausted too rapidly under the influence of emotions that are too frequent or too strong. The cerebral wear for G. is perhaps greater than in other people, the arterial capillaries too few or too narrow. Perhaps he experiences too rapid an elimination of the regressive products, particularly phosphates.

Whatever the case may be, in this state of relative poverty, the slightest expenditure of strength, the electric influence, a storm, an emotion, constantly subtract from and exhaust his energy, his vitality. On each occasion, he is neuroparalyzed or, to put it better, neurolyzed, which results in the frequent need to sleep, sleep being the greatest and most powerful restorer of the weakened organism. This opinion is shared by Dr. Delasiauve who, early in his journal, wrote that, "exposed to rapid losses, the nervous system needs to be reimmersed in immobility and rest."

Given this explanation, borrowed from physiology, if we try to determine the exact anatomical location of this neurosis, I believe that, supported by the authority of Dr. Vulpian, we can place it in the annular protuberance. "The annular protuberance," says Dr. Vulpian (1), "must be considered the center of association for emotional movements: whether the excitement comes from the brain or from outside (and he lists several examples), in great emotional expressions, in dreams and in crying, the protuberance plays the most significant role. Under the influence of joy, happiness, sadness, distress (which is certainly the case here), or fright, a certain number or most of the active elements of the protuberance are affected and, through an associated excitation of the motor fibers, a harmony of movement breaks out which varies depending on the intensity of their affliction." What a strong argument in support of our cause! In our subject, there is incontestable over-activity of the protuberance, which enters into a spasm, exaggerating its function on the least provocation, and reacting on the other nerve centers. The result is, on the one hand, a momen-

tary paralysis of the cerebrospinal axis, a suspension of nervousity, resulting in astasia and falling and, on the other hand, momentary anemia which, in turn, causes sleep. These two results that constitute narcolepsy are immediate because, in G., there is some sort of shattering of the annular protuberance and cerebral stun.

To complete this observation, I must say something about the treatment that I employed.

Initially, given the appearances and the diagnosis presented to me, and acknowledging the effect of the emotions on the reappearance of these sleep attacks, believing that the spasm of the vessels could cause cerebral anemia common to sleep and to epilepsy, I used picrotoxin, which has the characteristic of preventing the vessels' spasmodic contraction by keeping them in a relaxed state, and I added various bromides to reduce irritability and the reflex action of the cerebrospinal axis.

I must admit that I did not achieve any positive results by using this medication. On the contrary, my patient lost strength and had an increased tendency to sleep. I abandoned that approach.

Along the same lines, I advised that he inhale amyl nitrite vapors poured onto a handkerchief as soon as the narcoleptic attack began. In fact, the amyl nitrite made the intracerebral circulation and the visceral circulation more active, and it expanded the vessels. We did not overlook the fact that G.'s pulse fell even further, clearly causing an intracranial void, a whirlwind blowing in his head. The use of this medication thus seemed to be indicated. It appeared to be successful for several days, and the subject blushed when inhaling it. But its use did not prevent the attacks, and we then abandoned it, convinced that cerebral anemia played no role in the neurosis at hand.

Then I used subcutaneous injections of apomorphine, which are extolled in Germany in cases of convulsive neuroses, initially in very moderate doses, then up to levels causing nausea, without obtaining any positive results.

Then, I decided to turn the symptoms into a medicine, i.e. directly fighting the drowsiness. I placed a seton directly on the nape of the neck, which I maintained, and I prescribed grains of caffeine and caffeine valerianate. He improved slightly, but, being eager for more pronounced results, I was perhaps mistaken in abandoning this medication to consider another idea.

I used strychnine arsenate in progressive doses, and I did not stop until the patient felt tremors in his limbs. I hoped that using this power agent, I would increase the general tone of the economy, fighting the collapses and constant neurolytic exhaustion. At the same time, I had him take phosphates, very tonic food, and warm showers that were revulsant on the spinal column. I even used hypodermic injections of curare. In sum, I did my best to treat the patient aggressively. Nevertheless, I must admit in all humility that by using these methods I barely managed to obtain a few hours of rest and constant work without sleep in the morning and evening. As we both acknowledged that these successes were not in keeping with our mutual efforts, we lost contact, leaving to time and to nature the care of healing or improving this painful neurosis.

Is the ineffectiveness of these remedies one of the characteristics of this neurosis? (remainder of paragraph deleted, and placed in the Appendix^b).

It is clear from what we have said above that the treatment of narcolepsy is entirely open to study. This is one more point of similarity that it shares with the other neuroses, which are so often the stumbling block of our therapeutic means. Whatever the case

may be, I am glad to have been able to present this initial study to my colleagues. I am sure that it will result in further studies, for I have already received from a doctor in Lyon all the elements of a third observation of narcolepsy, which I propose to publish somewhat later.

APPENDIX

- a) Moreover, neither Dr. Casse nor Dr. Semelaigne concluded that there was an analogy between the illness of their subject, M., and the sleeping sickness among negro populations. Dr. Semelaigne added, amusingly, "For the time being at least, let us leave sleeping sickness to the negroes. Whites have quite enough other illnesses without that one."
- b) In treating M., Dr. Casse used tea, coffee, quinine sulfate, ferruginous agents, purgatives, and Seine baths with running water. A vesicant was applied to the nape of the neck, all to no avail. When the symptoms were even worsened by digestive function problems, heaviness of the head, and more difficult locomotion, our colleague advised the waters at Brides. The highly ozonized mountain air, the action of the waters internally and externally, stimulated his appetite and strength, and the skin regained a livelier color. Finally, after a season followed by a trip to Switzerland, M. returned to Paris much improved, but not entirely cured.
- c) The translator of Gélineau's report made an additional comment that "The French words 'voiture' and 'camion' translate as 'carriage' or 'automobile' and 'wagon' or 'truck', respectively, depending on the historical period. Since this article was written at a time when primitive diesel vehicles may well have been coursing the streets of Paris, i.e., at a time when 'voiture' could refer either to horse-drawn or horseless carriages, we have used 'carriage' and 'wagon' to translate these two words, but the reader should be aware that the author might well have had in mind 'automobile' and 'truck.'"