

From Dreams to Hallucinations: Jean Lhermitte's Contribution to the Study of Peduncular Hallucinoses and the Dissociation of States

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Jean Lhermitte (1877–1959) was one of the pioneers of behavioral neurology, including the field of hallucinations. This article focuses on his work concerning the relationship between hallucinations, sleep, and dreams. From 1910, Lhermitte became interested in sleep and its disorders, particularly narcolepsy and its accompanying symptoms. He also reported on sleep disorders and hallucinations occurring in people with lesions of the diencephalic region ("infundibular syndrome"), and later encephalitis lethargica. In 1922, he described a syndrome of complex, predominantly visual hallucinations in patients with vascular damage to the midbrain, known as peduncular hallucinosis. Twelve historical cases of peduncular hallucinosis, including 10 from Lhermitte, are reviewed.

He gave a precise phenomenological description of peduncular hallucinosis, and put forward the hypothesis that the lesion disrupted the anatomy and connections of a center regulating wakefulness and sleep, thus enabling a dissociation of the mechanisms of dream and waking states. Although the pathophysiology of peduncular hallucinosis remains to this day partly obscure, the model of a limited subcortical lesion acting through complex mechanisms and ultimately involving the cortex remains valid. Lhermitte was also a pioneer in characterizing what contemporary sleep specialists call dissociation of states.

J Neuropsychiatry Clin Neurosci 2022; 34:16–29;
doi: 10.1176/appi.neuropsych.20120314

Jean Lhermitte (1877–1959), a renowned neurologist and a psychiatrist from the Paris school, was one of the pioneers of behavioral neurology and of the neuropsychiatric interface (1, 2). An important part of his work was devoted to hallucinations, about which he published an insightful book in 1951 (3). Here, the focus is specifically on his works concerning hallucinations in their relation to dreaming and sleep. Lhermitte had been interested in sleep and its disorders, particularly narcolepsy and its accompanying symptoms (4, 5), since the 1910s. In 1922, he was the first author to describe a syndrome of complex hallucinations following vascular damage to the midbrain (6), which is still known as peduncular hallucinosis. From this seminal observation, Lhermitte put forward the hypothesis of a dissociation between components of sleep. Subsequently, he published other peduncular hallucinosis cases and remained constant in his interpretation all his life, disputing with some of his contemporaries. In spite of its limitations linked to the knowledge and means of the time, his work remains of great interest for the accuracy of its clinical descriptions and for his pathophysiological hypotheses, which were in many ways innovative. The books written by Lhermitte are well known, but his numerous articles are scattered over many publications. I systematically searched for all articles having

Lhermitte as author or coauthor between 1910 and 1955, in *Revue Neurologique (Paris)* and other medical French journals available online, mainly on the websites of the Bibliothèque Interuniversitaire Santé de Paris (biusante.parisdescartes.fr/histoire/medica/periodiques.php), the Bibliothèque Nationale de France (data.bnf.fr and gallica.bnf.fr), and the Bibliothèque Henri Ey of the Centre Hospitalier Saint-Anne (ghu-paris.fr/fr/bibliotheque-henri-ey). Other articles written by Lhermitte or contemporary authors that he cited in his work were also collected.

BEFORE PEDUNCULAR HALLUCINOSIS: INFUNDIBULAR SYNDROME AND ENCEPHALITIS LETHARGICA

Several observations paved the way for the description of peduncular hallucinosis: the so-called infundibular syndrome, and the hallucinatory phenomena reported by some patients following encephalitis lethargica.

Infundibular Syndrome

In 1917, Henri Claude and Jean Lhermitte (7) published the observation of a 25-year-old man, a submarine mechanic, admitted in 1916 to a military neurology center in Bourges

(a city in central France) for deterioration of his general state, insomnia, polyuria with polydipsia, and bitemporal hemianopia. He had a history of syphilitic chancre. Examination of cerebral spinal fluid (CSF) showed unquantified lymphocytosis and albumin at 0.56 g/L. The initial diagnosis was syphilitic meningitis. After clinical fluctuations, “the subject falls into a deep sleep from which it is impossible to wake him. This ‘narcolepsy’ attack, which lasts about five hours, leaves the subject amnesic and astonished when he wakes up.” Thereafter, his state worsened: “The patient is overcome by a confusional delirium in an oneiroid state [“onirism”]. He says his bed is wet from the rain and fog of the sea; he thinks he is in Toulon [a naval base in the south of France] in springtime.” In the following weeks, the patient had new “attacks of narcolepsy,” and he died 6 months after onset, probably from coincidental pulmonary tuberculosis. The autopsy revealed a retrochiasmatic tumor occupying the interpeduncular fossa and compressing the neighboring structures. The tumor thinned the floor of the third ventricle (in particular, the infundibulum and the lamina terminalis), without reaching the pituitary gland. The histological description suggests a craniopharyngioma (Professor Jacques Poirier, personal communication). Based on this observation, the authors described an “infundibular syndrome.” They observed that, “if one does not confuse them with coma, somnolence and sleep, authentic narcoleptic attacks are not exceptional as manifestations of infundibular tumors.” In his 1910 reports (4, 5), Lhermitte had developed a broader conception of narcolepsy as a syndrome with various causes, particularly tumors, alongside idiopathic or essential forms, as described by Jean-Baptiste Gélineau in 1881 (8), which Lhermitte considered rarer.

Encephalitis Lethargica

Lhermitte and de Saint-Martin described two cases of encephalitis lethargica in 1918 under the name of “primitive poliomesocephalitis with narcolepsy,” stressing the sleep disorder that gave the condition its name (9). In the review he later published, Lhermitte (10) noticed that daytime sleepiness was sometimes associated with insomnia and “a certain degree of agitation,” the latter being “associated with a mild and quiet delirium of a confusional and hallucinatory nature,” which the author attributed to “the close relationship between the dreaming associated with confusion and physiological dreaming. . . . In both cases, it is the same sequence of images that projects its disturbing and capricious phantasmagoria onto the sleeping consciousness, the same lack of insight, the same inconsistency, and the same lack of astonishment at the appearance of the most unexpected and disconcerting phenomena.” These images appeared in the evening, were almost exclusively visual, and could, through a series of intermediaries, lead to an acute delusional syndrome. Lhermitte aligned himself with an opinion previously defended in France by Jean Camus (1872–1924), according to which “lesions of the midbrain and the deep gray-matter nuclei can lead to mental

disorders independently of cortical lesions,” suggesting the existence of extracortical regulatory centers of the psychic functions (11).

PEDUNCULAR HALLUCINOSIS: THE SEMINAL 1922 ARTICLE AND FOLLOWING OBSERVATIONS

Recent articles have summarized the history of peduncular hallucinosis (12–14). My aim is to analyze more precisely the emergence of this concept in the context of Lhermitte’s time, and then, in the next sections, to integrate peduncular hallucinosis into Lhermitte’s more general theory on the relationship between dreams and hallucinations, and to compare it with contemporary data.

The first observation of peduncular hallucinosis was reported by Lhermitte in 1922 to the Neurological Society of Paris under the title “Syndrome de la calotte du pédoncule cérébral. Les troubles psycho-sensoriels dans les lésions du mésencéphale” (“Syndrome of the peduncular cap [tegmentum]). Psycho-sensory disorders following mesencephalic lesions”) (6). The patient was a 75-year-old woman. She initially had headache and vomiting, and within 2 weeks developed left paralysis of the abducens nerve, then of the oculomotor nerve, and on the right side facial paralysis, a Babinski sign, and kinetic cerebellar syndrome. CSF cell count and albumin concentration were normal. The picture mostly corresponded to Claude’s syndrome (15) with additional pyramidal signs, and suggested involvement of the midbrain tegmentum. Damage to the left abducens nerve, without lateral gaze palsy, is unusual in this setting and could have been due to compression by a vertebrobasilar dolichoectasia (16). Two weeks after completion of the somatic neurological disorders, as depicted by Lhermitte, “the patient spontaneously told us that during the day and especially at nightfall, she sees various animals walking around on the floor of the ward. There are cats, and hens slightly strange in appearance, their dilated pupils possessing a strange luster. In order to verify the reality of these perceptions, the patient tried to touch these animals. She told us that her contact with them resembles that of real animals, but as soon as she touched them, they slowly disappeared through the floor.” The patient “does not think that these are true perceptions, since, when questioned, none of the patients in the same hospital room had experienced them.” In addition, “sleep seems to be strongly disturbed and insomnia at night is associated with some sleepiness during the afternoon.” Three weeks after they began, “the visions are no longer of animals, but of human beings decked out in bizarre and tattered outfits, or children playing with dolls. The patient sees them in the beds of her neighbors.” Finally, insight weakens: “These images are so vivid, she tells us, that they can only correspond to reality,” but “no delusional ideas accompany these visual hallucinations.” In his comments Lhermitte, referring again to Camus (11), stated that “it was not extravagant” to wonder if the mental disorders were related to the peduncular

lesion. He insisted on the “striking similarity” between the almost exclusively visual sensory phenomena experienced by the patient and dreams, leading to the statement that hallucinosis was the expression of a dream in a half-asleep subject. Finally, based on his own previous work (7) and that of other authors, Lhermitte concluded that the visual hallucinosis resulted from a “disturbance of the sleep function,” a condition that was “the equivalent of narcolepsy” and was related to the mesencephalic lesion.

In 1924, Ludo van Bogaert (1897–1989), the renowned neurologist and pathologist from Antwerp, Belgium, made the first clinico-anatomical observation of what he was the first to call “hallucinosé pédonculaire” (peduncular hallucinosis) (17). The patient, a woman aged 59, had a neurological picture close to Claude’s syndrome, with visual hallucinations, mainly zooptic: “From the very first evening, she saw a dog’s head on her pillow, and the opposite wall frequently bore a picture of a horse on a pink background. . . . On one occasion, she reported the presence of green snakes in her bed for a whole day. She touched them, pushing them away from the bed. They were rough against her hand. . . . None of these hallucinations provoked an emotional reaction in the patient; she shows no surprise . . . and is absolutely convinced of the reality of the animals she sees.” There were also dyschromatopsia and episodes of psychomotor agitation, independent of the hallucinations. A few years later, van Bogaert published additional clinical and autopsy data (18). During the 14 months of follow-up, the patient still experienced “hallucinatory outbursts.” The lesion was “a focal peduncular softening by a syphilitic arteritis of the retromamillary pedicle,” extending to the superior colliculus and the pulvinar. This patient, unlike Lhermitte’s, did not have sleep disorders, leading van Bogaert to argue that “the release of the imaginative automatism can be dissociated from the hypnic state.” He thus diverged from Lhermitte’s interpretation and, referring to the conceptions of the psychiatrist and philosopher Pierre Quercy (19), he stated that “the essential disorder is a momentary weakening of the self which disrupts the function of reality.”

However, for his entire life, Lhermitte remained convinced that peduncular hallucinosis made it possible to approach “the physiological basis of the dream” (20). Following his 1922 article, he published nine other cases of peduncular hallucinosis or similar syndromes that strengthened his conviction. They are summarized in Table 1, together with the first observation and two other historical cases. The next observation (21) was that of a 70-year-old woman who suddenly fell into a “deep sleep,” with bilateral ophthalmoplegia suggestive of bilateral damage to the oculomotor nerves, bilateral pyramidal syndrome, and cerebellar syndrome. When the patient came out of her “lethargy,” she had in the evening “multiple, colorful visions” of animals or people “moving silently.” Sometimes “she thinks she is at the theatre, attending a variety of performances.” These phenomena did not evoke an emotional response. They were associated with memory impairment for recent events and

confabulations. The picture retrospectively suggests a bilateral paramedian mesencephalic and thalamic infarction (22).

A new observation, published with Gabrielle Lévy (1886–1935), differed from the others (23). A 60-year-old man, suffering from long-standing tabes dorsalis, went into a coma after a suicide attempt with hypnotics. On waking, he had a crossed syndrome suggestive of mesencephalic damage (Table 1). For a month, at nightfall, the subject experienced visual hallucinations, which were at first dreamlike: “He thought his room had been transformed into a railway or subway carriage.” Soon after, “since this carriage was in more or less close connection with an aeroplane service . . . he arrived on a platform overlooking a marvelous landscape resembling that of Arabia. A plane descended from the sky and landed on the platform. He climbed aboard. . . . Then he flew over wonderful landscapes for a few moments.” After a few days, the hallucinatory phenomena changed in nature. In the evening, “the walls of the room, the various objects that furnished it, come to life under the astonished gaze of the patient. Two pairs of trousers hung in front of him and a coat rack in the room become two bustling women.” Even the bare wall of his room became animated and lively: “Workers come to nail down tapestries, a modern kind of tapestry, but this tapestry becomes animated in its turn. Characters move around, curtsy to each other, make themselves understood.” The images are colorful and silent, they “are not unpleasant,” and the subject “realizes that he is the plaything of an illusion.” About 3 weeks after its onset, the hallucinosis decreased, with a few visions remaining before sleep: “heads that look at him strangely” on a wall outside. After 1 month, the hallucinatory phenomena subsided. The patient died of a pneumopathy, and a new article provided anatomical findings (24). There were no macroscopic lesions, while histological examination showed “alterations of the peduncular cap (midbrain tegmentum) with metachromatic degeneration associated with an alteration of the ventral nucleus of the third nerve and of the median and intermediate nuclei.” Without excessive conviction, the authors linked these lesions with the drug intoxication. For Professor Françoise Gray (personal communication), the description is compatible with an ischemic lesion dating back several weeks, and leads to the hypothesis of low cerebral blood flow at the time of the intoxication with hypnotics, possibly in arteries with stenotic lesions (atheroma? syphilitic arteritis?).

In 1931, Lhermitte and Gabrielle Lévy (25) published the observation of an elderly patient who 5 years earlier (26) had suffered a “slight stroke” followed by a “pronation spasm of the right arm” (probably dystonia) and sensory disturbances in this limb. Since this accident, the patient “very frequently [saw], at night, a man’s head appear on the wall facing her bed.” The head was “black and grey on a white background . . . the figure [was] very recognizable.” To make it disappear, the patient focused on another object. The vision was rather pleasant, and insight was retained. The patient also suffered from nightmares with screaming.

TABLE 1. Cases of peduncular hallucinosis published by Jean Lhermitte and his colleagues and by contemporary investigators referring to Lhermitte's work (in chronological order)^a

Study	Sex	Age at onset (years)	Neurological signs	Hallucinations	Insight and emotional reactions	Sleep disorders	Time to onset after stroke, schedule, and duration	Presumed topography	Anatomical lesions (autopsy)	Presumed cause
Lhermitte (6)	Female	75	Left: VI, extrinsic III Right: cFP, Babinski sign, and cerebellar syndrome (limbs)	Visual: animals, people Tactile	Yes, later no No	Insomnia, Daytime somnolence	Two weeks Evening ND (>10 days)	Midbrain	NA	Ischemic
Lhermitte and Toupet (21); Lhermitte (40)	Female	70	Bilateral: ophthalmoplegia (at least both III), Babinski sign, and cerebellar syndrome Memory impairment and confabulations	Visual: animals and people	No No	Coma ("lethargy") and later daytime somnolence	One month (when coma resolved) Evening NA	Midbrain (probably thalamic, see article text)	NA	Ischemic
Van Bogaert (17, 18)	Female	59	Right: III Left: III initial, cFP, cerebellar syndrome (limbs), and Babinski sign Vertigo (initially)	Visual: animals Tactile Visual illusions (changed colors)	No No	No (episodes of agitation in the evening)	First day Evening 14 months (until death)	Midbrain: "inferior red nucleus syndrome"	Right: red nucleus, pulvinar, superior colliculus, fibers III, decussation of superior cerebellar peduncles, substantia nigra, periaqueductal gray	Ischemic and syphilitic arteritis
Lhermitte and Levy (23); Lhermitte et al. (24)	Male	60	Intoxication with hypnotics, coma, then: dysarthria, right III palsy; bilateral AIM, and later left upper-limb "choreo-athetotic" movements	Dream-like (oneiroid) visual: complex scenes Later visual: people Visual illusions	Yes No	ND	Probably several days Evening One month	Midbrain	Tegmentum and nuclei of III, bilateral predominating on the right side	"Toxic" (probably ischemic)
Lhermitte and Levy (25)	Female	ND	"Pronation spasm" (presumably dystonia) of the right upper limb	Visual: human head	Yes No	Vivid dreams and shouting while dreaming	Soon after (ND) Evening Five years	Subthalamic (probably thalamic, see article text)	NA	Ischemic

continued

TABLE 1 continued

Study	Sex	Age at onset (years)	Neurological signs	Hallucinations	Insight and emotional reactions	Sleep disorders	Time to onset after stroke, schedule, and duration	Presumed topography	Anatomical lesions (autopsy)	Presumed cause
Lhermitte and Levy (29)	Female	42	Transient right hemiplegia 1 year before Loss of consciousness Left: hemiplegia with partial recovery, Babinski sign, AIM of the lower limb, and thermo-algesic sensory loss	Visual illusions: a man's red head, moving Visual: skulls	Yes Moderate (fear)	ND	Three weeks Evening About 2 months	Midbrain	NA	Ischemic, syphilitic arteritis
de Morsier (30)	Female	51	Dysarthria, ataxia Left: cerebellar signs, AIM of the hand, and astereognosia Right: mild ptosis Visual field and visual acuity: normal	Visual: people, animals (bright), once a scene (people on an avenue) "Like a movie" In the left hemifield only	Yes No, or mild (pleasure)	Occasional insomnia	Twenty months Evening At least 1 year	Midbrain (and thalamic)	NA	Vascular
Lhermitte et al. (37)	Male	33	Right: hemiparesis, VII, VI and X, and myoclonus of the shoulder Left: hemianesthesia (pain, temperature) Dysarthria and dysphagia	Visual: people	Yes Moderate	ND	Two weeks Evening and night "Very ephemeral"	Medulla and pons	NA	Hemorrhage
Lhermitte and Bineau (34)	Female	71	"Obnubilation," "lethargy," bilateral ptosis, limitation of lateral gaze, and diplopia Left: hemiparesis, Babinski sign, and dysmetria	Visual: animals, people, and swastikas Visual illusions (changed colors)	Yes Moderate (fear)	Daytime somnolence	Five days ND Ten days	Midbrain	NA	Ischemic

continued

TABLE 1 continued

Study	Sex	Age at onset (years)	Neurological signs	Hallucinations	Insight and emotional reactions	Sleep disorders	Time to onset after stroke, schedule, and duration	Presumed topography	Anatomical lesions (autopsy)	Presumed cause
	Female	71	Loss of consciousness, balance disorder, somnolence, and bilateral dysmetria	Visual: animals Illusion (changed colors)	ND ND	Daytime somnolence	Within days ND "Several months"	Midbrain	NA	Ischemic
Lhermitte (35)	Female	71	Progressive left hemiparesis, diplopia; 2 years later, brisk AIM of left upper limb, later of lower limb, small steps, left Babinski sign, lost pupillary reflexes, anisocoria, convergent strabismus, and upward gaze palsy	Visual: people, animals Auditory verbal (concomitant with visual hallucinations of persons)	Delayed Mild	No	About 35 years Five episodes at night, one in daytime Several months syndrome of the red nucleus"	Midbrain and subthalamic area: "contralateral red nucleus"	NA	Tuberculoma
Lhermitte (36)	Male	66	Left hemiplegia and sensory loss (abating); bilateral Babinski sign; ophthalmoplegia, including palsy of vertical gaze and skew deviation; right Horner's syndrome; and later AIM of left upper limb	Auditory: ringtone Visual: objects, people Nocturnal dream-like scenes (or possibly dreams)	ND ND	Daytime somnolence	One day ND and night At least 2 weeks	Midbrain	NA	Hemorrhage (xanthochromic CSF)

^aCases with presumed lethargic encephalitis are not included. AIM=abnormal involuntary movements; cFP=central facial nerve palsy; CSF=cerebral spinal fluid; NA=not available; ND=not disclosed; III, VI, VII, X=corresponding cranial nerve palsies.

Lhermitte believed that the lesion was located in the subthalamic area and related this observation to those previously published of peduncular hallucinosis. The lesion more likely involved the posterolateral region of the thalamus (27, 28). The same authors published a similar case soon after (29).

An observation by Georges de Morsier (1894–1982), a Swiss neuropsychiatrist, generated new discussions (30). A 54-year-old woman suddenly developed Claude's syndrome; 18 months later, she reported predominantly evening visions of colorful figures or animals, appearing for a few seconds. The images unfold "like in the cinema." They "always come from the left and move from left to right until they reach the median line where they disappear." The visions did not give an impression of reality and did not cause anxiety. They occurred irregularly and could stop for several weeks. Sleep was mostly undisturbed. Although visual fields and visual acuity were normal, de Morsier postulated the existence of a unilateral, retrochiasmatic lesion of the visual pathways. He did not propose any mechanism to explain these hallucinations limited to a visual hemifield, in the absence of detectable damage to the visual field, unlike the hallucinations in a hemianopic field after an ischemic stroke first described in the United States by Seguin in 1886 (31) and in France by Lamy in 1895 (32). No anatomical study was available in this case. Lhermitte stated, at the end of this presentation, that lesions of the optic tract or lateral geniculate bodies never cause hallucinations, which is not certain, even if observations are rare (33). More generally, Lhermitte constantly rejected the hypothesis that an alteration of the visual pathways could contribute to peduncular hallucinosis.

Subsequently, Lhermitte and his collaborators published four more cases of peduncular hallucinosis (34–36), one of them secondary to a peduncular hemorrhage (36), and one observation of hallucinations following a vascular lesion situated in the pons and medulla (37) (Table 1). In addition, five cases presumed to be due to hemorrhage of the peduncular tegmentum were summarized in a review article (38).

PEDUNCULAR HALLUCINOSIS AS A "DISSOCIATION OF THE HYPNIC STATE"

In 1932, Lhermitte summarized his conceptions of peduncular hallucinosis in an article published in *L'Encéphale* (39). He gave a definition of hallucinosis, in line with that proposed by his contemporaries: "hallucinatory states which do not lead to true delusions"; and of peduncular hallucinosis: "hallucinatory manifestations which appear and develop in patients suffering from lesions limited to the mesodiencephalon, i.e., to the ventral region of the third ventricle and the peduncular cap [tegmentum]." Lhermitte underlined the common features of his cases: "visions of mobile, colorful, and silent animals or animated characters," occurring in the evening, in the absence of surprise and anxiety, without delusions. Insight, on the other hand, differed from one patient to another; patients might "become caught up in the

hallucination when it occurs with crudeness, vividness and naturalness." Hallucinosis was associated with sleep disturbances. Lhermitte's views on pathogenesis can be summarized as follows: There is a regulating center for wakefulness and sleep in the mesodiencephalon; sleep has a negative feature, the "suspension of consciousness," and a positive feature, dreaming; lesions causing sleep alteration can also modify dream activity; and in peduncular hallucinosis observations, there are sleep disorders and visions with "the same attributes as those of dreaming" and "obvious similarities with hypnagogic images." Lhermitte concluded that a patient with peduncular hallucinosis "is therefore a dreamer awake or insufficiently asleep, a subject whose profoundly disturbed hypnic function has been dissociated by the whim of an anatomical disorganization." A dissociation of dream mechanisms, however, was not sufficient to explain peduncular hallucinosis. Lhermitte attempted to reconcile his conception with that of van Bogaert, according to whom hallucination was due to "a weakening of the sense of reality . . . causing images and representations to take on an abnormal brilliance." For Lhermitte, "to sleep and to dream is to slacken one's attention to reality . . . and to scatter before a drowsy consciousness the images and representations that constitute the capricious and incessantly moving weft of the dream" (23).

In a 1934 review (40), Lhermitte added a few recent cases, including that of Garcin and Renard (41) associating multiple cranial nerve damage, sleep disorders, and visual hallucinations, all transient phenomena attributed to a viral infection affecting the brainstem. The message was that, whatever the cause (vascular, toxic, infectious), the mesodiencephalic region was altered and responsible for the hallucinosis. However, one observation contradicted this assertion (37): A 33-year-old man was suddenly affected by a crossed syndrome (Table 1) presumed to be hemorrhagic and suggestive of a lesion located in the medulla and the pons. Besides somatic neurological symptoms, the patient had, on the one hand, the impression that his legs occupied a position far above the bed plane, and, on the other, visual evening and night hallucinations, in the form of characters moving silently. The first symptom was a postural illusion, similar to phantom limbs, probably related to sensory deafferentation, a symptom rarely reported following brainstem lesions (42). Besides, the hallucinosis suggested that a lesion of the medulla or the pons could generate a syndrome similar to peduncular hallucinosis, as already reported by Stenvers (43) in a patient with a pons tuberculoma who had, in addition to focal signs, visual and auditory hallucinations. Lhermitte, reluctant to admit that a pontine lesion could induce a peduncular hallucinosis, evoked a "disruption in the equilibrium of the organo-vegetative system," referring to Raoul Mourgue, author of an "organo-vegetative" theory of hallucinations that hardly survived its author (44). In a new review, Lhermitte (45) commented on the dreamlike and hypnagogic phenomena of narcolepsy and the hallucinations associated with sleep paralysis. All

these phenomena had in common that they were the positive side of a hypnic function disorder. In his 1951 book (3), Lhermitte devoted 20 pages to “hallucinosis of peduncular origin.” He added some new brief observations, which widened the phenomenological picture: ocular paralysis could be missing; auditory hallucinations, including verbal ones, could occur; and lesions could involve the floor of the third ventricle or the pons. Lhermitte remained true to the concept of a dissociation of the hypnic state and specified that the causal focal lesion only generated hallucinations because it “activated extremely complicated mechanisms, which took place throughout the entire extent of the brain, and especially in the cerebral cortex.” Lhermitte thus refuted the objections of Mourgue, who reproached him for confusing “the anatomical localization with that of the functional syndrome” (44).

PEDUNCULAR HALLUCINOSIS: THE LEGACY OF JEAN LHERMITTE

The aim of this section is to show, through four questions raised by the concept of peduncular hallucinosis, to what extent Lhermitte’s approach, in spite of the limited anatomical and physiological knowledge of the time, was innovative. The detailed history of peduncular hallucinosis from the 1950s to the present time will not be systematically reviewed.

Is Peduncular Hallucinosis a Clinico-Anatomical Clinical Entity?

For Lhermitte, the core features of peduncular hallucinosis were visual hallucinations without delusions, a predominance in the evening, sleep disorders, and an anatomical lesion located in the mesodiencephalic area. However, Lhermitte acknowledged that the syndrome was heterogeneous. Besides visual hallucinations, visual illusions and auditory (including verbal) and tactile hallucinations were reported. Insight into the hallucinatory nature of the experience and the emotional impact varied from one patient to another, or even in the same patient. In most cases, visual hallucinations consisted of people, heads, or animals projected onto an unchanged environment. The patient passively attended the scene (e.g., “she thinks she’s at the theatre attending various performances”) (40). In at least one case (23), the hallucinations were dreamlike, fitting the definitions of onirism (oneiroid syndrome). The patient was acting in a complex and bizarre scenario, moving in an unreal setting along with the hallucinated people. Sleep disorders (e.g., insomnia, daytime sleepiness, or lethargy) were part of the core peduncular hallucinosis syndrome, although van Bogaert (17, 18) had shown that these features could be missing.

Anatomically, Lhermitte postulated that the lesion disrupted a regulatory sleep center located in the midbrain tegmentum or the third ventricle floor, possibly in the pons, but he repeatedly ruled out the possibility of the lesion being localized in the lateral geniculate nucleus or the

thalamus. However, the patient reported by van Bogaert did have a thalamic lesion (of the pulvinar) associated with those of the midbrain. Interestingly, Lhermitte and Julian de Ajuriaguerra, in an article devoted to hallucinations associated with lesions of the visual pathways (46), reported on patients with ophthalmopathy and visual hallucinations resembling those of peduncular hallucinosis in whom a neuropathological study revealed a thalamic lesion. In Lhermitte’s time, anatomical data were rare, and thalamic lesions could be missed. The possible involvement of thalamic nuclei is now recognized. Galetta et al. (12) collected data on 86 patients with peduncular hallucinosis, among whom 69 had a computerized tomography or MRI brain scan; of these 69 patients, 21 (30%) had a thalamic lesion, isolated or associated with other lesions. In another series of 23 published cases of peduncular hallucinosis, selected on stringent criteria including the exclusion of coexisting lesions of the cortex or visual pathways, Boes et al. (47) found that 16 patients (70%) had a thalamic lesion. It must also be emphasized that peduncular hallucinosis is a rare condition. Peduncular hallucinosis was not recorded in a series of 22 infarcts limited to the midbrain (48), and it was not mentioned (although it is not stated whether it was systematically looked for) in several series totaling 102 patients of infarcts of the midbrain, either isolated (49, 50) or associated with other lesions (51).

In summary, it currently seems relevant to apply the name of peduncular hallucinosis to hallucinations predominating in the visual modality and associated with a demonstrated lesion located in the midbrain, pons, or thalamus, the last of these being commonly involved.

Is Peduncular Hallucinosis a “Waking Dream”?

The first way to address this question is to compare the phenomenological characteristics of dreams and peduncular hallucinosis. Although dreams can occur in all stages of sleep, “it is clear that rapid eye movement (REM) sleep represents the sleep state most conducive to sensorily vivid, motoric and emotionally salient dreams with the most elaborated narrative structure” (52). Lhermitte was not aware of the existence of REM sleep. REMs in sleep were described in 1953 (53), but REM sleep as a specific sleep stage was characterized by Michel Jouvet under the name “paradoxical sleep” after Lhermitte’s death, in the 1960s (54). In his descriptions of dreams, Lhermitte highlighted the features of REM-associated dreams (20, 55).

Hallucinations associated with neurological conditions, including peduncular hallucinosis, are usually quite different from dreams (Table 2). In most cases of complex visual hallucinations of neurological or ophthalmological origin, the hallucinated features, most often people or animals, are projected onto an unchanged environment and do not result in a complex and bizarre scenario (56). There are, however, hallucinatory states close to dreaming within the framework of what Emmanuel Régis (1855–1918), a Bordeaux psychiatrist, called *délire de rêve* or *délire onirique* (oneiroid

TABLE 2. Comparison of the phenomenological features of peduncular hallucinosis, dreams in rapid eye movement (REM) sleep, and hallucinations in primary narcolepsy^a

Characteristics	Peduncular hallucinosis	Dreams in REM sleep	Hallucinations in narcolepsy
Sensory modalities	Visual (always), unfrequently auditory, and tactile	Visual (always), commonly auditory, somatic	Visual (most cases), commonly auditory, and somatic
Perceptual range	Elements in a scene	Immersive and panoramic scenes	Elements in a scene
Superimposed on veridical perceptions	Yes	No	Yes
Visual illusions or feeling of presence	Possible (illusions)	No	Possible (illusions and feeling of presence)
Case subject is an actor in the scene	No	Yes	Yes
Elaborated and bizarre scenario	No	Yes	No
Emotional content	No (rarely yes)	Yes	Variable
Content linked to memory	No	Yes, frequently	No
Content recalled	Yes	Poorly	Yes
Insight	Yes, no, or varies according to time	No	Yes, in most cases (during experience or delayed)
False belief (i.e., role of an external agent)	No	No	Seldom
Relationship with sleep	Yes: occurs in the evening, association with daytime somnolence (according to Jean Lhermitte)	Yes: association with REM	Yes: association with sleep onset and/or offset
Somatic changes during perceptual experience	No	Yes: atonia, REM	Possible association with sleep paralysis

^aData on peduncular hallucinosis were derived from the historical cases presented in Table 1; data on dreams were derived from Waters et al. (70), Hobson et al. (92), and Nir and Tononi (93); and data on hallucinations in narcolepsy were derived from Waters et al. (70), Fortuyn (94), Leu-Semenescu et al. (95), and D'Agostino and Limosani (96). REM=rapid eye movement.

delirium) (57, 58). For Régis, this state fell within the category of mental confusion (i.e., delirium) and was associated with intoxications or infections. The author emphasized the similarities with dream images, as well as the nocturnal predominance and the association with sleep or daytime sleepiness: "They are active, moving sleepers" (58). This gray zone between hallucinations and dreams had already been emphasized by Charles Lasègue in 1881 in the context of delirium tremens in an article with a self-explanatory title: "Alcoholic delirium is not a delirium but a dream" (59). The term *onirism* (*oneiroid state*), specific to French nosography, later freed itself from its original link with mental confusion, with, for example, Raoul Benon: "Onirism is characterized by the development in the waking state of illusions and hallucinations which resemble those observed in the normal dream, and which are not accompanied, in pure cases, by delirium or mental confusion" (60). Among the historical cases of peduncular hallucinosis, the term *onirism* only applies to the patient described by Lhermitte and Lévy (23, 24); more precisely, to the phenomena immediately following his awakening from intoxication, before the appearance of hallucinations of a completely different type. In this case, the two types of hallucinations probably were of different origin: dream-like hallucinations of toxic origin first, then typical peduncular hallucinosis hallucinations secondary to a focal lesion.

Clearly, then, peduncular hallucinosis is phenomenologically distinct from dreaming, a fact that Lhermitte, probably

influenced by his pathophysiological conceptions, was reluctant to accept. He constantly insisted on the similarities between dreams and peduncular hallucinosis, from his first review paper (39) ("Hallucinatory images [of peduncular hallucinosis] possess the same attributes as those of dreams") until his last article on this topic (61) ("This type of hallucinosis corresponds to a real waking dream"). Even in the posthumous eighth edition of his book about dreams (55), Lhermitte wrote that the images of peduncular hallucinosis "only differ from the images of the physiological dream in that the former are internalized, while the latter are externalized." At most, Lhermitte referred to "dream fragments," (34) "dreamlike fragmentation" (45), or images "similar" to those of dreams (3).

The relationship between peduncular hallucinosis and the hallucinations accompanying states of transition between wakefulness and sleep is a more difficult topic. These phenomena were described by Jules Baillarger in 1845 (62), then by Alfred Maury in 1848, who defined them as *hypnagogic* (when falling asleep) or *hypnopompic* (when waking up) (63). The very nature of the sensory experience remains debated; notably, the editors of DSM-5 dismissed these phenomena from the realm of hallucinations (64). These phenomena, which for convenience I will call hallucinations, are very common in the general population (about 25%) and even more frequent in younger subjects (65). In primary narcolepsy, these hallucinations are more frequent, more vivid, and have greater emotional repercussions than

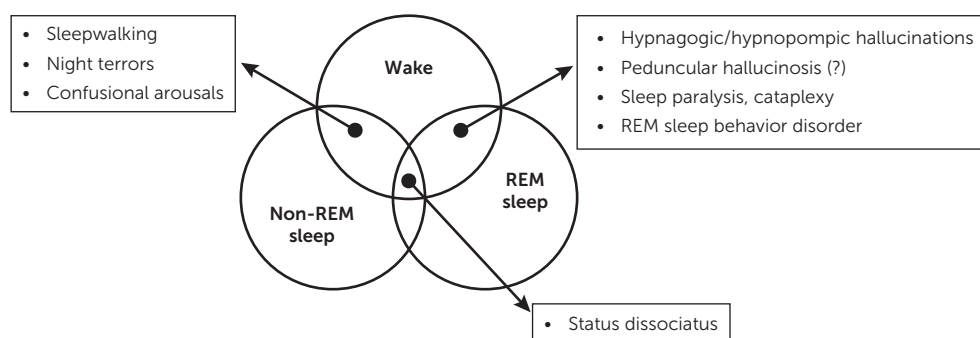
in healthy subjects. They are also frequently accompanied by sleep paralysis, which makes them more frightening (66). Lhermitte, in his 1927 report (67), emphasized their usually visual character and described them as “images,” or “visions,” differentiating them from hallucinations and dreams. He briefly mentioned that in narcoleptic syndromes, “sometimes, before falling into deep sleep, the patient was subject to auditory or visual phenomena of a hypnagogic nature.” Phenomenologically, the hallucinations of peduncular hallucinosis are closer to those described during sleep–wake transitions than to the dreams associated with REM sleep (Table 2). However, the distinction between “half-sleep visions,” “hallucinosis,” and “authentic hallucinations” was of limited relevance for Lhermitte: “If from the clinical and phenomenological point of view the distinction must be maintained, this no longer holds true from the psycho-physiological point of view” (68).

Recently, it was proposed that in some conditions such as Parkinson’s disease, hallucinations could result from REM dream intrusions into the waking state (69), but this remains controversial (70). It is unknown whether narcoleptic-like sleep-onset REM periods occur in patients with peduncular hallucinosis, which would be the first step in linking hallucinations to REM dream intrusions. The rarity and transient nature of peduncular hallucinosis probably made it impossible to perform polysomnographic studies during the syndrome. However, in two cases, it was possible to show by polysomnography that a REM sleep behavior disorder could be associated with peduncular hallucinosis. The first case involved a hypothetical mechanism (compression of the left subthalamic region by a dilated carotid termination) (71). The second followed a small ischemic lesion of the rostro-dorsal region of the pons (72). The patient reported visual hallucinations and illusions, which persisted in an attenuated form during the years of follow-up. He also developed a REM sleep behavior disorder. This case demonstrates that the same circumscribed pontine lesion can lead to both types of disorders, suggesting an overlap in the mechanisms.

From Brainstem or Thalamic Lesions to Visual Hallucinations: Facts and Missing Links

Lhermitte stated that a lesion of a sleep regulating center would allow the “release” of dreams in a waking subject, through mechanisms involving “the entire extent of the brain, and especially the cerebral cortex” (3). This is not the place to elaborate on the complex mechanisms regulating wakefulness, REM and non-REM sleep, and dreams. Briefly, these phenomena depend on the interaction of a number of neurotransmitter systems in the brainstem, hypothalamus, and basal forebrain, which act by modulating the activity of thalamic nuclei and cortical areas (73). Importantly, mechanisms regulating dreams are at least partially distinct from those regulating sleep stages (74). This complexity precludes the possibility of putting forward a mechanistic model linking a lesion of the upper brainstem to the occurrence of transient episodes of peduncular hallucinosis.

One way to approach the neural basis of peduncular hallucinosis without a prior theoretical framework is to identify the regions functionally affected by lesions producing this syndrome. Indeed, the classical clinico-anatomical method has given way to connectivity studies, where the focus is on the circuitry involved in the symptoms and not the location of lesions in individual cases (47, 75). Boes et al. (47) collected 23 cases of peduncular hallucinosis with identifiable causative brain lesions, mapped these lesions onto a standard template brain, and used normative connectome data and overlapping lesion-associated networks to identify regions common to peduncular hallucinosis syndrome. Although the lesions were heterogeneously distributed, 22 of 23 lesions were negatively correlated with extrastriate visual cortex, a region implicated in visual hallucinations, based on prior functional imaging in patients (75, 76). The authors suggested that sites of anticorrelation (i.e., extrastriate visual cortex) predict sites of postlesion hyperactivity (47). Moreover, the peduncular hallucinosis lesions were positively correlated with the lateral geniculate nucleus. Interestingly, using a similar method, Kim et al. (75) showed that lesions causing visual hallucinations were connected to the lateral geniculate nucleus and that all subcortical lesions were again negatively correlated to extrastriate visual cortex. Thus, a plausible final common pathway of peduncular hallucinosis is the activation of extrastriate visual cortex, possibly secondary to an activation of lateral geniculate nuclei. The missing links between the brainstem lesion and visual cortex hyperactivity have been discussed by Müri (77), who summarized two models. The first one derives from the anatomical description in monkeys of a loop linking the area TE of the inferotemporal cortex (an area involved in object, face, and scene perception) to the basal ganglia (78). The proposed loop starts from TE, then projects successively on the tail of the caudate nucleus, the substantia nigra pars reticulata (SNr), the nigral territory of the thalamus (VAmc in Olszewski’s nomenclature), and finally, back to TE. In this model, a brainstem lesion interrupting the excitatory output from the subthalamic nucleus to the SNr would remove the inhibitory input of SNr to VAmc, leading to increased thalamic input to TE. One major limitation of this model is that SNr lesions are not constantly present in cases with peduncular hallucinosis. The second model proposes that a brainstem lesion produces an imbalance in several transmitter systems, including serotonergic and cholinergic systems (79). This imbalance would impair the control of inputs to the thalamus and modulate the gating and filtering mechanisms of the thalamus to the visual and other sensory cortices. The key thalamic nuclei in this model are the dorsal lateral geniculate nucleus and the lateral pulvinar. Schematically, “lesions of the raphe nuclei may produce disinhibition of the dorsal lateral geniculate nucleus and impair the fidelity of retino-geniculo-cortical transmission” (79). In line with Lhermitte, Manford and Andermann suggested that hallucinations in narcoleptic

FIGURE 1. State dissociation^a

^aAdapted from Mahowald and Schenck (85). The diagram shows areas of overlap between states of being and some state dissociation disorders. The inclusion of peduncular hallucinosis in these disorders is in line with Lhermitte's hypothesis but is debatable. REM=rapid eye movement.

syndromes and peduncular hallucinosis were likely to “share a similar pathophysiological basis” (79). Finally, the concept that, in various settings, visual hallucinations are linked to aberrant activity within thalamo-cortical networks remains relevant today (80, 81).

From Peduncular Hallucinosis to State Dissociation

Lhermitte explicitly described a dissociation between normal elements of sleep, waking, and dreaming. Throughout his work, he insisted on this concept with regard to peduncular hallucinosis, writing for example, “Pathological processes can carry out real dissociations of the hypnic function and generate somatic sleep in some cases, psychic sleep in others” (39). Lhermitte also used the term “dissociation” to describe the origin of cataplexy: “We are justified in considering cataplexy as the striking image of a dissociation of the function of sleep, a dissociation which results in inhibition, that is to say the deactivation of muscle tone which contrasts with the conservation of the functions of the mind” (3). He also described sleep paralysis under the term of “awakening cataplexy,” a phenomenon that would seem to “border the physiological state”: “Immediately after waking up in the morning, while his consciousness is perfectly awake, the subject, although he has the desire and the will, finds himself paralyzed. . . . This state is accompanied by an impression of diffuse and extremely unpleasant anguish.” Here again, the phenomenon originates with “the dissociation between psychic awakening and somatic awakening” (67). In short, “the mind watches over a sleeping body in cataplexy” (82). Lhermitte later came back (45) to what Kinnier Wilson had called in the meantime “sleep paralysis” in the context of narcolepsy (83, p. 90). He noticed that sleep paralysis could occur not only on waking, but also on falling asleep or in the middle of the night and that it could be associated with various sensory hallucinations, “psychic hallucinations,” and a “feeling of presence.” In peduncular hallucinosis and narcolepsy, “morbid sleep . . . can . . . manifest a positive side which, contrasting with the inhibition of consciousness or motor

activity, allows the deployment of a complete or fragmentary dreamlike activity” (45).

Although described and discussed by Lhermitte, the concept of state dissociation was only recently formalized by Mark Mahowald and Carlos Schenck, two prominent sleep disorder specialists (84, 85). Healthy individuals have three states of being: wakefulness, REM sleep, and non-REM sleep. Either physiologically or in pathological circumstances, some features usually related to one state may occur in another state (Figure 1). For instance, sleepwalking results from a dissociation between wakefulness and non-REM sleep. In summary, “none of the changes occurring with sleep are invariably coupled to sleep” (86). The concept of dissociation of states, or state dissociation, is now widely accepted (87). Lhermitte was interested in and wrote about most of these states, including hypnagogic hallucinations, cataplexy, sleep paralysis, confusional arousals, and somnambulism (20, 61). For him, peduncular hallucinosis hallucinations belonged to this category of phenomena. Interestingly, Mahowald and Schenck (85), in line with Lhermitte, mentioned peduncular hallucinosis and other types of hallucinations as examples of dissociation between wakefulness and REM sleep, an interpretation that is still under discussion. Finally, one of the most studied dissociation of states at present, REM behavior disorder, was not identified by Lhermitte or contemporary authors but was first described by Carlos Schenck in 1986 (88).

CONCLUSIONS

Jean Lhermitte, in his 1922 article and following works, proposed or anticipated three innovative concepts that still have implications for contemporary clinicians and researchers. The first issue is that a focal lesion, located in the upper part of the brainstem, may cause hallucinations in the visual modality and possibly in others. At a time when psychic phenomena and higher cognitive functions were primarily ascribed to the cortex, Lhermitte claimed that “thought requires, for its normal functioning, the integrity not only of

the cortex but of the underlying centers” (6). His model of a limited lesion acting through complex mechanisms and ultimately involving the cortex, possibly inspired by John Hughlings Jackson (1835–1911) (89), remains valid (75). This view also prefigures contemporary advances taking into account the lesion-induced functional alterations in anatomically intact, connected regions, including in the field of hallucinations (47). Secondly, Lhermitte postulated that peduncular hallucinosis was the result of a dissociation of the mechanisms of dream and waking states, the lesion disrupting the anatomy and connections of a center regulating wakefulness and sleep. The definition of peduncular hallucinosis has since been extended to cases involving a thalamic lesion, but the syndrome described by Lhermitte, although rare, remains accepted. His pathophysiological assumptions were limited by a lack of knowledge, at his time, of the complex and still partially obscure mechanisms regulating wakefulness, the different stages of sleep, and dreaming. The attractive but overly simplistic idea that peduncular hallucinosis is due to a mere intrusion of dreaming in a waking subject has not been confirmed, and the precise mechanisms involved in peduncular hallucinosis remain mysterious. However, discussions on the relationship between dreams and hallucinations are still going on (70, 90). Thirdly, Lhermitte identified that a dissociation of states, as conceptualized nowadays, underpinned several phenomena related to sleep, such as hypnagogic hallucinations, cataplexy, sleep paralysis, confusional arousals, and somnambulism.

According to MacDonald Critchley, Jean Lhermitte “was the *beau idéal* of a neuropsychiatrist” (91). With the description of the peduncular hallucinosis and the discussion of its mechanisms, Lhermitte initiated a broader reflection on hallucinations, which, almost a century later, perfectly illustrates the close relationship and the necessary dialogue between neurology and psychiatry.

Recent studies have examined the relationship between hallucinations and dreams (92, 93), as well as hallucinations in narcolepsy (94–96), building on Jean Lhermitte’s model.

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Supported by the Agence Nationale de Recherche (grant ANR-17-EURE-0017).

Dr. Fénelon reports no financial relationships with commercial interests.

Dr. Fénelon thanks Profs. Jacques Poirier and Françoise Gray for their review of the histological descriptions of two historical cases, Prof. Emmanuel Broussolle for providing sound advice on this article, and Ms. Anna Fitzgerald for editorial assistance.

Received December 30, 2020; revision received April 23, 2021; accepted May 7, 2021; published online October 29, 2021.

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