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## Original Papers

### THE PATHOGENESIS OF NARCOLEPSY

WITH A CONSIDERATION OF SLEEP-PARALYSIS AND LOCALIZED SLEEP

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#### INTRODUCTION

THE purposes of this paper are (1) to call attention to the frequency, in narcolepsy, of a symptom which I propose to call, after Wilson, 'sleep-paralysis' \*; (2) to report the case of a patient who had sleep-paralysis and cataplexy; and (3) to show how certain clinical features of narcolepsy may be interpreted in the light of Pavlov's studies of inhibition.

'Cataplexy' denotes transitory paralysis with tonelessness, occurring in response to sudden emotion (usually laughter). By 'narcolepsy' I mean

\* The term sleep-paralysis was first used by Kinnier Wilson (p. 90 of his paper), who reported the first two cases in which this phenomenon occurred in association with narcolepsy. The terms predormitium and postdormitium were first used by Weir Mitchell.

a syndrome characterized by cataplexy and morbid sleepiness (Gélineau's syndrome).

#### DEFINITION OF SLEEP-PARALYSIS

By sleep-paralysis I mean paralysis occurring either while the subject is falling asleep (we may call this 'predormital' or hypnagogic paralysis) or immediately upon his awakening from sleep ('postdormital' or hypnopompic paralysis). The paralysis may be local or general, in the latter event the subject being utterly unable to move or make a sound. Predormital paralysis may last as long as several minutes and is succeeded by sleep, save that in some instances the inability to move is associated with anxiety and loss of that peace of mind necessary for slumber, the subject becoming once more fully awake. Postdormital paralysis, the commoner of the two, lasts from several seconds to several minutes and goes away gradually or suddenly, leaving the subject as normal as ever; in exceptional instances it lasts longer than a few minutes.

Sleep-paralysis was first described by Weir Mitchell who spoke of it as 'night palsy' or 'nocturnal paralysis.' These terms are open to objection, since the phenomenon may occur in relation to diurnal as well as nocturnal sleep, a fact unknown in Mitchell's time. In 1903 Pfister called the phenomenon 'verzögertes psychomotorisches Erwachen.' The phenomenon received little or no further attention until Rosenthal's papers appeared, in 1927 and 1928. Rosenthal retained Pfister's designation of the phenomenon, while terming the individual attacks 'Wachanfalle.' In 1928 Lhermitte wrote of sleep-paralysis, shrewdly terming it 'cataplexie du réveil,' a term whose aptness will presently become apparent.

Mitchell, Pfister, Lhermitte and (in his 1927 and 1928 papers) Rosenthal, were aware of only the postdormital variety of sleep paralysis. As far as I know, the first instance of *predormital* paralysis was reported in 1928 (Wilson's Case III). In his 1932 paper Rosenthal reported three cases of predormital paralysis, the attacks of which he designated as 'hypnagog-kataplektische Zustände.'

#### THE OCCURRENCE OF SLEEP-PARALYSIS IN CASES OF TYPICAL AND ATYPICAL NARCOLEPSY

Of the more than 200 cases of typical narcolepsy thus far recorded, sleep-paralysis was described in no less than 16. I particularly call attention to this frequency, since no other author has done so, specifically. None of the cases, curiously, was reported before 1928. The 16 cases are the following:—Wilson, 1928, Cases II and III; Brock, 1928; Stransky, 1929, Case I; Zehrer, 1929, Case II; Serejski and Frumkin, 1930, Case I; Redlich, 1931 (an unnumbered case on p. 136); Cave, 1931, Cases V, XVII, XVIII and XXIII; Doyle and Daniels, 1931, Case IV; Rosenthal, 1932, Cases II, III and IV; and Harding and Berg, 1932, Case II.

Thiele and Bernhardt record the fact that of 25 narcoleptics seen at the

Charité Clinic, four had noted the occurrence of 'verzögertes psychomotorisches Erwachen.'

Six more cases have also to be considered. In two of these there were sleep-paralysis and cataplexy, but no morbid somnolence (Rosenthal, 1928, Case VII; Fouché, 1931). In the remaining four there were sleep-paralysis and morbid somnolence, but no cataplexy (Fischl, 1885; Friedmann, 1915, an unnumbered case on p. 92; Rosenthal, 1928, Case V; and Bonhœffer, 1928, Case II).

Riese reported an instance of sleep-paralysis occurring in a patient with cataplexy; we do not know whether the patient also had morbid somnolence.

The following citations illustrate concrete instances of sleep-paralysis.

*Postdormital Paralysis.*—In Rosenthal's Case V (1928) the patient awoke on two or three occasions 'completely conscious but unable to move. . . . He had a slight feeling of tightness in his chest and a slight feeling of anxiety; finally he "gave a wrench with all his might," . . . whereupon he was able to raise his head and open his eyes, the condition therewith clearing up.'

Wilson thus described the phenomenon in the report of his Case II:—

'Of the greatest interest is the fact that when he has been asleep and dreaming, the emotional content of the dream has precipitated an attack of powerlessness. The worst he has ever experienced lasted for about a quarter of an hour, and occurred under the following circumstances. He had fallen asleep and was dreaming of a murder. The emotion experienced in association therewith brought on one of "loss of power" attacks, so that he at once awoke and was fully conscious but was utterly unable to move a single finger. He thought he heard his brother and sister coming up the stairs; he tried to call to them but could not make a single sound; the more he tried the more intense became his emotion and the more absolute his helplessness; he lay thus, flat on the floor, motionless but suffering acute mental distress, for some fifteen minutes ere the attack dissolved itself spontaneously. Similar but less severe attacks have occurred when he has been dreaming of "terrible happenings" on other occasions.'

As a rule the final 'awakening' from a state of postdormital paralysis occurs suddenly. Sometimes the merest touch of another person will promptly dissipate the attack—a fact of profound significance, as I shall later try to show. In Brock's case the paralysis generally cleared up by degrees, rather than all at once. To quote Brock: 'Powerless at first, he begins to "wiggle his fingers," then his hands, and then his feet; next he moves a foot and a leg; then he stiffens his abdominal wall; finally, summoning all his strength, he manages to sit up and almost always shakes his head about as if to "clear it." . . . It takes him from five to ten minutes to get up when so seized.'

*Predormital Paralysis.*—In Rosenthal's Case III (1932) the phenomenon occurred as follows: 'Before falling asleep at night, or when she lies down by

day, the patient suffers states of anxiety and oppression, as though there were a tight band about her chest ; she is fully awake, hears everything, but can neither speak nor move. People about her notice her condition from her deep breathing.'

Only one patient had both predormitial and postdormitial paralysis (Rosenthal's Case II, 1932).

In two instances sleep-paralysis was incomplete. Zehrer reported that when his patient (Case II) awakes, he 'is fully awake, *can answer*, but for a short time cannot open his eyes or move his body' (*italics mine*). In Case II of Harding and Berg postdormitial paralysis was limited to the legs.

Later I shall allude to the frequency and the significance of hallucinations during states of sleep-paralysis.

The following report is of a case recently studied by me.

#### PERSONAL CASE

An intelligent, unmarried woman of 21, first seen in July, 1932, complained of a marked inconstant tremor of the upper extremities and the trunk, beginning in 1928.

*Examination.*—The hands, arms and trunk showed an inconstant coarse rhythmic tremor, both at rest and during movement. The family noted that *the tremors were occasionally present during sleep*. There were no alterations of tone. The skin was unusually dry, and since the age of 17 the patient had been abnormally sensitive to cold. However, the basal metabolic rate (August, 1931) was within normal limits. There was evidence of gonadal disorder. The menses (which had begun at 13) were irregular, the periods being separated by as many as six months. Heterosexual interest was weakly developed. Laparotomy disclosed very small hard ovaries.

*Sleep-paralysis.*—In the middle of 1931 the patient began to have the frequent experience of awakening in the morning fully conscious, but unable to move. 'I've tried to lift my arms and legs and turn over, but I just couldn't move. It just seemed like there was no life in the body. I could think of everything and could hear, but the rest of the body seemed to be dead. Then all of a sudden you limber up, and you're able to move again.' The attacks were more apt to occur at the end of a restless sleep. They lasted only a few minutes. She had had no opportunity to learn whether they could be terminated by such extraneous stimuli as a pinch or a loud call.

At times the eyelids were uninvolved in the paralysis, so that the patient opened her eyes but was unable otherwise to move. She never tried to speak.

During these attacks the patient sometimes had auditory and visual hallucinations. Thus she heard her father talking about flowers (they both enjoyed gardening), and sometimes her girl friends. Sometimes, with her eyes open, she saw these people ; they seemed so real that she did not recognize their unreality until the attack had come to a close.

Similar attacks occurred during the night, especially after bad dreams. Thus, when dreaming that a black man was chasing her, she awoke with an attack of paralysis, which lasted several minutes.

Attacks occurred occasionally during the predormitium. 'While falling asleep, I have sort of lost all power for a few minutes, and then I jump—it's a sort of shock, and it wakens me.'

Sometimes the patient awakened able to move, but 'in a daze—I don't know where I am, and I might think it's a couple of days later than it really is.'

*Cataplexy.*—In May, 1932, the patient began to have attacks of cataplexy. 'If anything is real funny, I get sort of weak and shaky in the knees—I get all limber and

drop everything.' On rare occasions the attacks were so severe that she fell: 'my knees get real weak; they sort of slip out.' More often she did not fall, but was required to lean against something in order to avoid doing so. Annoyance sometimes provoked the attacks, as when her younger brothers and sisters disturbed her with their noisy play. Mild attacks sometimes were unassociated with any discoverable emotion. Thus, while reading or writing she occasionally dropped her book or fountain-pen 'for no reason.'

The attacks of cataplexy never lasted more than a minute and there was no disturbance of consciousness.

*Paroxysmal hypertonia.*—In July, 1932, the patient began to have attacks, precipitated by sudden fear, in which she became 'stiff' and was unable to move for several minutes. The significance of this symptom I hope to consider at another time.

The patient at no time in her life showed the least indication of morbid somnolence. Her nocturnal sleep was quite poor and was disturbed by restlessness. She frequently talked and (especially as a child) walked in her sleep.

#### THE SIGNIFICANCE OF PAVLOV'S WORK FOR THE MAJOR PHENOMENA OF NARCOLEPSY: MORBID SOMNOLENCE, NOCTURNAL RESTLESSNESS, CATAPLEXY AND SLEEP-PARALYSIS: DEFINITION OF LOCALIZED SLEEP

That Pavlov's work on inhibition is profoundly important for the interpretation of the two cardinal symptoms of narcolepsy (sleep attacks and cataplexy) has already been emphasized by several authors, notably by Adie and Worrall. I shall try to show how Pavlov's work makes possible also the interpretation of sleep-paralysis and of nocturnal restlessness as well as of certain other less prominent clinical features of narcolepsy. It is assumed that the reader is acquainted with the general outline of Pavlov's work and with his concepts of 'internal inhibition,' 'irradiation,' 'concentration,' and 'induction.'

The gist of Pavlov's views on sleep may be stated as follows. Sleep, or inhibition, prevents undue fatigue of the cortical elements, allowing them, after they have been subjected to much stimulation, to recover their normal state. Inhibition is occurring all the time, even in a seemingly alert animal. In the alert animal, however, inhibition exists only in scattered areas of the cortex. When from these areas it irradiates over the entire brain, the animal, as we say, falls asleep. In Pavlov's words, 'internal inhibition during the alert state is nothing but scattered sleep, sleep of separate groups of cellular structures; and sleep itself is nothing but internal inhibition which is widely irradiated, extending over the whole mass of the hemispheres and involving the lower centres of the brain as well. Internal inhibition in the alert state of the animal represents a regional distribution of sleep which is kept within bounds by the antagonistic nervous process of excitation' (p. 253 of his book entitled *Conditioned Reflexes*).

We start out with the assumption that the narcoleptic patient possesses a brain in which inhibition occurs with undue ease. This assumption accords with the fact that the narcoleptic is abnormally susceptible to ordinary sleep-provoking situations. In some cases of narcolepsy sleep attacks occur also in

response to strong emotions. Here, presumably, the excitatory process associated with the emotion produces, by negative induction, widespread inhibition. In *cataplexy* this induced inhibition is confined to the motor and tone centres, without affecting the 'substrate of consciousness.'

In *sleep-paralysis*, as in cataplexy, there obviously is inhibition of the motor and tone centres but not of the substrate of consciousness. In *post-dormital* paralysis the inhibitory process existing during sleep fails, as the patient awakens, to disappear from all parts of the brain *simultaneously*. Instead, it disappears from the substrate of consciousness but persists a little while longer in the motor and tone centres, so that the patient awakens to find himself conscious but paralysed. In *predormital* paralysis, as the patient lapses into slumber, the inhibitory process fails to irradiate uniformly over the entire brain. To be accurate, we ought to say inhibitory processes, since sleep represents the coalescence of many irradiating processes. In predormital paralysis these processes invade the motor and tone centres an appreciable length of time before invading the substrate of consciousness.

In certain dogs, under proper experimental conditions, Pavlov found what is virtually an exact duplicate of predormital paralysis in the human. A remarkable instance is described on p. 266 of his book: 'A dog in which work had hitherto proceeded without any interference by sleep began to show signs of drowsiness—due to its being frequently left in the stand in the experimental room for hours at a stretch without any application of conditioned or any other stimuli. . . . The inhibitory effect of the environment became so strong that the mere introduction of the dog into the experimental room had an immediate and obvious inhibitory effect which became still more pronounced after the animal was placed in the stand. It had to be roused in all manner of ways to keep it from falling fast asleep before the preparations for the experiment had been completed (a matter of only a few minutes). When the experimenter left the room and closed the door in order to start the experiment from outside, and then without losing a minute began to apply one or another conditioned stimulus, the normal conditioned reflex was fully present; a normal secretion of saliva was obtained and the animal immediately took the food. When, however, after leaving the room an interval was made of four or five minutes before the application of the first stimulus, this stimulus now produced the following remarkable result. The conditioned secretory effect was present and the salivary secretion was sharply augmented on food being presented; *the animal however did not take the food*, which in order to effect adequate reinforcement had to be placed in its mouth. During this time no relaxation of the skeletal muscles could be observed. When an interval of ten minutes was made after leaving the room no conditioned alimentary reflex could be obtained, and the animal was found fast asleep with a relaxed musculature and occasional snoring. Only one possible explanation of these observations suggests itself. The inhibition must have spread in the first place *only over the motor area of the cortex*, so that excitation

could be initiated by a conditioned stimulus belonging to any other analyser and could spread to the salivary gland but not to the muscles—with a resulting one-sided alimentary reflex lacking its motor component. Later the inhibition spread over the whole mass of the cortex and over the lower parts of the brain, bringing about complete sleep with a relaxation of the skeletal muscles. In this experiment the stages of a gradually developing sleep were brought about . . . ' (italics mine). Like a man with predormital paralysis, this dog in falling asleep passed through a stage in which the centres for voluntary motion were inhibited while the substrate of consciousness continued to be active. This 'demarcation of excitable areas from areas which have undergone complete inhibition' manifests itself in what Pavlov calls 'localized sleep' (p. 266).

At this point we may state the conclusion that *cataplexy and sleep-paralysis represent special instances of 'localized sleep,' the motor centres being in a state of dormancy while the substrate of consciousness is in a state of activity.*

Since inhibition sometimes reigns in the motor centres and not the substrate of consciousness, it is of interest to note that it may also reign in the latter and not the former. On forced marches many soldiers sleep. During the sleep attacks of narcolepsy elaborate motor activity is frequently in evidence. Thus many narcoleptics have sleep attacks while walking. In Case II, in my 1929 paper, the patient fell asleep while cycling and continued to ride until awakened by the cries of pedestrians who saw him heading straight for a bus. The patient of Lhermitte and Roques was a stenographer; during attacks occurring at work she sometimes continued her typing. In these and similar cases, the motor substrate is in a state of activity, while the substrate of consciousness is in a state of inhibition—a type of 'localized sleep' precisely opposite to cataplexy and sleep-paralysis.

Elaborate motor activity may occur not only during the sleep *attacks* of the narcoleptic, but also during his nocturnal sleep. Contrary to the notion held by some students of the subject, the nocturnal sleep of a strikingly large number of narcoleptics is restless, as has been emphasized by Redlich and Rosenthal. The patients toss about and often talk and walk in their sleep. This point is of the greatest importance, since it prevents the conclusion that the sleep disorder of narcolepsy is simply one of excess. If the sleep disorder consisted only of an excessive need of sleep, we would expect nocturnal sleep to be anything but restless. We must conclude that narcolepsy is characterized not only by an excess of sleep, but also by an *increased tendency to localized sleep*. When inhibition is localized in the motor centres, the patient is conscious but paralysed, as in cataplexy and sleep-paralysis. When it is localized in the substrate of consciousness, the patient is asleep (in the everyday sense of the term), but the motor centres continue to be active, i.e. the sleep is accompanied by an unusual degree of restlessness and elaborate motor behaviour.

Since there are cases in which motor activity occurs while the subject is fully asleep (using 'asleep' in its ordinary everyday sense), there must a fortiori be cases in which it occurs while he is *partially* asleep. Such cases have been known for many years, especially in the German literature, where they are designated as cases of 'Schlaftrunkenheit' (see, e.g., Gudden's paper). In these cases the subject, having incompletely awakened from sleep, is able to move freely while not yet fully alert.

We shall now consider a number of minor clinical phenomena, which, though they may seem of trivial importance, are really of great significance.

#### THE DISPERSION OF LOCALIZED SLEEP THROUGH DISINHIBITION

In Wilson's Case III and Cave's Cases XVIII and XXIII it was reported that the patient instantly recovered from his sleep-paralysis when someone touched him. A similar observation was made in Odell's Case VI; an attack of cataplexy cleared up promptly when the patient was touched. This seemingly strange observation becomes understandable in the light of Pavlov's discovery that inhibition may in numerous ways be dispelled by excitatory stimuli. See, e.g., the experiments on pp. 64 and 65 of his book, showing that an extinguished alimentary reflex may be restored by (a) exhibiting a stimulus which evokes an unconditioned defence reflex, or (b) combining the conditioned alimentary stimulus with some casual extraneous stimulus. This dispersion of inhibition by an excitatory process Pavlov calls 'disinhibition.' (The nature of disinhibition will be appreciated by anyone who has tried to fall asleep in a noisy room.) In the four cases cited at the head of this paragraph it is evident that the touch of another person dispelled the inhibitory process which at the moment reigned in the motor centres, thereby returning to the patient the voluntary control of his muscles.

It should be remembered that not every excitatory stimulus may cause the termination of an attack of sleep-paralysis. Thus it is nowhere recorded that an attack was terminated by simply shouting at the patient. Moreover, in Wilson's Case III attacks of sleep-paralysis more than once occurred while the patient had a cigarette in his hand, 'and he has actually burnt his fingers badly without being able to make a single movement to let it drop.' Why severe pain should be inadequate, and touch adequate, to terminate an attack is for the present not clear. Since we do not know what part of the cortex is concerned with pain, we cannot discard the possibility that for topographical reasons the excitation of a touch stimulus irradiates to the motor centres more easily than that of a pain stimulus.

In this connection it is pertinent to refer to Cave's Case II, in which the patient by pinching himself brought to a stop his atypical cataplectic attacks, and to his case XXI, in which voluntarily increasing the tension of the muscles of the legs or the tongue served to avert an impending cataplectic

attack. These are two more instances in which excitatory processes, by disinhibition, averted or cut short a manifestation of localized sleep.

In Rosenthal's Case III (1932) it was noted that if during an attack of predormital paralysis the patient was placed in the sitting position, the attack came to an end. Calling to the patient and touching her had no such effect.

#### CLINICAL MANIFESTATIONS OF LOCALIZED SLEEP RESULTING FROM 'CROWDING' OF INHIBITION

The patients in Wilson's Cases II and III, Cave's Cases VI and X, and Rosenthal's Case II (1932) made the observation that attempting to fight off a sleep attack was apt to give rise to an attack of powerlessness. Here again is an observation which at first may seem puzzling, but which is understandable in the light of Pavlov's theory of sleep. Sleep arises through the irradiation and coalescence of many scattered inhibitory processes. When a person fights sleep off, a multitude of excitatory processes come into play, opposing the irradiation of the inhibitory processes. The latter, when not entirely checked, continue to irradiate where they are least opposed. They will invade the motor areas, if these areas are (as in narcolepsy they presumably are) unduly inhibitable.

To my knowledge no observer has made the experiment of forcibly keeping a narcoleptic from succumbing to a sleep attack. It would be desirable to know whether under such conditions powerlessness occurs with significant frequency.

An instructive case is Cohen's Case II. The patient gave the following account of one of her cataplectic attacks: 'I think I could have stopped it in the first stages, but unfortunately I was stupid enough to concentrate not on sitting still, but on trying not to spill the coffee from the cup I was holding, with the result that I kept the coffee cup quite safe, but slipped to the floor beside it.' Here, in the clearest way, we see the 'crowding' of inhibition from one part of the brain to another—in this instance, from the central mechanism of the upper limb to that of the trunk and the lower limbs. One is reminded of the football player who, when rushing the ball, is 'crowded' by his opponents into that part of the field where he meets with the least opposition.

In two carefully studied cases reported by Rothfeld (his Cases II and III) erection habitually followed when the patient attempted to ward off a sleep attack. These patients, also, generally had erections during their sleep attacks. Study of cases of spinal transection has shown that the lower sexual reflexes are inhibited by higher parts of the nervous system. I therefore suggest that in Rothfeld's patients the excessive inhibitability of the narcoleptic brain applied particularly to the higher sexual-inhibitory centres; when sleep was warded off, inhibition was 'crowded' into these centres, their inhibition releasing the lower sexual reflexes.

### ATTACKS OF POWERLESSNESS 'WITHOUT APPARENT CAUSE'

A number of narcoleptics have reported that in addition to cataplexy (powerlessness precipitated by emotion) they had attacks of powerlessness coming apparently out of a clear sky (Wilson's Cases III and V and Cave's Case XXIII, to mention but a few). I believe that Pavlov's work helps us to understand these attacks. Pavlov has shown that even in the outwardly alert animal inhibitory processes may be demonstrated here and there in the cortex. At just what stage in the irradiation of these processes the individual begins to 'feel sleepy' we, of course, do not know. It may conceivably happen that a sleep attack is 'in the making' and is checked by excitatory stimuli before the patient has had any inkling of the fact that he was on the way to falling asleep. Should the inhibitory processes, checked in their irradiation, invade the motor centres, the patient will be under the impression that he had an attack of powerlessness 'without apparent cause.'

### HALLUCINATIONS AND OTHER DISTURBANCES OF MENTATION IN SLEEP-PARALYSIS

In clinical medicine we expect to find transitional stages between phenomena belonging to the same series. Sleep-paralysis is, in a certain sense, a partial sleep, the substrate of consciousness being 'awake' while the motility substrate is dormant. We therefore ought to find, between sleep and sleep-paralysis, intermediate stages in which the patient is paralysed and consciousness is imperfect. As an actual fact, such intermediate stages are seen in seven cases in which hallucinations occurred during attacks of sleep-paralysis: the cases of Brock; Serejski and Frumkin; Doyle and Daniels; Rosenthal, Cases II, III and IV (1932); and the case which I have here reported.

A discussion of the different levels of mentation would be beyond the scope of this paper. It will suffice to say that the consciousness of a hallucinating person is of an inferior sort. It is the inferior consciousness existing even in healthy people as they gradually fall asleep, when thoughts become 'blurred,' associations follow inferior patterns and hallucinations occur. Thus we may say that in sleep-paralysis accompanied by complete alertness and in the absence of hallucinations, the substrate of consciousness is (as far as we now know) rigidly excluded from the inhibitory process; in sleep-paralysis accompanied by hallucinations the substrate of consciousness is partially inhibited.

In Cave's Cases V and XVIII and in Riese's case, during attacks of sleep-paralysis the patients showed evidence of inferior mentation other than hallucinations.

### THE OCCURRENCE OF POSTDORMITAL PARALYSIS AFTER FEAR DREAMS

In Wilson's Case II, in Cave's Case XVII and in the case which I have here reported, fear dreams precipitated attacks of sleep-paralysis. The

analogy to the cataplexy of the awake state is obvious. Evidently, strong emotion can not only lead to the inhibition of the (uninhibited) motor centres during the waking state, but, occurring during sleep, can also restrain the disinhibition of the motor centres which is normally incident to awakening.

#### SLEEP ATTACKS AND OTHER SYMPTOMS OCCURRING IN CONSEQUENCE OF THE VOLUNTARY SUPPRESSION OF POWERFUL REFLEXES

My second patient, reported in 1929, had made an observation which puzzled me before I became acquainted with Pavlov's work. He stated that on two occasions he had succumbed to a sleep attack in the dentist's chair *while the dentist was actually drilling his teeth*. Considered from the standpoint of everyday experience, sleep precipitated by pain is incomprehensible. But Pavlov has shown us that in this connexion the nature of the stimulus itself is less important than the inhibition associated with it. 'As there is practically no stimulus of whatever strength that cannot, under certain conditions, become subjected to internal inhibition, so also there is none which cannot produce sleep' (p. 252). As an example he mentions an instance in which a powerful electric shock applied to the skin was used as a conditioned alimentary stimulus. After many months of experimentation the stimulus acquired inhibitory properties and became a 'most effectual agent in inducing sleep.' Similarly, in the case of my patient, the painful drilling of the teeth must have acquired inhibitory properties sufficient to induce sleep. In the child painful stimuli arouse uninhibited defence reflexes. As the child grows up a chain of developments occurs, which for the sake of brevity I shall express in the following psychological terms: the child, desiring not to appear a coward, strives to suppress his defence reflex to pain. This process is in all essential respects identical with inhibition. It is therefore understandable that in the dentist's chair pain stimuli arouse powerful inhibitory processes which—in the susceptible person—may eventuate in sleep.

A similar instance has been alluded to by Gowers. The case was that of a man who had attacks of sleep, 'which were induced by most varied influences. He had a nasal fistula, and whenever a probe was passed down this he fell asleep.'

The significant feature of these two cases is that the patients were in situations which required them to bear pain without flinching. If either of them had hurt himself by stubbing his toe, he would not have had to suppress his normal reaction thereto, and would in consequence probably not have fallen asleep.

In this connexion I allude to Rothfeld's Case II. The patient once lay in bed next to a girl and would have had intercourse, had it not been for the presence of a third person. 'He was sexually very excited and suddenly had the feeling as though ejaculation were going to occur. Simultaneously he was overcome by great weakness, so that he lay as though paralysed and

was able neither to move a limb nor call for help. He was perfectly conscious. This condition lasted a little while, after which he got up and felt perfectly well.' Here the inhibition of a powerful reflex led, not to sleep, but to a form of localized sleep. In principle, this incident duplicates that which occurred in Gowers' patient and in mine.

### CONCLUSIONS

The clinical features of narcolepsy accord with the assumption that the basis of the disorder lies in the undue 'inhabitability' of one or more parts of the brain. When inhibition invades simultaneously the consciousness substrate and the motility substrate, the resulting sleep attack is indistinguishable from ordinary sleep. The characteristic stamp of narcolepsy lies in the frequency with which inhibition invades one of these substrates without invading the other, thus giving rise to one of the manifestations of 'localized sleep.'

When inhibition is confined to the substrate of consciousness, the patient is 'asleep' in the ordinary sense of the term, but is restless or performs complex motor acts automatically (narcoleptic patients are frequently restless in their nocturnal sleep). When inhibition is confined to the motility substrate, the patient is conscious but unable to move, as in cataplexy and sleep-paralysis.

An explanation is possible for the following phenomena: the dispersion of cataplexy and sleep-paralysis by extraneous stimuli, such as a touch; the occurrence of powerlessness in consequence of the effort to ward off an impending sleep attack; the occurrence of powerlessness 'without apparent cause'; the occurrence of hallucinations during sleep-paralysis; the occurrence of postdormital paralysis after fear dreams; and the occurrence of sleep attacks as a reaction to pain that has to be borne without flinching.

It need scarcely be added that a variety of causes may weaken the nervous structures so as to render them abnormally inhabitable, and that narcolepsy is but a symptom of some underlying disease.

### SUGGESTIONS FOR THE CLINICAL STUDY OF CASES OF NARCOLEPSY

I suggest that those who have cases of narcolepsy under their observation pay particular attention to the following questions.

1. In attacks of sleep-paralysis, will a touch cause the attack to go away? If so, may it be a touch on any part of the body? What other stimuli, if any, have a like effect? The same question should be asked in regard to attacks of cataplexy.

2. During attacks of sleep-paralysis and of cataplexy what precisely is the state of consciousness? Is there evidence of an inferior sort of consciousness, such as hallucinations, dreamy thinking, etc.?

3. During sleep attacks to what extent is automatic motor activity present?

4. When the patient tries to fight off a sleep attack, do clinical manifestations of localized sleep supervene? *What happens when the observer forcibly restrains the patient from succumbing to a sleep attack?*

5. What symptoms, if any, appear when the patient is in a situation requiring him to suppress powerful reflexes, as when the dentist is drilling his teeth?

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