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Isolated Sleep Paralysis: A Web Survey

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Isolated Sleep Paralysis (SP) occurs at least once in a lifetime in 40-50% of normal subjects, while as a chronic complaint it is an uncommon and scarcely known disorder. A series of messages written by subjects who experienced at least one episode of SP, containing more or less detailed descriptions of this disorder, were collected from the Sleep Web site of the University of California in Los Angeles between January 1996 and July 1998. Two hundred and sixty-four messages fulfilling the International Classification of Sleep Disorders (ICSD) (Thorpy, 1990) minimal criteria for SP were analyzed. A wide spectrum of severity was evident, with a frequency of episodes ranging from one in a lifetime to almost every night, and a variety of emotional and hallucinatory experiences associated with SP episodes were reported. Clinical similarities between the recurrent form of isolated SPand channelopathies (in particular, periodic paralyses) are discussed. An activation of limbic system structures is suggested in order to explain some of the most common subjective experiences associated with SP.

CURRENT CLAIM: Isolated Sleep Paralysis may occur with a wide spectrum of severity, including a recurrent form which closely resembles the ion-channel pathologies, and it is often accompanied by stereotyped subjective experiences which suggest an activation of limbic system structures.

Sleep Paralysis (SP) and hallucinations in the wake-sleep (hypnagogic) or sleep-wake (hypnopompic) transition can occur separately or in association, and they seem to share a common neurophysiopathologic substrate, as shown by polysomnographic registrations of such events occasionally captured in a laboratory. In either case, clinical and electrophysiologic assessment evidences a mixed pattern of REM sleep phase and wakefulness (Takeuchi et al., 1994; Dyken et al., 1998). REM sleep is a brain-activated state, characterized by a desynchronized electroencephalographic activity resembling that of wakefulness. Bursts of phasic events, such as REMs (rapid eye movements) and MEMA (middle ear muscular activity), are also present and seem to be related to ponto-geniculo-occipital (PGO) spikes, which originate in the mesopontine region and propagate to the cerebral cortex. REM sleep phasic activities are supposed to underlie the hallucinoid imagery of dreams. In particular, PGO spikes project to the visual lateral geniculate nucleus and occipital cortex and may trigger the visual imagery of dreams (Steriade and McCarley, 1990). In spite of this high brain activation, REM sleep differs from wakefulness because of muscular atonia, with abolished H-reflex, caused by inhibitory postsynaptic potentials in spinal motoneurons. Furthermore, late cerebral responses to somatosensory stimulation (P100, P200, P300) disappear during REM sleep (Goff et al., 1966; Velasco et al., 1980). SP differs from normal REM sleep since there is not such blocking of exteroceptive stimulation, and waking consciousness is largely retained (Hishikawa and Kaneko, 1965). SP may occur in an isolated form in otherwise healthy individuals, as well as in a familial form transmitted genetically and as one of the classical symptom tetrad of narcolepsy. Isolated SP occurs at least once in a lifetime in 40-50% of normal subjects, while as a chronic complaint it is much less common, affecting 3-6% of survey respondents, many of whom had rare episodes (Thorpy, 1990). The aim of this study is to contribute to a better clinical characterization of this still poorly understood disorder.

METHODS

We collected all the messages regarding "Sleep Paralysis" posted on the Sleep Home Pages Web site (http://www.sleephomepages.org), between January 1996 and July 1998. In these messages, subjects who experienced at least one episode of SP described their experiences in a more or less detailed fashion. Since all the messages were written freely, following the original one posted by a doctor in physics affected by this condition, not all subjects gave complete information. It is possible that a few messages, in a random way, escaped the collection.

Inclusion Criteria

We followed the minimal criteria of the ICSD (1990): A (a complaint of inability to move the trunk or limbs at sleep onset or upon awakening) + B (presence of brief episodes of partial or complete skeletal muscle paralysis) + E (not associated with other medical or psychiatric disorders); the latter criterion has been applied as far as possible according to the available data. On the other hand, all participants reported a substantially stereotyped symptomatology (a disorder arising at sleep onset or on awakening, characterized by skeletal muscle paralysis associated with a high level of alertness, with sparing of ocular movements and a cessation that occurs in seconds or minutes when spontaneous, or suddenly when the patient is touched by a relative) that seems to warrant a sufficient diagnostic certainty.

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RESULTS

We collected 337 messages about SP; of these, 73 were rejected according to either criterion E of ICSD (exclusion of other medical or psychiatric disorders), or because they didn't concern the matter of the study (e.g., questions, explanations given by "experts," suggestions for new researches on the topic), or because they were replies from authors of previous messages. Two hundred and sixty-four messages containing more or less detailed descriptions of SP were retained for analysis.

Epidemiological Characteristics

Most authors are Americans, but Canadians, Australians and Europeans are also represented. Males are by far prevalent, but it's not possible to provide definite data because several subjects signed their messages with a pseudonym. Eighty-two patients declared their age, which is so distributed: 14-19 years, 18 patients; 20-29 years, 37 patients; 30-39 years, 20 patients; >39 years, 7 patients.

Only six subjects reported that they had received a defined or possible diagnosis of narcolepsy by qualified physicians; all the others reported SP as the only significant complaint. Anyway, since the messages were written freely and no patient was clinically or polysomnographically examined in this study, it is not impossible that a diagnosis of narcolepsy could have been considered for some other case.

Natural History of the Disease

Tables 1-4 show data regarding the age of onset, the frequency and time of occurrence (sleep onset or awakening) of the episodes and the reported precipitating factors.

Only a minority of patients reported that they had one or a few episodes through their life; all the others reported a recurring disease, with a highly variable frequency ranging from a few episodes per year to almost every night. In most cases, the onset of the disease occurs during childhood or adolescence; the course is sometimes characterized by clusters of episodes alternating with even long periods of remission, with episodes often decreasing in frequency in adulthood. The length of single episodes is subjectively estimated in seconds or a few minutes in almost all cases. The time of occurrence (sleep onset or awakening) is available, though not always certain, for 112 patients. As shown in Table 3, there seems to be a prevalence of hypnopompic episodes, with attacks occurring in the middle of the night or at the end of nocturnal sleep. Eight patients (3%) had SP only in the course of diurnal nap. Twenty-nine patients (11%) reported that sometimes SP can occur more consecutive times at every attempt to fall asleep. In this case, the first episode can be either hypnagogic or hypnopompic. Sometimes the only way to stop this phenomenon involves definitively interrupting sleep for some time; that is to say, the patient has to completely awaken (for example: getting up after the first episode, walking around, getting a drink of water, etc.) before falling back to sleep again.

Table 1. Age of Onset (Reported by 96 Patients)

	Nº	%
"As long as I remember"	14	14.6
≤12 yrs	34	35.4
13-19 yrs	35	36.5
≥20 yrs	13	13.5

Table 2. Frequency of the Episodes

	Nº	%
<5 lifetime	27	10.2
1-5/year	37	14.0
"Regularly" (0.5-4/month)	27	10.2
>1/week	13	4.9
Irregular/Clusters	20	7.6
"Several" or "Innumerable"	27	10.2
"Recurring"	95	36.0
Progressively decreasing in adulthood	18	6.8

Table 3. Time of Onset

	Nº	%
At sleep onset	37	33.0[a]
On awakening	59	52.7[a]
Either	16	14.3[a]

[a]Percentage calculated on 112 reports for which the datum was available.

Table 4. Precipitating Factors

	Nº	%
Nap	22	8.3
Sleep schedule alteration	21	8.0
Supine position	17	6.4
Mental or physical fatigue	17	6.4
Stress	14	5.3
Alcohol /caffeine	9	3.4
Jet lag	2	0.8

Not reported by all subjects; some subjects reported more than one. Nº is the number of subjects reporting a precipitating factor; % refers to total sample.

Subjective Experiences During SP

One hundred and ten subjects (41.6%) reported that, before they had access to reliable information about SP by means of "Web Forums" and sleep medicine Web sites, they had no idea about the nature of the phenomenon. In particular, 56 patients thought they were the only ones to have such a disease, 18 patients thought that it was a psychiatric illness, 11 patients were afraid of being "possessed by the devil" or "visited by spirits," while 17 patients told their experiences to friends and 8 patients told their physicians. As a result, no one believed

Table 5. Subjective Experiences Associated with SP

	Nº	%	
Fear/terror/panic	189	71.6	>2/3
"Presence"	87	32.9	1/3
"Phantom noises"	77	29.2	
Visual hallucinations	37	14.0	
Acoustic hallucinations[a]	35	13.3	
Pressure on the body	33	12.5	
"Phantom movements"[b]	31	11.7	
"Out-of-body experiences"[b]	28	10.6	
"Floating"[b]	19	7.2	
Apnea/dyspnea	22	8.3	
Physical touch	20	7.6	

"Phantom noises:" unstructured acoustic hallucinations; see examples in Appendix I, 6, 7.
"Phantom movements:" a false, subjective sensation of movement; see example in Appendix I, 8.
[a]Other than "phantom noises."
[b]Some patients could have mistaken these experiences; see examples in Appendix I, 8-10.

Table 6. "Paroxysmal Hallucinations" Associated with SP

Motor hallucinations	Jerking; shaking; thrashing; convulsing; eye blinking, twitching or fluttering
Sensorial hallucinations	Electrical shocks; a sense of energy running through the body; a shiver; tingling; tickling
Visual hallucinations	A spinning swirl of colors; flashing lights; colorful static (like when TV is on the fritz)
Acoustic hallucinations	Buzzing sound; loud ringing; rumble noise; radio waves and computer bleeps
Vestibular hallucinations	Rolling; spinning

Table 7. Differential Diagnosis of the Conditions which could Mimic Isolated SP

Conditions which could mimic isolated SP	***Differential diagnosis***
Cataplexy	Occurrence in waking state; triggered by emotional stimuli
Atonic seizures	Usual occurrence in waking state
Drop attacks	Unrelated to sleep-wake transitions; older patients
Hypokalemic Paralysis	Low serum potassium levels during attacks; familial transmission
Hysterical and psychotic states with immobility	Associated clinical features
Drug withdrawal/abuse	Anamnesis
REM rebound	Anamnesis (sleep deprivation)

Table 8. Clinical Similarities Between PISP and Channelopathies

	"Periodic" Isolated Sleep Paralysis	***Hypokalemic Periodic Paralysis***	***Other Periodic Paralyses; other chan - nelopathies***
Episodic occurrence	+ (at sleep onset or on awakening)	+ (on awakening)	+
Genetic factors	+ (Familial form)	+	+
Precipitating factors	Stress, fatigue, alcohol, sleep sched. alter., nap	High carbohydrate meals, alcohol, sleep	Stress, fatigue, exercise, certain foods, alcohol

them or gave them reliable explanations. Several patients were convinced that SP and related experiences were not simply a physical phenomenon, but that they fell into the realm of the paranormal. Twenty-five reported that they used to pray during the episode, getting a great help from this practice.

Table 5 shows the most common subjective experiences related to SP. Each episode may occur with different characteristics in the same patient. The reported frequencies were calculated according to the number of patients that experienced a specific symptom at least once.

More than two in three subjects reported feelings of panic/fear/terror during the episodes. This sensation can be very intense and persist in patients'memories for years. Many subjects reported that, in spite of the recurrence of episodes and of the awareness that the event would stop with no harm within seconds or a few minutes, the experience was always as terrifying as it was the first time (Appendix I, 1-3). Some patients, however, get used to the episodes and find them no longer terrifying but simply annoying. A few subjects enjoyed the episodes and tried to study them or to use them as a "launching pad" toward some particular states of consciousness, i.e., "lucid dreaming" and "out-of-body experiences."

Approximately one in three patients reported that during some SP episodes they felt a "presence" in the sleeping environment. In most cases this was not reported as a visual experience but as a dim perception; only 15 patients referred to it as a "dark," "shadowy" or "black" figure. In almost all cases the "presence" had a definite "evil" character and it was felt as a threat (Appendix I, 4-5).

Less than one in three patients perceived, during some episodes, an acoustic hallucination described as a "buzzing sound," "loud ringing," "rumble noise" or something of this sort. These sounds often represented the first sign of a forthcoming episode. Their emotional intensity can be so high that some subjects consider them the most characterizing

Table 9. Clinical Characteristics Suggesting Limbic System Activation During SP

Sleep Paralysis related experiences (% patients in our survey)	***Limbic System***
Fear/terror/panic (72%)	Fear (amygdala stimulation) Emotional reactions (cingulate gyrus)
"Threatening presence" (33%)	Self-protection (amygdala) Attention/surprise/defense reactions (amygdala stimulation)
"Phantom noises" (29%)	Tinnitus within a reflex of protection against impending dangers (hypothesis)
"Oneiric vision" of surroundings[a]	Memory (hyppocampus, amygdala)

[a]Appendix I, 11-13

element of the experience and particularly, if the patient struggles to move, they can change into a sense of vibration of the whole body or into a physically painful sensation (Appendix I, 6-7).

The other subjective sensations more frequently reported were: pressure on the body; physical contact; "phantom movements;" "floating;" "out-of-body experiences;" dyspnea/apnea; and visual hallucinations or acoustic hallucinations (other than the sounds reported above). Table 5 reports their respective frequencies.

A series of motor and sensitive/sensorial hallucinations may sometimes mimic sleep-related epileptic seizures (Table 6). Such "paroxysmal hallucinations" are vividly perceived during sleep and they suddenly stop at the moment of full awakening.

Finally, many patients reported that they can distinctly see the sleeping environment during SP; nevertheless, in most cases this seems to be an oneiric–though extremely detailed–experience, as results from some patients who note that such "vision" occurs with eyes still closed, or from others who note some small but significant differences from their "real" environment. One subject has even conceived little experiments, putting some objects in unusual places and concluding, not having identified them during the experience, that what is "seen" must be stored in the patient's memory of the surroundings (Appendix I, 11-13).

DISCUSSION

Our study leads us to some general observations about this peculiar condition. First, it is noteworthy that more than 40% of patients reported that they had no idea (or had wrong/irrational ideas) about the nature of the disease, conjecturing a mental disease or spiritual/paranormal phenomena before they found a scientific explanation through the Internet sleep Web sites and Discussion Forums. A similar way of thinking had previously been described among subjects affected by SP in developing countries, where patients rarely spontaneously volunteer these fears and doctors pay them scant attention (Ohaeri, 1992).

A second surprising datum of this survey deals with the high number of patients reporting isolated SP as a chronic condition, with a frequency ranging from a few episodes per year to almost every night. According to ICSD (Thorpy, 1990), SP as a chronic complaint affects only 3-6% of individuals; reported data seem particularly representative of such a restricted group of patients. This population has probably been selected because of a bias due to the kind of disease that is likely to induce a patient to perform personal research on the topic. This could explain the very low presence, in this survey, of narcoleptics (who are more likely to consult qualified physicians and to get adequate information on their condition) and the surprisingly high ratio among subjects with recurrent attacks (who are more likely to search for information) and subjects having had only one or very few episodes.

Since this study was based upon a series of messages written freely by subjects who seemed to experience the same disorder, but no patient was examined, it is possible that not all the collected stories of "isolated SP" represented the same pathophysiologic phenomenon. In Table 7, the conditions that could mimic isolated SP are listed. Though some of the collected stories could be of doubtful interpretation, in most cases the disorders listed in Table 7 could be ruled out with a high degree of certainty on the grounds of the information given by the subjects.

By analogy with a group of neuromuscular diseases called "periodic paralyses," we could define "Periodic Isolated Sleep Paralysis" (PISP), the recurrent disorder reported by most patients of the present study.

While in narcoleptic patients the hypnagogic form of SP is largely prevalent (Thorpy, 1990; Hishikawa, 1976), isolated SP seems to be more often hypnopompic (Table 3), with episodes occurring at the end of nocturnal sleep or in the middle of the night. From a physiopathological point of view, this observation somehow challenges the current interpretation of SP as due to the marked dissociation between the level of alertness and skeletal muscle atonia during sleep onset REM periods (SOREMPs) (Hishikawa, 1976; Hishikawa and Shimizu, 1995). Furthermore, while some hypotheses have been formulated about the neural and biochemical anomalies accounting for the occurrence of SOREMPs in narcoleptic patients (Hishikawa and Shimizu, 1995), at present nothing is known about the mechanisms underlying anomalies of REM-wakefulness transition in the middle or end of the night. From an etiopathogenetical point of view, some clues might come from some evident clinical similarities between PISP and a group of neurologic disorders known as "channelopathies" (which comprise periodic paralyses, some kinds of migraine and of epilepsy and some episodic movement disorders). Such similarities appear in the periodic occurrence, the possible influence of genetic factors, and the presence of similar precipitating factors (Ptácek, 1998) (Table 8). The episodic

occurrence of periodic paralyses and, in general, of channelopathies suggests the existence of some biologic anomaly that must be mild enough to let patients be perfectly normal between attacks. Certain factors can push the patient "beyond the border" and induce an attack. Known precipitating factors include stress, physical fatigue, and some foods and drinks. Such analogies seem to suggest that all these conditions share a common physiopathologic basis (Ptácek, 1998).

In particular, Hypokalemic Periodic Paralysis (HypoKPP), which is due to mutations in a gene of calcium channel in skeletal muscle, is cited by the ICSD (Thorpy, 1990) as "…the only condition that closely mimics sleep paralysis. The attacks usually occur during rest; paralysis occurs on awakening as in true sleep paralysis." Furthermore, several patients with an established diagnosis of HypoKPP report that during their attacks (and not at other times) they present vivid lucid dreams and frank hallucinations closely resembling the ones which occur during SP(Appendix II, Hypokalemic Periodic Paralysis Resource Page). This seems to suggest a disorder of REM sleep regulating mechanisms during some HypoKPP episodes. We could speculate that in some cases of HypoKPP, the ion-channel anomalies responsible for muscle weakness may somehow also affect the neural systems regulating REM sleep and trigger dissociated REM episodes during the attacks. This would be a further link between SP and periodic paralyses.

A second bias affecting this study is due to the fact that the epidemiologic characteristics of patients are influenced by the kind of subject who can have access to an American sleep medicine Web site; that is to say, it is a population made up mainly of young American males. Subjective experiences associated with SP may be influenced by cultural factors, as appears from the different interpretation and denomination of such experiences, i.e., "old hag," "Kanashibari," etc. (Dahlitz and Parkes, 1993). In the present survey, such cultural influences can be found in some reports of "alien abduction" beside more traditional "visits" by "spirits" and "demons" (Appendix I, 14). The central subjective experience associated with SP, however, seems to be highly stereotyped through all ages and countries, and consists of feelings of fear or terror and of a threatening presence, while the subject believes to see, quite distinctly, his surroundings. Such experiences suggest an activation of limbic system structures during SP. In fact, either the terror, which is not simply due to the awareness of paralysis, since most patients keep on experiencing it though they have become aware that the disease is not harmful and it will stop in a few seconds or minutes, or the feeling of an impending threat suggest an involvement of structures (the amygdala in particular) controlling emotions and individual attention/surprise/defense reactions (Davis, 1997; Sleep Paralysis Page, 1998) (Table 9). Also, the acoustic hallucinations ("buzzing sound," "loud ringing," etc.) perceived by approximately one third of the patients during SP may be explained in a similar way, according to a hypothesis on "phantom sounds" (Hazell, 1995; Dauman, 1998). According to this theory, tinnitus would take rise as a "pathologic product" of a reflex of protection against impending dangers. Such a reflex is supposed to be a phylogenetic inheritance of a mechanism—of vital importance for wild animals—of individuation and amplification of even minimal noises which could represent a threat, during nighttime in particular. In support of their theory, the authors emphasize that tinnitus often starts or increases during states of alertness or anxiety disorders. Finally, as noted above, the reported "distinct vision" of surroundings during SP seems to be, in most cases, an oneiric experience based upon a recollection of information stored in the patient's memory and involving, once more, an activation of specific areas within the limbic system (LeDoux, 1993).

Recent reports suggest an activation of the limbic system during physiologic REM sleep (Nofzinger et al., 1997; Braun et al., 1998). We could speculate that the arising of a high level of alertness while limbic structures maintain a condition of activation different from that of normal wakefulness causes the typical, stereotyped subjective experiences of SP. These hypotheses can find validation only in more sophisticated research.

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APPENDIX I
Examples from the Examined Cases

1. "Nothing I have ever encountered in my life has ever come close to the absolutely unreal feeling of terror that grabbed me the night when I had my S.P. experience."
2. "I was filled with the most awful terror I have ever experienced."
3. "…and it is always just as terrifying as it has ever been."
4. "I often feel a sense of an evil presence when I have one of these episodes."
5. "I always felt a 'presence'in my bedroom during this stage, however I could not identify it clearly."
6. "They always start with this buzzing sound getting gradually louder and suddenly I'm trapped."
7. "…the ringing or buzzing sensation in my ears. This occurs just before the paralysis occurs. I've found that if I can wake up then, I can prevent the paralysis from occurring."
8. "Sometimes I get up and move, only suddenly to wake up in my bed like I didn't ever leave. Maybe what they call astral travel? It is hard to articulate the experience."
9. "I too have floated around the room during some of the paralysis occurrences and then the next day tried to explain it to someone only to get strange looks and people thinking I'm nuts."
10. "I looked down and realized that I could see my body below me. I was on the ceiling."
11. "I think I'm awake, so I look at my alarm clock to check,

and if the bright green led is not there, then I immediately know that is a sleep disorder experience…my bedroom seems the same as it is during waking, only the lights don't work."
12. "I have some awareness that my eyes are closed, but I can see my surroundings very clearly. My bedroom is exactly as it is in reality."
13. "I usually end up on the ceiling looking at myself in bed...I've placed things on top of the wardrobe to try to see (while I'm on the ceiling), because I need some evidence that what I'm feeling is real or a hallucination. So far I haven't been able to identify anything, so I guess what you see is just stored in the memory of your surroundings."
14. "Lately I have been having sleep paralysis dreams about aliens abducting me. It feels so real, I try to scream but I can't move!"

APPENDIX II
Vivid Dreams and Hallucinations in Course of Hypokalemic Periodic Paralysis[a]

1. "The lucid dreams serve as a 'warning system' as I recognize from the character of the dream that I am entering an episode, and that I need to awaken and get some potassium."
2. "The hallucinations generally occur only when I awaken completely paralyzed, and thus they resemble in some ways a sleep paralysis. I have never found these frightening, as many people seem to do, but then I usually believe I am awake and I am doing something when in fact I am totally inert."
3. "I am one that has tremendous hallucinations when I am in an abortive attack…I have had an imaginary choir of children come to sing to me when I was not feeling well and too weak to get out of bed."
4. "I have very vivid dreams when I'm going into an episode…I'll realize that I'm going into an episode and I'll wake myself up enough to flail around to get my husband's attention."

[a]Information achieved from members of the HypoKPP On-line Support Group; the group can be contacted at the address reported on the Hypokalemic Periodic Paralysis Resource Page (see References).

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