PREVALENCE OF ISOLATED SLEEP PARALYSIS IN BLACK SUBJECTS

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Sleep paralysis is a state of consciousness experienced while waking from sleep or falling asleep. It is characterized by an experience of being unable to move for several seconds or minutes. This study represents the first survey to measure the incidence of this disorder in a black population of healthy subjects and psychiatric patients.

Sleep paralysis has been described as an unusual neurologic phenomenon that is usually considered a component of the narcolepsy tetrad of sleep attacks, cataplexy, sleep paralysis, and hypnogenic hallucinations.[1] Sleep paralysis is a state of consciousness experienced either while waking up or falling asleep, characterized by feeling unable to move for several seconds or minutes. The individual who experiences this state is fully aware of the condition and has complete recall of the episode. Vivid and terrifying hallucinations often accompany this state of consciousness and a sense of acute danger may be felt. Once the episode of paralysis passes, the individual often sits up with a start and experiences symptoms of anxiety (eg, tachycardia, hyperventilation, fear) only to realize that the perceptions of danger were false.

In *Interpretation of Dreams*[2] Freud mentions hypnagogic hallucinations citing Johannes Muller's 1826 term for this experience, "imaginative visual phenomena"; however, it is not clear that Muller's imaginative visual phenomena occurred in conjunction with sleep paralysis. The first definite case was reported by Binns, who described a patient's "daymare," an episode of sleep paralysis.[3] The next report of cases of sleep paralysis came in 1876 from Mitchell,[4,5] who referred to them as cases of "nocturnal hemiplegia," "night palsy," "nocturnal paralysis," and "sleep numbness." Other authors have called this state of consciousness "delayed psychomotor awakening"[6] or "cataplexy of awakening,"[7,8] "postdormital chalastic fits,"[9] "hypnapompic or hypnagogic hallucinations,"[10] and "predormital or hypnagogic sleep paralysis and postdormital or hypnapompic sleep paralysis."[11] Wilson, in 1925,[12] first used what has become the most accepted term for this state of consciousness: sleep paralysis. He noted that it was a "transient physiologic disorder, a physiological cataplexy."[12]

The great majority of cases of sleep paralysis have been associated with cases of narcolepsy and the incidence of sleep paralysis in patients with narcolepsy has been reported at 24 percent by Yoss and Daly,[13] 8 percent by Levin,[14] and 18 percent by Goode.[15] However, there has been increasing recognition that sleep paralysis has occurred as an isolated entity and not in association with narcolepsy or cataplexy.[12,16-18] Schneck reports 14 cases of isolated sleep paralysis in his extensive writing on the subject.[19-22] Two surveys were done to ascertain the incidence of sleep paralysis in a generally healthy population.

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Goode[15] studied four populations: his group I consisted of 163 medical students; group II, 68 junior and senior medical students; group III, 53 nursing students; and group IV, 75 hospital private inpatients (67 medical and 8 open-ward psychiatric). He found that the incidence of sleep paralysis in the four groups was 6.1, 7.8, 0, and 2.7 percent, respectively. His total population was 359 individuals with 17 (4.7 percent) having experienced sleep paralysis (one of whom reported narcolepsy). Of these 17 subjects, 5 had experienced sleep paralysis only once, 8 had less than six episodes per year, and 4 had approximately one a month. Eighty percent of his population was male. Everett[23] studied a first-year class of 52 medical students and found that seven of the 47 men and one of the five women reported having had isolated sleep paralysis (a total of 15.4 percent). Finally, there is the report of two families who had a severe form of isolated sleep paralysis.[24] In the first family 14 persons over four generations suffered from sleep paralysis, and in the second family four persons over three generations were affected. This finding supports the heredofamilial trend that Goode[15] noted in some of the subjects he studied.

In the majority of case histories reported and in both surveys in generally healthy populations, the issue of ethnicity was not addressed. However, considering that the surveys focused on the early 1960s medical and nursing student populations of Duke Medical School and School of Nursing, the University of Oklahoma Medical School, and the Johns Hopkins School of Medicine, it is highly unlikely that blacks were present in significant numbers. In fact it is likely that the two populations surveyed for the incidence of isolated sleep paralysis were predominately white.

The present study marks the first survey measuring the incidence of isolated sleep paralysis in a black population of generally healthy subjects and psychiatric patients.

METHODS

In response to the lack of basic psychiatric research on the psychodynamics of black populations, the Community Mental Health Council (CMHC), Chicago, began a research project to qualify and quantify the states of consciousness experienced by control subjects, who had never suffered a mental illness; precare patients, who had been treated for a mental illness only as outpatients; and aftercare patients, who had received both inpatient and outpatient treatment for mental illness. The control subjects were selected from the research team's nominations of individuals known to them personally, and the precare and aftercare subjects were taken from volunteers from the CMHC population of patients.

A structured interview was designed in which a trained interviewer read the definition of sleep paralysis to the research subjects. Next, the subjects were asked to decide whether they had experienced this state of consciousness and, if so, to tell the interviewer about it. Subjects who had not experienced sleep paralysis were asked whether they had ever heard of it, or knew someone who had experienced it. This part of the questionnaire was designed to ensure that the subjects understood the nature of the state of consciousness being investigated and that their reported experience matched the definition given. Once it was established that the subject had experienced the defined state, a standard series of questions was asked to determine whether this state could be self-induced or self-terminated, whether it could be induced by others or terminated by others, or whether the subject had any control whatever over the state of isolated sleep paralysis. Subjects were asked whether they wanted to experience this state more often and to explain why they did or did not wish to do so. They were asked how often this state occurred and how long it lasted. Subjects were asked whether this state made them comfortable or uncomfortable, and how; whether it ever got them into trouble or difficulty, and how; and whether it ever helped them to cope, and how. Finally, they were asked whether this state of consciousness had changed with age, and, if so, how it had changed.

In addition to this structured states-of-consciousness interview, the subjects were given objective written tests (Holmes-Rahe Social Readjustment Scale, Saafir Stress-Anxiety Diagnostic Scale, and the Million Clinical Multiaxial Inventory (MCMI). The interviews were audiotaped and reviewed by the principal investigator to check for

TABLE 1. EPISODES OF SLEEP PARALYSIS

	Control (%)	Precare (%)	Aftercare (%)	Total (%)
None	22 (61.1)	18 (50.0)	24 (66.7)	64 (59.3)
Less than one yearly	7 (19.4)	4 (11.1)	6 (16.7)	17 (15.7)
One or more yearly	5 (13.9)	8 (22.2)	2 (5.6)	15 (13.9)
One or more monthly	2 (5.6)	4 (11.1)	2 (5.6)	8 (7.4)
One or more weekly	0 (0.0)	2 (5.6)	2 (5.6)	4 (3.7)
Total	36 (100)	36 (100)	36 (100)	108 (100)

χ^2 analysis—not significant.

consistency in the interviewing technique and validity of the subjects' reports. In addition to being asked about their experience with sleep paralysis, subjects were also asked about their experience with dreaming, deep sleep, daydreaming, repressed memory, trance, meditation, internal scanning, regression, fragmentation, coma, stupor, intoxication, lethargy, hyperalertness, frenzy, rapture, and expanded states of consciousness.[10] The data were tabulated and certain correlations became evident.

RESULTS

It was discovered that of the total population of 108 black subjects, 44, or 41 percent, had experienced at least one episode of isolated sleep paralysis. This group was composed of 14 of 36 controls (39 percent), 18 of 36 precare subjects (50 percent), and 12 of 36 aftercare subjects (33.3 percent) (Table 1). Owing to the bulk of data and results, the findings on the other states of consciousness will be reported in a future paper.

It was of interest to find that isolated sleep paralysis was correlated with frenzy in the aftercare subjects ($r = .307$, significant at the .06 level) and was negatively correlated with meditation in the controls ($r = -.319$, significant at the .058 level).

Holmes-Rahe Social Readjustment Scale scores revealed that 34.3 percent of the total population had scored above 300 (indicating a major life change) and that only 19.4 percent had scored below 149, indicating very little life change during the year prior to the survey (Table 2). Subjects with sleep paralysis had an average Holmes-Rahe score of 310 (n = 44) and those without sleep paralysis had average scores of 248 (n = 63). Control subjects with sleep paralysis had average Holmes-Rahe scores of 303 (n = 14), as compared with controls without sleep paralysis, who had average scores of 230 (n=22). A similar trend was seen in precare subjects, revealing that those with sleep paralysis had average scores of 327 (n = 18) and those without it had average scores of 205 (n = 18). Aftercare subjects with sleep paralysis had average Holmes-Rahe scores of 292 (n=12) and those without it had average scores of 297 (n = 24). The correlation coefficient between sleep paralysis and Holmes-Rahe scores was significant at the .01 level ($r = .398$) in precare subjects, but was not significant in control and aftercare subjects.

Results of the Saafir Stress-Anxiety Diagnostic Scale revealed that none of the control, six of the precare, and 13 of the aftercare subjects had scores above 100, indicating clinically significant stress and anxiety. The MCMI disclosed no single particular personality pattern present in the subjects who had experienced sleep paralysis. When

TABLE 2. HOLMES-RAHE SOCIAL READJUSTMENT SCALE SCORES[29]

Score Range	Interpretation	Control (%)	Precare (%)	Aftercare (%)	Total (%)
300+	Major life change	11 (31.6)	14 (38.9)	12 (33.3)	37 (34.3)
250-299	Serious life change	2 (5.6)	4 (11.1)	8 (22.2)	14 (13.0)
200-249	Moderate life change	8 (22.2)	2 (5.6)	5 (13.9)	15 (13.9)
150-199	Mild life change	6 (16.7)	9 (25.0)	6 (16.7)	21 (19.4)
0-149	Very little life change	9 (25.0)	7 (19.4)	5 (13.9)	21 (19.4)
Total		36 (100)	36 (100)	36 (100)	108 (100)

χ^2 analysis—not significant

considering sleep paralysis and the Saafir scores in the precare subjects r was .331, which is significant at the .06 level. Sleep paralysis and the MCMI symptom scale scores of hypomania were found to be associated in the control subjects ($r = .324$, significant at the .06 level).

Of the total population, 52 were men and 56 were women. Their average age was 31.3 years and average education was 13.8 years. Twenty-three were living with spouses (21.3 percent). Isolated sleep paralysis had been experienced by 34.6 percent of the total male population and by 46.4 percent of the total female population. Of the 44 subjects who had experienced sleep paralysis, 18 (40.9 percent) were male and 26 (59.1 percent) were female. In the comparison of the ages of the control, precare, and aftercare subjects, it was found that each of these three groups had an average age in the early 30s, and in the consideration of the presence or absence of sleep paralysis within the three groups as it related to age, the same pattern was manifested. In the comparison of educational levels, it was found that the control subjects had an average of 15.4 years of formal education, the precare subjects had an average of 13.6 years, and the aftercare subjects had an average of 12.5 years. Controls with sleep paralysis and controls without sleep paralysis had similar educational levels, and this also was true for both the precare and aftercare populations. Of the control subjects 41.7 percent were living with a spouse, compared with 11.1 percent of the precare and aftercare subjects; it did not appear, however, that marital status was related to the absence or presence of sleep paralysis.

DISCUSSION

Of the total population of 108 black subjects, 44, or 41 percent, had experienced at least one episode of isolated sleep paralysis. The conviction with which the vast majority of subjects who had experienced sleep paralysis responded (many with the "Aha!" experience), when asked about this state of consciousness, gave a strong subjective impression about the validity and reliability of their reports. Unfortunately, the incidence of isolated sleep paralysis in white precare and aftercare patients has never been studied, so that the authors are unable to compare the incidence of sleep paralysis in these two groups with the prevalence of sleep paralysis in this study's precare and aftercare populations (50 and 33.3 percent, respectively). However, the literature gives the distinct impression that white precare and aftercare populations do not experience sleep paralysis nearly so often as did the black patients in this study; there are fewer than 100 cases reported in American psychiatric literature from 1842 to the present. The literature[15,23] does report the incidence of isolated sleep paralysis in a presumably "normal" or control white population as being between 4 and 15

percent, whereas this study's control population had isolated sleep paralysis occurring in 14 of 36 black subjects (39 percent)—at least twice the incidence reported in whites. This finding should be thoroughly investigated by sleep paralysis surveys in control, precare, and aftercare white and black populations. Should the results of the present study be verified, the psychiatric and neurologic professions will be confronted by a puzzle: "What causes blacks to have an incidence of isolated sleep paralysis at least two times that in whites?"

The mechanism for sleep paralysis is at present unknown; however, the work on the sleep-dream cycle by Hobson et al[25] and McCarley and Hobson[26] may hold some answers. Those authors maintain that an interaction between the neurons of the raphe and locus ceruleus (aminergic level-setters) and the neurons of the gigantocellular tegmental field (cholinergic generators) of the pontine brainstem is the mechanism for sleep-dream cycle generation. In awake consciousness, perception is vivid and externally generated, thought is logical and rational, and movement is voluntary with high muscle tone. The electroencephalographs (EEGs) taken during this state show a desynchronized pattern of low-voltage fast waves; the eye movements are voluntary. In the awake state the aminergic, locus ceruleus neurons are maximally active whereas the cholinergic, giant pontine neurons are minimally active. In deep sleep perception is dull or absent, thought is repetitious or absent, and movement is infrequent, but possible, with medium muscle tone. EEGs taken in this state show a synchronized pattern of high-voltage slow waves; the eye movements are slow. In the deep-sleep state the aminergic locus ceruleus neurons have diminished activity, whereas the cholinergic, giant pontine neurons become progressively more active. In dreaming, perception is vivid and internally generated, thought is illogical and delusional, and movement is impossible with muscle tone absent (ie, the body is paralyzed). EEGs taken during this state show the desynchronized mode; eye movements occur automatically and rapidly, without relationship to the visual field. In this state the aminergic, locus ceruleus neurons are minimally active, whereas the cholinergic, giant pontine neurons are maximally active. Sleep paralysis is a state of consciousness characterized by a dysrhythmia of the sleep-dream cycle, in which the aminergic locus ceruleus neurons are maximally active, and the cholinergic, giant pontine neurons are also maximally active. This would account for perception being vivid and both internally and externally generated, thought being logical and rational, but at the same time being panicky and delusional, and movement being impossible with absent muscle tone. The EEG in this state shows a pattern of low-voltage mixed frequency waves with bursts of rapid eye movements that is characteristic of dreaming sleep.[27] Implicated in the etiology of sleep paralysis because of its role in the sleep-dream cycle, the locus ceruleus is also involved because stimulation of the locus ceruleus has been found to be associated with fear responses[28]—the essential emotional response in sleep paralysis.

If blacks experience significantly more sleep paralysis than whites and the etiology of this prevalence of isolated sleep paralysis lies in an overactive locus ceruleus, what would cause blacks to have a more active locus ceruleus than whites? The answer to this question may lie in part in the findings of Vander Heide and Weinberg,[18] who found a connection between sleep paralysis and combat fatigue. Recent work suggests that post-traumatic stress disorder in Vietnam veterans involves an abnormal arousal of the central adrenergic system, and that these disorders respond to adrenergic blockers such as clonidine, which blocks the $\alpha 2$-adrenergic receptors in the locus ceruleus. The post-traumatic stress syndrome is accompanied by frequent episodes of frenzy and is manifested by hyperalertness or an exaggerated startle response, explosiveness, nightmares, intrusive thoughts, and sleep disturbance. Consideration of this information suggests that there may be a connection between the frequency of sleep paralysis, the frequency of frenzy, and the presence of stress. The association of sleep paralysis with hypomania in the controls, with life changes and stress-anxiety in the precare subjects, and frenzy in the aftercare subjects supports this hypothesis, but other factors that mediate the response to life changes and stress must be taken into account by further analysis of the data to confirm or refute this hypothesis. For example, states of meditation, which have been shown to lower cortical and autonomic arousal during a wakeful state, were demonstrated to have been negatively correlated with sleep paralysis in the

controls. Certainly all three groups, as indicated by the Holmes-Rahe scores, had undergone considerable life change over the year prior to the study.

The interpretation of the scores for the total population indicates that 34.3 percent will be susceptible to a major illness within the coming year, 13 percent will have lowered resistance to diseases over the next year, and 13.9 percent are susceptible to depression in the year following the study.[29] Also, although the incidence of sleep paralysis among the three populations (39 percent in control, 50 percent in precare, and 33.3 percent in aftercare subjects) was not found to be statistically different, the difference in the incidence of frenzy reported among the three groups was statistically significant.[30] Since all three groups experienced basically the same degree of life change in the year preceding the survey, and yet showed a significant difference in their reports of frenzy, there may be a difference in how the control, precare, and aftercare populations cope with change. This difference may influence the manifestations of stress such as sleep paralysis or frenzy.

The results presented thus far support the previously published concept[31-33] that being black in this society is associated with stress due to racism with its attendant lack of parity in housing, health care, employment, nutrition, education, and opportunity. This stress is best described, not as a "traumatic neurosis"[34] (or, as when the stress occurs during battle, "combat fatigue") but as "survival fatigue," stress that is the result of the tremendous effort necessary to survive in a harsh environment. Further, a great number of black persons suffer from "survival fatigue," a situational predicament that may result in an overaroused adrenergic central neurobiologic system, leading to a variety of behaviors, attitudes, and psychophysiologic diseases. It may well be that the high amounts of sleep paralysis seen in this population are the results of "survival fatigue," and this may also hold true for the amounts of frenzy reported.

With the exception of the surveys done by Goode[15] and Everett,[23] previous cases of isolated sleep paralysis have been reported in case history, anecdotal fashion, and such occurrences were psychoanalytically interpreted as an actualization of a passive-aggressive conflict present in the patient's personality makeup. The patient having an episode of isolated sleep paralysis wanted to move (be aggressive), but was unable to move (remained passive). Vander Heide and Weinberg[18] noted in their 12 patients the indecisiveness and lack of goal-directed behavior as well as the aggressive intent but docile action. Schneck[35] also noted a parallel between sleep paralysis and conflicts between active, aggressive functioning and inactive, passive behavior in a number of cases he treated. Payn[36] reported a case of a black woman who had aggressive feelings she needed to inhibit because of guilt and who, thus, remained passive. After the patient's sleep paralysis was interpreted as a sign of an inhibition against hostility, the patient is reported to have improved. Levin[37] discusses sleep paralysis and cataplexy from the standpoint of conditioned inhibition. The stereotypes held of blacks (regarding their rage and aggressiveness and their need to keep these forces in check and remain passive[38]) may, then, offer a way of explaining why blacks have a prevalence of isolated sleep paralysis. However, the results from the MCMI personality profiles suggest that no single personality type or intrapsychic conflict underlies sleep paralysis; the profiles revealed a wide range of personality types.

In addition to psychoanalytic factors being implicated in the occurrence of isolated sleep paralysis, the reports of both Goode[15] and Roth[24] indicate heredofamilial predisposition. It is conceivable that blacks, in addition to being at high risk for having excessive life change, have a genetic predisposition to high amounts of sleep paralysis possibly because of a more sensitive central adrenergic system. One finds cultural evidence for the high incidence of sleep paralysis in blacks in American black folklore, with references to the experience "the witch is riding you." This may refer to the common report of sleep paralysis victims that they feel as if someone is sitting on their chest or standing over their bed. Certainly a genetic predisposition toward sleep paralysis among blacks would help to explain the finding that black cultural cosmology in black Africa[32] is in part based on the existence of genies and spirits. It may be that the prevalence of sleep paralysis in blacks is an indication of a generally greater access to various states of consciousness, as there is some evidence that North Africans have more daydreams than Westerners.[39]

More investigation into these issues is needed,

and indications are that such inquiry will be fruitful.

PROPOSALS

The hypothesized neurologic model for sleep paralysis could be tested by animal studies using electrodes to stimulate the locus ceruleus, while doing sleep studies on the subjects. An overstimulated locus ceruleus etiology for sleep paralysis could also be confirmed in a study of patients who are withdrawing from opiates, as it has been shown the withdrawal syndrome is produced by excess locus ceruleus activity,[40] raising the possibility of high amounts of sleep paralysis in patients withdrawing from opiates. This hypothesis could also be tested by the discovery that patients with frequent attacks of sleep paralysis could be treated with clonidine, an antihypertensive medication that has been shown to reduce overstimulation of the locus ceruleus neuropharmacologically through agonistic α2-adrenergic activity. It is of interest that tricyclic antidepressants have been used to treat sleep paralysis[27,41-43] and panic attacks,[44] as well as to decrease locus ceruleus arousal[45] (possibly by preventing adrenergic neurotransmitters from re-entering the nerve cells of the locus ceruleus nucleus). There is evidence that there is involvement of a brain norepinephrine system in the production of anxiety,[46] and a study of sleep paralysis may well be the vehicle by which to confirm this hypothesis. The finding that hypnosis is useful in treating sleep paralysis[47] is not surprising. The present study revealed that sleep paralysis and meditation were negatively correlated and it may be that both hypnosis and meditation can be used as nonpharmacological treatments, lowering cortical and autonomic arousal with an accompanying decrease in locus ceruleus activity.

The study of the incidence of isolated sleep paralysis in both white and black patients with posttraumatic stress syndrome may help delineate whether the etiology of sleep paralysis is rooted in a difference in the sensitivity of the central adrenergic systems of blacks as compared to whites, or in the difference between the amounts of stress that blacks and whites experience from day-to-day living in America. The finding of sleep paralysis in aftercare patients may indicate an overaroused adrenergic central system, which is superimposed upon some more grave neurochemical disorder responsible for the aftercare patient's fragmentation. Perhaps the treatment of these overaroused central adrenergic systems might benefit those patients who are fragmented, but who also report anxiety (the current trend being to treat only the patient's fragmentation with antipsychotics and to hope the anxiety will abate as the psychosis abates).

The significant possibility that sleep paralysis indicates a difference in the neurobiology of whites and blacks should be proven and properly interpreted. A racial difference in neurochemistry may have great influence on how racial groups respond to medication or drugs, and indeed this has been demonstrated in the ways in which different racial groups respond to the pharmacokinetics of lithium[48] and alcohol.[49,50]

The possibility that sleep paralysis may be an early warning sign for the development of an overaroused central adrenergic system should be considered as it may be a useful clinical marker of a patient's potentially developing other proposed hyperadrenergic syndromes such as states of frenzy, post-traumatic stress syndrome, and hypertension.

Finally, there should be consideration of the possibility that sleep paralysis (often accompanied by hypnogenic hallucinations) may indicate that blacks have more of these types of hallucinatory experiences than whites and are more predisposed to the hallucinatory phenomena reported in the literature.[51,52] This difference may be in part responsible for the misdiagnosis of black patients with manic depressive illness[53,54] or other disorders.[55]

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