CASE REPORTS

Sleep paralysis: a study in family practice

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SUMMARY. Over a period of two years, five patients with sleep paralysis referred themselves to four family practices in Israel serving a population of 6800. None of the patients suffered from daytime sleep attacks or cataplexy and all were from the oriental (sephardi) community. The two who were tissue typed had HLA haplotypes different from those which are exclusively associated with narcolepsy and one of them who also underwent polysomnography had a normal tracing. There was considerable delay in consulting a physician despite the physical and mental anguish caused by the disorder and some improvement was noted once the diagnosis was explained. The serious nature of the components of the differential diagnosis — myocardial infarction, seizure disorder, cardiac arrest, anaesthetic accident — makes it important that sleep paralysis be more widely recognized.

Introduction

SLEEP paralysis is a phenomenon 'the recognition of which still eludes most physicians'. It has long been regarded, along with sleep attacks, cataplexy and hypnagogic or hypnopompic hallucinations, as part of the narcolepsy tetrad and a textbook published as recently as 1987 states that it may be the sole manifestation of the disorder in as many as 5% of narcoleptics. A growing literature deals with what is now called isolated or independent sleep paralysis in otherwise normal individuals. Indeed, the original description by Mitchell in 1876 related to its occurrence in two healthy young men.

During an episode of sleep paralysis, which can last from a few seconds to minutes, the sufferer is powerless to move and may not be able to speak or even to open his eyes, although he can recall every detail of his surroundings and what happens there. Extreme anxiety often accompanies the attack and breathing difficulty, sweating, palpitations, a sense of tingling or buzzing spreading throughout the body, shrill hissing or roaring sounds in the ears, a feeling of suffocation and visual or auditory hallucinations have all been described. Eye witnesses have observed unresponsiveness on the part of the patient to verbal command or noxious stimuli, a glassy-eyed stare, irregular respiration with periods of apnoea, sighing and tachypnoea, flaccid paralysis, areflexia and the emission of faint moans or gasps. An attack may be mistaken for post-anaesthetic depression, a nocturnal seizure disorder, cardiac arrest or myocardial infarction. There are usually no sequelae, but numbness of the extremities and exhaustion have been reported and, if the patient does not immediately rouse himself, he may be subject to a second episode.

This study was based in four family practices in Israel, two each from towns 200 km apart. The practices have a patient population of 6800. Over a period of two years five sleep paralysis sufferers were seen in these practices. All the patients attended because of the physical and mental anguish the attacks caused and to receive a diagnosis. In most instances, diagnosis led not only to alleviation of anxiety but also to a lessening of symptoms. The details of these cases are reported here.

Case reports

Case 1

M.M., a 36-year-old unemployed man, married with no children attended his family doctor because he had had a number of frightening attacks of paralysis in all parts of his body. These invariably occurred in the morning as he was waking. At times he was aware of his wife getting out of bed to go to work but was unable to communicate with her and once he heard someone knocking on his door but could not respond. He estimated that each episode lasted several minutes. His overall health was poor as he suffered from myotonic muscular dystrophy and had frequent paroxysms of atrial fibrillation that were difficult to control. His physician was familiar with sleep paralysis and the patient seemed reassured by the explanation of his condition. He has since been lost to follow up.

Case 2

Y.Y., a 29-year-old mechanic, married with three children, visited his family doctor seeking a referral to a psychologist. He reported at least 10 episodes of paralysis occurring when he was about to fall asleep at night. Each was brief, and his wife was unaware of the problem because if she spoke to him during an attack he could answer with only a slight delay. He emphasized that he could both see and hear everything going on about him but was utterly powerless to respond. The paralysis was accompanied by excessive sweating, palpitations and, on recovery, by a sense of having had a narrow escape from a dreadful event. A few seconds before each episode he experienced a feeling of pressure in the head.

The first attack occurred during the patient's compulsory military service when he was 19 years old. The army doctor told him that it was a reaction to stress and other doctors with whom he discussed the problem urged him to see a psychologist. This worried him because he considered himself to be a calm and dependable person. After the diagnosis of sleep paralysis was explained to him he had no further attacks. Most of his subsequent visits to the doctor were in connection with his children but he continued to be anxious about his mental health and frequently mentioned the subject.

Case 3

R.Y., the 27-year-old brother of case 2, married with two children, attended his family doctor reporting frequent attacks, similar to those experienced by his brother. He had been having these attacks for 11 years but they had started to occur every evening

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as he was falling asleep and he had become concerned that they might indicate a heart condition. At earlier consultations he had been told that his complaint was due to emotional stress. On this occasion he presented the facts in a somewhat hostile tone, warning his doctor not to waste her time 'looking for psychological problems'. He was unaware, as was his brother, of a family history of sleep paralysis and was relieved to learn that the disorder had no serious implications for his future health. He refused any further examination. When asked why he was so anxious to keep others in ignorance of his condition he implied that he perceived it as a slur on his manhood.

His doctor suggested that he attempt to move his eyes from side to side during an attack. He reported some time later that he had had only three more episodes, that the proposed manoeuvre appeared to help and that he felt he could now live with the problem.

**Case 4**

S.E., a 29-year-old woman, married with two children, consulted her family physician because she had had eight attacks of sleep paralysis over a period of two months, generally when she was watching television in the evening and had begun to doze off. On one occasion her husband had witnessed the episode and thinking she had fainted doused her with cold water. She had hesitated about coming to the clinic because she feared for her sanity and was certain that nobody would believe her. She volunteered that she was under considerable stress. Her oldest child, a boy, had been deaf from birth and there being no family history of hearing disorders, a consultant in genetics to whom she was referred in connection with a planned third pregnancy estimated that the chance of a second male child being born deaf was around 15%.

The patient expressed a feeling of relief when the doctor explained her condition but over the next few weeks matters worsened and she experienced so many episodes that she feared going to sleep. She readily agreed to undergo tests in a sleep laboratory, and the results, as expected, were normal. When last seen, the patient reported that her attacks occurred much less often.

**Case 5**

M.E., a 13-year-old girl, came to the clinic with her mother who was anxious about a breathing difficulty reported by the girl on two occasions. There was nothing in the patient's history to suggest heart or lung disease and the physical examination was within normal limits. Further questioning revealed that the difficulty occurred in the early hours of the morning and was not associated with sleepness in the usual sense but rather, as the patient put it, 'I seemed not to be breathing'. The mother had not witnessed either of the attacks and when her daughter was asked why she had not called out she replied that she had been unable to move or utter a sound. The phenomenon was explained as a benign disorder of the sleep-wake cycle and mother and daughter left the clinic reassured.

**Discussion**

These case histories shed some light on the pathogenesis of sleep paralysis. First, none of the patients suffered any other manifestation of narcolepsy and a complete examination at a sleep laboratory for case 4 revealed nothing untoward - the absence, on polysomnography, of a period of rapid eye movement within 10 minutes of going to sleep virtually excludes the diagnosis of narcolepsy. Furthermore, narcolepsy is rare in Israeli Jews and is associated exclusively with the HLA DR2/DRW2 haplotype. Cases 4 and 5 underwent tissue typing and the relevant antigens were DR1; DR7/DRW53 and DR9; 8/DRW13 respectively. Thus, the clinical histories, polysomnographic findings and tissue types suggest that sleep paralysis may be an independent phenomenon that does not always belong to the narcolepsy tetrad.

Secondly, in cases 1–4 anxiety could be regarded as a predisposing factor to the attack, a concomitant of it and, possibly, one of its outcomes. Stress is emphasized in the survey by Bell and colleagues among black patients with sleep paralysis, while Penn and colleagues, interviewing medical students, found that arguments and frightening films could bring on an attack. Ness, studying the old hag phenomenon in Newfoundland, an experience he equates with sleep paralysis, postulates a connection between an episode and an intense emotional upset; he adds fatigue, hasty eating and the supine position as other possible triggers. Fukuda and colleagues, investigating a phenomenon called kanashibari in Japan, which is symptomatically identical to isolated sleep paralysis, found that half of those who had experienced it reported fatigue, psychological stress, irregular life pattern and sleep loss as predisposing factors. In folklore sleep paralysis represents a hex or voodoo spell, or is caused by evil spirits.

Thirdly, cases 2–4 showed improvement after the phenomenon was properly interpreted by a physician. This suggests that a self-perpetuating cycle of anxiety and attack was broken by the explanation. Hishikawa, in an extensive review of sleep paralysis, discusses treatment only in relation to the narcolepsy tetrad while Spector and Bourke, reporting their experience in an anaesthesiology practice, state that intravenous physostigmine can terminate an 'isolated' attack within one minute. Bell and colleagues, who note a strong association between sleep paralysis and panic attacks in black patients, describe successful treatment of both disorders with tricyclic antidepressants. Other suggested therapies include a light sensory stimulus applied by a witness, an attempt by the victim to move his eyes from side to side or 'going along with' the paralysis in the knowledge that it is self-limiting. Folk cures are also reported and they are as varied as the beliefs in supernatural causes.

Fourthly, most of the patients described here waited a long time before consulting a physician, having concluded that the disorder was a serious psychological disturbance. Bell and colleagues remarked that many of their subjects never discussed their condition with others in case they were thought 'crazy' and cases 2 and 3 were unaware of one another's problem despite being brothers. Similar fears were expressed by respondents in the surveys carried out by Everett and Ness and the extent of the repression is indicated by Schneck's finding that sufferers who have never mentioned their condition before will always bring it up when subjected to hypnosis for the first time. Thus, the impression that sufferers do not consider the problem a medical one and fail to consult a doctor about it may stem from their anxiety about the nature of the condition.

Finally, all five patients in this study belong to the oriental (sephardi) community, although the ratio of ashkenazi to sephardi patients attending the four practices is 2:3. Lavie and Peled, who believe that they have identified virtually all the Jewish narcoleptics in Israel where the disorder is between 60 and 160 times less common than in western countries, report that four of the six narcoleptics are ashkenazi and two sephardi. This again suggests that the cases reported here represent a separate disease entity. Bell and colleagues' study among American blacks, the work of Ness among Newfoundlanders whose ancestors emigrated from a defined area of the United States, and the work of Lavie and Peled among Israeli Jews suggests that sleep paralysis may be a distinctly Jewish phenomenon.
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