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Does Recurrent Isolated Sleep Paralysis Involve More Than Cognitive Neurosciences?

JEAN-CHRISTOPHE TERRILLON

Human Information Processing Research Laboratories (HIP),
Advanced Telecommunications Research Institute International (ATR),
2-2 Hikaridai, Seika-cho, Soraku-gun, Kyoto, Japan 619-0288
e-mail: terril@hip.atr.co.jp or terrillon@softopia.prof.gifu.jp

SIRLEY MARQUES-BONHAM

Austin Community College
512 Kingfisher Creek Dr., Austin, TX 78748

Abstract—Isolated sleep paralysis (ISP) is a poorly understood phenomenon that has attracted increased attention in recent years both in the medical community (Dahlitz & Parkes, 1993; Hishikawa & Shimizu, 1995) and in psychological research (Fukuda et al., 1987, 1991; Fukuda, 1993; Takeuchi et al., 1994; Wing et al., 1994). Although the occurrence of ISP is relatively common, recurrent ISP (RISP) is a rarer variant of sleep paralysis characterized by frequent episodes or a complex of sequential episodes whose total duration may exceed 1 hour, and particularly by the range and sense of perceived reality of the subjective phenomena experienced during episodes. Although such phenomena are usually categorized as hypnagogic or hypnopompic hallucinations, there is at present no integrated model that adequately explains the ensemble of physiological, neurological, cognitive, and psychological components of RISP. As researchers who experience RISP, in this paper we attempt to synthesize information on RISP gathered from various sources and to conjecture possible connections between RISP and other as yet poorly understood phenomena at different levels: at a neurophysiological level, with anxiety disorders (Suarez, 1991; Paradis et al., 1997), the Periodic Paralyses (Stedwell et al., 1992), and with Sudden Unexplained Nocturnal Death Syndrome (SUNDS) (Nimmanit et al., 1991; Randall, 1992; Adler, 1995); at a neurocognitive level, with lucid dreaming (LaBerge, 1985); and finally, at a level that examines the possibility of the manifestation of paranormal phenomena during RISP episodes, with Out-of-Body Experiences (OBE) (Tart,1968; Osis, 1981) and with Near-Death-Experiences (NDE) (Moody, 1976; Ring, 1979). Finally, we performed a statistical analysis on RISP by use of a sample of 250 direct or indirect respondents to a message posted initially by one of the authors on the sleep web site of the University of California in Los Angeles (UCLA).[1] Preliminary results indicate that over 90% of respondents experience intense fear during their RISP episodes, about 50% have invoked a paranormal or supernatural explanation, and that a typical RISP episode may be described as consisting of three main phases. We conclude with a brief description of possible strategies to cope with RISP.

Keywords: sleep paralysis — hypnagogic/hypnopompic hallucinations — periodic paralyses — lucid dreaming — out-of-body experience

1. Introduction

The phenomenon of sleep paralysis has attracted increasing attention in the scientific community only in recent years, even though the occurrence of what may be described as sleep paralysis has been documented as early as Hellenistic times. At a fundamental level, the term "Isolated Sleep Paralysis" (ISP) is used to differentiate between an abnormal generalized muscle atonia and the usual generalized muscle atonia which is characteristic of REM sleep (dream state), as well as to distinguish such a condition from sleep paralysis occurring as an ancillary symptom of narcolepsy (which exhibits a combination of several symptoms). ISP is a common condition with a prevalence of 5–62% (Dahlitz & Parkes, 1993) depending on geographic location, but most affected people have a single episode or infrequent episodes during their life and the duration of an episode is generally less than a minute. In contradistinction to ISP, Recurrent Isolated Sleep Paralysis (RISP) is a rarer variant of sleep paralysis characterized by frequent episodes or a complex of sequential episodes of generally longer duration, and in particular by the range and intensity of the perceptual phenomena occurring during episodes. RISP is found to be familial in most cases (Dahlitz & Parkes, 1993; Roth, Buuhova, & Berkova, 1968). RISP is a complex phenomenon that, at present, cannot be explained by any appropriate and comprehensive model that would integrate low-level neurophysiological processes, high-level neurocognitive functions, and possibly higher level processes. Indeed, at a higher level, to our knowledge there has been until now no research that has thoroughly addressed the content and significance of the perceptual phenomena induced by RISP. Most psychological studies have rather focused on the symptomology of RISP (e.g., circumstances of occurrence, type of sensory experience; Roth, Buuhova, & Berkova, 1968), on the emotional reactions of the affected persons to their episodes (Roth, Buuhova, & Berkova, 1968), or on their psychological profile (Fukuda et al., 1991).[2] As researchers who experience RISP, in this paper we attempt to summarize information on RISP gathered from various sources and the possible connections between RISP and other poorly understood conditions at different levels. Most importantly, we address some "hard questions" relating to the range and sense of reality of the perceptual phenomena inherent to RISP and offer some conjectures as to the causes of such phenomena that include paranormal components such as Out-of-Body Experiences (OBE; Tart, 1968; Osis, 1981).

The paper is organized as follows: in Section 2, we present the profile of a typical RISP episode (or a complex of sequential episodes). This profile has been constructed by use of a sample of 250 direct or indirect respondents to a message posted initially by one of the authors (J.C.T.) on the sleep web site of the University of California in Los Angeles,[3] and is based as well on the authors'

personal experiences. This initial description of RISP from a phenomenological viewpoint will help us, in the following sections, to use a bottom-up approach in describing the neurophysiological aspects of RISP and its possible connections with other conditions in Section 3, the neurocognitive aspects of RISP invoking hypnagogic/hypnopompic hallucinations and lucid dreaming in Section 4, and the possibility of the manifestation of paranormal phenomena (OBE) in Section 5. In Section 6 we present the preliminary results of a statistical analysis of the RISP experiences of the above-mentioned sample population. This analysis takes into account the emotional reactions of the respondents as well as their beliefs as to the cause of their experiences in the context of a more general socio-historical study of RISP. In Section 7, we briefly describe possible strategies that could be used to cope with RISP, involving both "conventional" medical treatments and a "psychological" approach. We conclude the paper in Section 8 with a summary of important issues that should be addressed in future research on RISP.

2. Phenomenology: Profile of a Typical RISP Episode

RISP is experienced while drifting to sleep or upon waking from sleep, and most commonly while lying on one's back. At the beginning of an episode, one feels progressively heavier and heavier as the generalized muscle atonia sets in. This progressive heaviness can be perceived as successive "pulses" that affect the whole body. One suddenly realizes that it is impossible to move or to speak, or to cry out. A strong pressure on the chest gives the sensation that an entity is sitting on the chest. It becomes difficult to breathe. A buzzing/ringing/roaring/whistling/hissing/high-pitched screeching sound in the ears sets in and becomes louder and louder to the point of becoming unbearable. Occasionally a sensation of explosion in the head occurs (Oswald, 1988; Pearce, 1988, 1989; Sachs & Svanborg, 1991) that could be compared to a seizure (Schneck, 1982). A presence, often perceived as malevolent, is felt and eventually seen in the room of the sleeper. It may take a variety of forms, from a shadow to animals to a humanoid form. Occasionally it is trying to pull one's legs. Voices or even music may be heard vividly. Flashes of bright light may be seen. By that time, intense fear is experienced and an attempt to move a part of the body (such as a hand or leg or the head) is made in order to liberate oneself of the atonia. If such is the case, and if one can finally move, the same phenomena as described above may occur again, repeatedly, after one tries to go back to sleep. If no attempt is made to move, that is, if the fear is overcome or if it is mild, another complex of phenomena sets in: what seems to be a "phantom body" slowly slips away from the physical body. There seems to be a dissociation from the immobile physical body, and consciousness is perceived to reside within the phantom body. At that point, the immediate surroundings of the room may be "seen," sometimes vividly, by the phantom body, and a sensation of rising and/or floating, sometimes rolling, is experienced. The phantom body can "see" under itself the physical body lying motionless on the bed.

It can stay inside the physical room or proceed to other locations perceived to be part of the physical world (e.g., other rooms in the house, a corridor adjacent to the room, etc.). The surroundings appear to the consciousness as they would while in the wake state, eventually with some odd modifications (a few more objects than there should be, or a change in the configuration of some objects, for example), or, in another possible scenario, the phantom body may feel accelerated inside a "tunnel" that seems to appear out of nowhere: the tunnel usually appears to be elastic and dark. The phantom body then "arrives" in what could be described as an "oniric scene": rooms in a house, a landscape (a countryside, a garden, mountains, etc.) with vivid colors, or on streets in a city, for example. In such as state, the "consciousness" that resides within the phantom body has to a certain extent a volitional control of the scenery, the "story," and what the phantom body can do—walking, flying, floating, going through walls—but still has the sensation of touching and grasping objects in the scenery, etc. In some cases, an "elastic cord" connecting the phantom body to the physical body can be perceived and even "seen" by the phantom body. The phantom body may then reenter the tunnel and either proceed to another "oniric scene" or go back to the physical room and reenter the physical body. In both scenarios, the return to the physical body may be abrupt or slow. In the second case, the phantom body may "hover" for some time above the physical body before "reentering" it. The whole process may be repeated several times in the course of one night, in what may be described as a complex of sequential or "back-to-back" episodes, or it may occur only once. Therefore a complex of episodes may last from a few minutes up to 2–3 hours in some rare cases (although the perceived time usually seems significantly shorter). Upon awaking, if such a phenomenon has not been experienced before or if it has been experienced only very infrequently, one usually is extremely puzzled or troubled and is unable to find a rational explanation let alone a name for the experience.

3. Neurophysiological Aspects of RISP and Possible Connections With Other Conditions

As the preceding section shows, RISP is characterized by a complex ensemble of puzzling phenomena occurring simultaneously. In this section we attempt a scientific investigation of RISP by starting at the neurophysiological level: *at this level, what are the mechanisms that induce the generalized muscle atonia while one is conscious or semi-conscious and that could explain some aspects of at least the first phase of a RISP episode (up to the perceived dissociation of a phantom body)?*

We base our approach on the literature that has already been published on sleep paralysis in various medical journals and we attempt to show that at a fundamental physiological level, there exists a common denominator between ISP/RISP, normal Rapid Eye Movement (REM) sleep characteristic of the dream state, and other as yet not well understood conditions, mainly neuromuscular disorders.

Physiologically, electrolytes such as Potassium (K^+), Sodium (Na^+), Calcium (Ca^{++}), and Chloride (Cl^-) play an essential part in such bodily functions as nerve conduction and muscle contraction. Maintaining an appropriate electrolyte balance between the blood serum (or plasma) and the intracellular medium of muscle cells and nerve cells is essential for muscle activity. An ensemble of different types of proteins that are embedded inside the membrane of each cell acts as channels and thus regulates the circulation of electrolytes between the inside of the cells and the blood serum (Andredi et al., 1980; Alberts et al., 1994). Each channel is specific to a given type of electrolyte, e.g., the Calcium channel regulates the circulation of Ca^{++} ions exclusively, and the cell membrane is thus semipermeable and selectively permeable. Nerve conduction is essential to excite muscle cells into initiating muscle contraction and thus body motion. It involves mainly the K^+ and Na^+ ions, which are used to generate an action current and thus nerve conduction. At rest, nerve cells are said to be polarized : the Na^+-K^+ATPase pump maintains a difference of electrical potential between the blood serum and the intracellular medium, with the blood serum positively charged (+) with respect to the cytoplasm (-) (usually, the potential difference is about -90 [mV] as measured from inside the cytoplasm). The concentration of Na^+ ions is larger in the blood serum, while that of K^+ ions is larger in the cytoplasm. When nerve cell excitation occurs, neuromediators (or neurotransmitters) such as acetylcholine, noradrenaline (a precursor of the hormone adrenaline), and thiamine (vitamin B_1) modify the membrane permeability (e.g., the permeability to Na^+ ions increases considerably, allowing Na^+ ions to enter the cytoplasm via the Na^+ channel), generating an action current and thus a depolarization of the membrane (or depolarization wave), followed by a repolarization and return to the initial state. In turn, Ca^{++} ions, as well as K^+ and Na^+ ions, play an essential part in muscle cell function, which is sensitive to even very small fluctuations in electrolyte concentration in the blood serum and in the intracellular medium.

In general, electrolyte balance is achieved via a very complex interplay of factors involving the number of different types of electrolytes, proper channel activity and Na^+-K^+ATPase pump activity, hormonal activity, and finally neuronal activity mediated by neurotransmitters in the brain. An electrolyte imbalance may occur due to a variety of causes: fluctuations in hormone secretion and hence levels in the blood serum, a mutation of a gene coding an electrolyte-specific channel, or a variety of toxins that inhibit channel activity or the activity of the Na^+-K^+ATPase pump. An electrolyte imbalance occurs when the difference of electrical potential between the blood serum and the intracellular medium is modified via an abnormal shift in electrolyte concentration: In the case of muscle cells, typically, the cells are (and may remain) partially depolarized (e.g., to -50 [mV]), but sufficiently so to prevent membrane excitation by the action current transmitted to them by the nerves. As a result, muscle contraction is compromised and partial to complete muscle atonia occurs. In the case of nerve cells, and when considering REM sleep, the neural

activities producing the generalized muscle atonia that is typical of REM sleep originate mainly in the pons region of the brainstem and involve the neurotransmitter acetylcholine (Hishikawa & Shimizu, 1995), that is, cholinergic "REM sleep-on" neurons in the brainstem are activated, while monoaminergic (serotonin or noradrenalin) "REM sleep-off" neurons cease to fire (Sandyk, 1995). Motoneurons in the brainstem and in the spinal cord are inhibited and a profound atonia occurs, which results in an almost complete paralysis of striated muscles. The activity of brainstem serotonin "REM sleep-off" neurons is regulated by the hormone melatonin, discovered to be the principal hormone of the pineal gland in 1963. Qualified by Sandyk (1995) as a "master hormone" involved in the control of circadian rhythms and other biological functions, melatonin reaches its lowest plasma levels during REM sleep when the serotonin neurons, whose activity it regulates, are inhibited, thus suggesting a causal relationship between the inhibition of melatonin secretion during REM sleep and the development of REM sleep atonia. However, still little is known about the influence of the pineal gland on motor control (recently it was suggested that not only the pineal gland, but also the suprachiasmic nuclei are involved in circadian rhythms). As indicated above, REM sleep atonia is directly caused by inhibitory postsynaptic potentials in spinal motoneurons. The biochemistry of "REM-sleep on" and "REM-sleep off" neurons is still not well understood and largely under investigation, so that the exact coupling mechanisms with melatonin secretion involved during REM sleep that induce the muscle atonia are not clearly known. We may conjecture that a low concentration of melatonin in the blood serum would prevent the depolarization current to occur in the nerves and consequently prevent muscle cell excitation. But given the present state of knowledge on REM sleep, the role of melatonin in inducing the REM sleep atonia should not be overestimated. It should be mentioned that body paralysis during REM sleep is a normal, protective mechanism that prevents the sleeper from "acting out" his/her dreams.

Sleep paralysis (whether RISP or ISP) is a pathological condition in the sense that there is a marked dissociation between the level of alertness and the muscle atonia that often occurs in sleep onset REM sleep periods (SOREMPs; Hishikawa & Shimizu, 1995). In other words, essentially abnormal REM sleep conditions occur in that a certain level of alertness (or awareness) is retained despite the muscle atonia characteristic of REM sleep that would be induced by a low concentration of melatonin in the blood serum. SOREMPs are characterized by vivid hypnagogic/hypnopompic hallucinations that accompany the state of sleep paralysis (the terms "hypnagogic" and "hypnopompic" refer to hallucinations that take place while drifting to sleep and during the process of waking up, respectively). Both sleep paralysis and SOREMPs, which together are the basis of RISP episodes, are ancillary symptoms of a rare condition (with an estimated incidence of about 0.05% of the general population) called narcolepsy. The narcoleptic "tetrad" includes also cataplexy (a sudden and sometimes long-lasting loss of muscle tone) and as a main symptom excessive daytime sleepiness (Hishikawa & Shimizu, 1995). It is caused by a genet-

ic defect, which has been localized to the short arm of chromosome 6,[4] but the chromosomal localizations of the genetic basis for RISP, if there is one, are not known at present. It should be pointed out that although narcoleptic patients generally experience RISP episodes, RISP also affects individuals who do not have narcolepsy. As the muscular atonia of REM sleep is physiologically and pharmacologically indistinguishable from cataplexy, it is possible that the pineal gland also influences the development of cataplexy (Sandyk, 1995). As stated in Hishikawa & Shimizu (1995), SOREMPs may occur when some of the neural mechanisms producing wakefulness and/or "non-REM" sleep that normally inhibit the occurrence of REM sleep are abnormally weak (the activity of serotonin neurons would be abnormally decreased), or when neural mechanisms facilitating the occurrence of REM sleep are hypersensitive or hyperactive (hyperactivity of cholinergic neurons), or both. This condition might be due to an anomaly of the control mechanisms that determine the timing of REM activity and somatomotor inhibition and excitation. During normal REM sleep, the brain is highly activated, but cerebral responses to somatosensory stimulation disappear, while during sleep paralysis with SOREMPs, there is no such cerebral blocking of exteroceptive stimulation (Hishikawa & Kaneko, 1965). We conjecture that sleep paralysis and SOREMPs could be caused by an inappropriate *timing* of the inhibition of melatonin secretion and, consequently, of low melatonin plasma levels. At the physiological level, nerve cells could not be depolarized despite the relative level of alertness, and thus the low concentration of melatonin in the blood serum at an inappropriate time would cause a transient "imbalance" or inappropriate concentrations of the K^+ and Na^+ electrolytes on both sides of the nerve cell membrane during sleep paralysis accompanied (or not) by SOREMPs. At a neurological level, a somatosensory input that during normal REM sleep would be blocked by the brain may actually reach the brain during sleep paralysis/SOREMPs episodes and may be a cause of the unusual sensory experiences (feeling of electricity throughout the body, hallucinations that would in fact be REM sleep imagery, etc.) occurring during such episodes.

In spite of many studies and published reports on REM sleep, as well as on sleep paralysis and SOREMPs, we are still far from a complete understanding of the pathophysiological mechanisms of ISP/RISP, let alone of the physiological mechanisms producing muscle atonia in REM sleep, though it is apparent that these mechanisms are closely related. The conjectures presented in the last two paragraphs cannot possibly, only by themselves, account for the complexity of a full-blown RISP episode, such as the typical episode whose profile was described in Section 2. However, there seems to be a clear relationship between RISP and narcolepsy in terms of symptomology, and future advances at the molecular, genetic, and neurophysiological levels might not only provide a more comprehensive picture of narcolepsy but also help to better understand the mechanisms and the causes specific to RISP. Also, the study of the possible connections between ISP/RISP and other specific (mainly neuromuscular)

conditions might help in the future to better understand some processes inherent to ISP/RISP.

First, the so-called "Periodic Paralyses" (PP) that include, but are not limited to, (familial) Hypokalemic periodic paralysis (HypoKPP), (familial) Hyperkalemic periodic paralysis (HyperKPP), and Hypokalemic Thyrotoxic periodic paralysis (HypoKTPP) (linked to an overactivity of the thyroid gland that induces the hypokalemia) are generally considered by the scientific community to have no relationship to sleep paralysis and RISP, yet they are known to closely mimic sleep paralysis. HypoKPP is an autosomal dominant muscle disorder caused by at least three different possible genetic mutations of the gene coding the Ca^{++} voltage-gated channel in muscle cell membranes, which is characterized by episodic attacks of muscle weakness and/or rigid or flaccid paralysis associated with an abnormal decrease in blood serum K^+ levels (Lapie, Lory, & Fontaine, 1997). HyperKPP is also an autosomal dominant muscle disorder, caused by several different possible genetic mutations of the gene coding the Na^+ channel, also in muscle cell membranes, and which produces episodes of generalized flaccid weakness and/or paralysis in response to abnormally elevated levels of blood serum K^+ (Fontaine et al., 1990). Both are part of a spectrum of rare muscular disorders (with an estimated average incidence of 0.001%) called "channelopathies" (Iranzo & Santamaria, 1999). At the physiological level, it is, as we mentioned above, an intrinsic partial depolarization of muscle cells probably due to the abnormally low/high K^+ levels in the blood serum that inhibits their excitability by the nerves and leads to attacks of muscle weakness or of paralysis. In this sense, these forms of periodic paralysis are caused by an intrinsic electrolyte imbalance. Concerning HypoKPP, the only medical information we could find in terms of differential diagnosis in relation with sleep paralysis is that, according to Thorpy (1990), hypokalemic periodic paralysis has to be excluded in order to diagnose sleep paralysis. However, more recently, the first case of a patient associating HyperKPP with multiple SOREMPs was medically documented by Iranzo and Santamaria (1999), who conclude that in this case, "SOREMPs may be explained by an increased extracellular potassium conductance related to HyperKPP." Could two conditions with a seemingly different etiology (namely, HyperKPP and RISP, or intrinsic muscle cell unexcitability due to HyperKPP and the polarization of nerve cells due to low levels of melatonin in the blood during SOREMPs or RISP, respectively) both generate a similar symptomology in terms of SOREMPs? Such a question begs for more research in the possible—but not necessarily probable—connections between some forms of the PPs and RISP. Sleep paralysis/SOREMPs (or RISP) may indeed be a symptom of a variety of different pathophysiological phenomena. More research also on the coupling between nerve conduction and the process of muscle contraction could help to understand such possible connections. Of course, one can experience sleep paralysis and SOREMPs without having any of the periodic paralyses, and periodic paralysis patients may not experience sleep paralysis or SOREMPs; moreover, the case presented by Iranzo and Santamaria (1999) is

isolated and, therefore, it is not statistically significant. However, a recent survey conducted by G. Buzzi[5] on a population of 35 adults diagnosed with either HypoKPP or HyperKPP and who are members of an electronic mailing list specifically dedicated to patients affected with various forms of periodic paralysis[6] lends some credence to the possibility that a REM sleep disorder such as RISP is present in some patients with HypoKPP or HyperKPP. Of the respondents to the survey, 31.4% have had sleep paralysis "at least sometimes" (8.6% "often or always"), 65.7% of the respondents have hypnogogic/hypnopompic hallucinations characteristic of SOREMPs "at least sometimes" (25.7% "often or always")[7]; moreover, 97.1% of the respondents complain of excessive daytime sleepiness "at least sometimes" (45.7% "often or always")[8], which, as we mentioned before, is the main symptom of narcolepsy. According to G. Buzzi, there are no statistically significant differences between respondents with HypoKPP and those with HyperKPP. The percentages for sleep paralysis, and especially for SOREMPs, are significantly higher than those for the general population, thus warranting further research. Finally, a majority of the respondents also have recurrent migraines. Interestingly, migraines (with or without headaches) are believed to result from a genetic mutation of a (brain-specific) Ca^{++} channel-coding gene and hence may also be a channelopathy, involving auditory and vestibular symptoms (Gardner & Hoffman, 1998; Baloh, 1997; Roel et al., 1996).

Second, there seems to be a significant relationship between ISP/RISP and anxiety disorders:[9] as reported in Suarez (1991), in the case of anxiety disorders with agoraphobia, it was found that the percentage of patients with sleep paralysis was 40% higher than that obtained in a control group not suffering from an anxiety disorder (20%). Anxiety is a neurocognitive event involving both psychological processes and physical processes, or some might prefer to call these somatic processes. Anxiety or panic, being somewhere near the extreme end of the emotional scale, results in the release of potent signal molecules that trigger all kinds of physical events. It is well known that a variety of neurological and cognitive events may be induced by transient fluctuations of electrolytes.

This leads us to conjecture that a genetic basis, in addition to environmental factors, may thus predispose to RISP and that it is not impossible that the occurrence of RISP may be linked to some channelopathies. These hypotheses may be supported by the fact that RISP is generally familial (Dahlitz & Parkes, 1993; Roth, Buuhova, & Berkova, 1968).

We conclude that a possible common denominator between ISP/RISP and those other conditions at the physiological level may well be at least transient fluctuations of electrolytes (or inappropriate concentrations of electrolytes on either sides of nerve or muscle cell membranes) and that it is possible for an individual to have a cluster of disorders rather than a single disorder, with associated genetic defects (or mutations). Finally, we hypothesize that a condition known as Sudden Unexplained Nocturnal Death Syndrome (SUNDS) might

be an extreme case of sleep paralysis (Nimmannit et al., 1991; Randall, 1992; Adler, 1995). SUNDS is a very rare condition that is prevalent in southeast Asia (mainly in northeast Thailand and in Laos), interestingly in populations where HypoKPP is endemic (but generally induced by environmental factors), and which affects mostly young adult males. The death is a result of a myocardial infarction, the sleepers are lying in a supine position (on their back), experience strong breathing difficulties (Tanchaiswad, 1995), and there appears to be little or no movement or struggle in the dying process of SUNDS. Many cases are reported to have a fixed and "terrified" expression on their face. One explanation could be that the muscle atonia during a sleep paralysis episode would become so severe that potentially lethal cardiac arrhythmias and respiratory failure would occur. Such muscle atonia would originate from a severe hypokalemia occurring in the middle of the night, as speculated by Nimmannit et al. (1991). Once again, one can see possible relationships at a physiological level between sleep paralysis (ISP/RISP), some of the periodic paralyses, and SUNDS in terms of electrolytes.

To conclude this section, we have attempted to investigate the relationship between the generalized muscle atonia and the first phase of a RISP episode from a neurophysiological viewpoint. It is, however, very difficult to determine the relationship between the muscle atonia, the awareness, and the REM-like "hallucinations" that are characteristic of a RISP episode since so little research has been conducted on this specific subject until now. In the next section we attempt to describe some neurocognitive processes involved in the hallucinations and the possible relationships between the second phase of a RISP episode and lucid dreaming.

4. Neurocognitive Aspects of RISP: Hypnagogic/Hypnopompic Hallucinations and Lucid Dreams

The commonly experienced sleep paralysis may last from a few seconds to several minutes, occasionally longer, and is a pure motor inhibition: individuals have a subjective impression of wakefulness, a veridical perception of the actual environment (in most cases they can open their eyes and look at their surroundings), and they are unable to move despite concerted efforts to do so. At a physiological level, the motor inhibition can be explained in terms of an inappropriate timing of melatonin secretion inhibition that in turn prevents nerve cell membrane depolarization, as we have seen in the preceding section. As we have also mentioned in Section 3, in the case of RISP, in addition to the primary feature of paralysis, a large ensemble of secondary features called hypnagogic and hypnopompic "experiences" occur. (The terms "hypnagogic" and "hypnopompic" refer to experiences that take place while drifting to sleep and during the process of waking up, respectively.) Finally, more rarely, other features (tertiary features) that go beyond the hypnogogic and hypnopompic experiences occur during "full-blown" episodes of RISP, and they will be examined in more detail in the next section. The "experiences" that are part of

both the secondary and tertiary features are described as "hallucinations" in the conventional scientific literature on sleep paralysis and on SOREMPs. As stated in http://watarts.uwaterloo.ca/~acheyne/:

> A hallucination is an experience of perception in the absence of an appropriate stimulus (from the actual world), but which has the impact of a conventional perception and is not under the control of the experiencer. It is generated by internal stimuli inside the brain. A hallucination has the quality of being a sensation related to an external event rather than merely a product of the imagination. It does not seem to be merely an idea. It has the quality of objectivity, that is, something beyond the willing and wishing of the experiencer. The "object" of the hallucination is taken to exist independently of the will of the experiencer. The experience is, in principle, a publicly available phenomenon. The hallucinator should also believe that any appropriately situated person should be able to confirm these experiences. These qualities of sensation, objectivity, existence, and independence, are among the defining qualities of hallucinations. There are probably several degrees of a hallucinatory experience, besides illusions and normal or conventional sensations. A "full-blown" hallucination seems like a real experience and is believed to be a real experience. One might say the individual is both hallucinating and is deluded by the hallucination in to accepting it as a real experience. A hallucination proper may be said to have occurred if the sensation seems quite authentic but the experiencer judges the experience to be, for some reason, suspect. It seems real but there is also something counterfeit about the experience. A pseudo-hallucination also has this counterfeit quality but it also lacks the fullness of a conventional sensation. It has an ethereal, "as-if" quality, lacking the richness of a true sensation. An illusion is simply a misinterpretation of a conventional stimulus.

Sleep paralysis seems to embody all the degrees of hallucinatory experience. However, in the case of full-blown episodes of RISP, individuals are convinced that these experiences have objective external sources. They are unlikely to describe their experience as one of sleep paralysis, but rather, for instance, as one of demonic possession or of spirit encounter, and the perceived reality of the "events" that occur during RISP episodes enhances their fear or even terror. Hypnagogic and hypnopompic hallucinations include visual, auditory, haptic (tactile), and more rarely olfactive hallucinations. The individual perceives vivid dream-like imagery and hears voices, song or music, and sounds such as footsteps or static from a radio, for example. The increasing buzzing or ringing in the ears as described in Section 2 may also qualify as an auditory hallucination, although it is part of a real condition called tinnitus. The tactile hallucination includes a pressure on the chest, a sensation of choking or of touch on different parts of the body, and occasionally of genital stimulation. Another very intriguing type of hallucination that occurs commonly is feeling a presence in the room. The presence is usually perceived as threatening or evil, occasionally ugly, but also sometimes benign. As described in Section 2, it may be human, animal, or neither, or even some combination, or undefinable. It may be simply watching, or it may be speaking, or even attacking, for example by pulling the individual's legs or by trying to smother the individual (this experience is associated with the choking sensation). The pressure

on the chest may procure the sensation that the presence (or "entity") is sitting on the chest.

The "hallucinations" that are part of the tertiary features and thus occur more rarely are proprioceptive and autoscopic hallucinations. In the case of proprioceptive hallucinations, the individual feels that he, or part of himself, is at a different location from the physical body: he might feel phantom limbs or he has the subjective experience of slipping away from the physical body in what appears to be a phantom body. Subjective experiences of floating, rising, and occasionally rolling also occur. In addition, when experiencing an autoscopic hallucination, the proprioceptive hallucination is coupled with visual hallucinations: the individual, while in a floating state, can see the actual room, and eventually his physical body lying motionless on the bed. Or he can see a fictitious, dream-like environment characterized by vivid imagery, or even what seems to be a superposition of both the physical world and a dream-like world. In all three cases he has a subjective experience of awareness and his experience is perceived to be real. These types of experiences are part of the so-called "Out-of-Body Experiences" (OBEs) phenomenon. We should point out here that the autoscopic hallucinations experienced during a full-blown RISP episode are not to be confused with the "classical" autoscopic hallucinations, in which case an individual, while in a fully awake state and moving, "sees" a (phantom) double of himself or "Doppelgänger." Interestingly, in the case of sleep paralysis the opposite situation seems to occur: the phantom body "sees" the physical body from some vantage point. The term "autoscopic" may still be used since there is a dissociation of a physical body and of a perceived (phantom) body double.

In addition, the tertiary features include other types of experiences such as derealization (the feeling that the surroundings of the individual are unreal), depersonalization (a loss of sense of personal identity), a subjective continuity of conscious experience as opposed to the shifting images of a usual dream, a dissociation of modalities, distortions of body image, and tingling or vibrating sensations. Usually, these tertiary features seem to occur later in a RISP episode than the secondary features, in what we call a second phase of the episode. Finally, in a third phase, the phantom body reenters the physical body, slowly or abruptly. The individual might then wake up or fall back into another episode, not uncommonly directly in the second phase.

Based on the above description of the different features of RISP, we now attempt to connect some of the basic features with what is known at present on the subject of awareness during sleep and hallucinations in the field of cognitive neurosciences.

Already in the 1960s, the Canadian neurologist Penfield (1963) had shown that auditory hallucinations can be induced in the wake state by electrical stimulation of the temporal lobe. Moreover, it has recently been shown that tinnitus, a condition that affects tens of millions of people worldwide and is in some cases permanent,[10] is closely associated with the activity of the auditory cortex and involves the temporal lobe (Lockwood et al., 1998). We therefore

may conjecture that the tinnitus (buzzing/ringing sound in the ears) that increases in intensity during the first phase of a RISP episode as well as other auditory hallucinations (such as voices or a variety of sounds) are associated with the temporal lobe and that the activity of the temporal lobe increases significantly at the beginning of RISP episodes. In the same way, internally generated visual stimuli (via the visual cortex) would be responsible for the visual hallucinations, at least during the first phase of a RISP episode, and would have REM-like qualities despite the fact that there still is a subjective experience of awareness. Very little, if anything, is known at present about the specific areas in the brain which would be involved in the other types of experiences, in particular the tertiary features of experiences. If we now address the issue of awareness, it has been shown that, whereas higher order mental functions associated with the activity of such brain centers as the prefrontal cortex are essentially shut down during normal REM sleep and the limbic system (an older part of the brain) that is responsible for emotional and visceral phenomena is activated (Braun et al., 1998), those same higher level functions are still operating during lucid dreams (LaBerge, 1985).

While lucid dreaming, loss of critical insight, diminished self-reflection, and impaired logic that is typical of normal dreaming does not seem to occur, critical thinking can still take place. The awareness that is retained during the second phase of a RISP episode and some characteristics of this phase (the dream-like surroundings) tend to indicate that a close connection probably exists between RISP and lucid dreaming, although it is improbable that a whole RISP episode is itself a lucid dream. Rather, lucid dreaming could be one of several different components of a RISP episode. Indeed, Takeuchi et al. (1992) elicited ISP from normal subjects by a nocturnal sleep interruption schedule. All of the subjects with ISP experienced inability to move and were simultaneously aware of lying in the laboratory. All but one reported auditory/visual hallucinations and unpleasant emotions. Judging from the polysomnogram recordings they obtained, ISP differs from other dissociated states such as lucid dreaming, nocturnal panic attacks, and REM sleep behavior disorders. However, the polysomnogram might have recorded only the first phase of a RISP episode, and recordings of a second phase might have shown similarities with lucid dreaming.

5. Is RISP Conducive to Paranormal Phenomena? Analysis of the Obe Hypothesis

In the preceding section we mentioned that the experiences that are part of both the secondary and tertiary features of sleep paralysis are described as "hallucinations" in the conventional scientific literature on sleep paralysis. In this section we will argue that at least some of the tertiary features may involve processes at a higher level than pure neurocognitive processes, more precisely that a full-blown RISP episode may induce some specific paranormal phenomena. The most obvious phenomenon is the "Out-of-Body Experience" (OBE)

that seems to occur from the beginning of the second phase of a full-blown episode.[11] To describe this phenomenon we have used the words "proprioceptive" and "autoscopic hallucinations" in the preceding section. On the other hand, there is also considerable evidence that people who tend to have OBEs also tend to have lucid dreams, flying and falling dreams, and the ability to control their dreams (Blackmore, 1984; Glicksohn, 1989; Irwin, 1988). Because of the strong connection between OBEs and lucid dreaming, some researchers in the area have suggested that OBEs are a type of lucid dream (Faraday, 1976; Honegger, 1979; Salley, 1982). One problem with this argument is that although people who have OBEs are also likely to have lucid dreams, OBEs are far less frequent and can happen to people who have never had lucid dreams. Furthermore, OBEs are quite plainly different from lucid dreams in that during a typical OBE the experiencer is convinced that the OBE is a real event happening in the physical world and not a dream, unlike a lucid dream, in which by definition the dreamer is certain that the event is a dream. There is an exception that connects the two experiences: when we feel ourselves leaving the body, but also know that we are dreaming. However this last case seems not to apply to RISP because the RISP experiencer is generally convinced of the reality of his experience. LaBerge and Levitan (1991) stress that even if some or most OBEs were in fact dreams or lucid dreams, we cannot say that a genuine OBE is impossible. However, he suggests that if you have an OBE, why not test to see if the OBE-world passes the reality test. Several different modalities (visual, auditory, etc.) can be used simultaneously as cues to distinguish a dream or a hallucination from a real experience: For example, is the room you are in the one you are actually sleeping in? If you have left your physical body, where is it? Do things change when you are not looking at them (or when you are)? Can you read something twice and have it remain the same on both readings? LaBerge asks "*If any of your questions and investigations leave you doubting that you are in the physical world, is it not logical to believe you are dreaming?*"

In the case of RISP at least, the decision of whether one (or rather one's phantom body or "non-physical double") sees the physical world during a RISP episode or instead one sees a dream world is often very difficult to make. From many accounts that can be found on the UCLA sleep web site where the respondents reported having an OBE during a RISP episode and from the personal experiences of the authors, at least during some episodes the physical world seems to be perceived, such as the room of the experiencer and his physical body (most often seen from above), various objects in the room, or the ceiling of the room, etc. A reality check made upon waking up positively identifies what has been perceived during the episode with the actual surroundings, but there are occasionally some anomalies, such as objects that were seen in the room during the episode but are in fact not there or distortions of actual objects. One could argue that somehow the mind has played a trick on the experiencer by "mimicking" or "cloning" the actual surroundings, that is, recording

them as a memory inside the brain while in the wake state and "projecting" them in a dream world during a RISP episode. This is not a plausible explanation if one considers that it is not possible to "prerecord" in the wake state an actual image of oneself as seen from above (or from different viewpoints), as it is perceived during some episodes. Instead, we are led to conjecture that it is not impossible that a genuine OBE could occur simultaneously with oniric imagery, with both external and internally generated stimuli being processed concurrently. The OBE component could be called "the signal" since it refers to sensing external stimuli that are part of the physical world, and the oniric imagery could constitute a form of "noise" if we are mainly interested in the paranormal phenomenon of the OBE instead of other possible paranormal phenomena associated with dreams. Hence there could be a superposition of two states; a conscious state carried out by the OBE and a dream state characterized by oniric imagery.

A statement made by Tart (1967) is in a sense strikingly similar to our conjecture: on the basis of his laboratory experiments, he concluded that perhaps the OBEs are a mixture of dreams and "something else." This something else might, he thought, be an Extra-Sensory Perception (ESP), such as a genuine OBE. We should point out, however, that Tart did not mention the words "sleep paralysis" in his research, and we do not know if the subjects in his experiments had sleep paralysis episodes or were having OBEs without the paralysis. Finally, it is also entirely possible that some RISP episodes might include only lucid dreaming, some others genuine OBEs, and still others a superposition of a genuine OBE and of a dream-state. Conversely, OBEs could occur in a variety of states, not only during RISP episodes. Indeed, we point out some similarities that exist between some RISP episodes and Near-Death Experiences (NDE). As Moody writes (1975, 1976)

> A man is dying and, as he reaches the point of greatest physical distress, he hears himself pronounced dead by his doctor. He begins to hear an uncomfortable noise, a loud ringing or buzzing, and at the same time feels himself moving very rapidly through a long dark tunnel. After this, he suddenly finds himself outside of his own physical body, but still in the immediate physical environment, and he sees his own body from a distance, as though he is a spectator. He watches the resuscitation attempt from this unusual vantage point and is in a state of emotional upheaval.

One can immediately recognize some common components: the buzzing/ringing sound in the ears, the OBE, and the tunnel that is seen during some RISP episodes. Moreover, occasionally intense RISP episodes include other common elements with NDEs, such as a feeling of peace and ineffability, vivid and beautiful landscapes, a light that appears at the end of the tunnel and that is bright but does not hurt the eyes, etc. The question then is "Are NDEs some form of sleep paralysis episode, or do some intense RISP episodes mimic what is experienced near death?" Unfortunately, to our knowledge, no research has addressed those two questions until now.

Another parallel may be made between the perceived OBE during the second phase of a full-blown RISP episode and, in seemingly completely different circumstances, the "ability" of shamans to "leave their body" at will and explore other "realms of existence." As is stated by Halifax (1991)

> The shaman, lord of the three realms of sky, earth and the underworld, is an individual endowed with the ability to enter profound trance states, a "technician of ecstasy," as Mircea Eliade (1964) has so aptly termed this religious specialist. In these visionary states, the shaman is open to contact with animal allies and spirit helpers. Or the wizard may leave his or her body behind like a husk while the disincarnate soul journeys to the celestial realms above or the underworld of disease and death.

It is interesting to note that shamanic initiation involves acute physical illness that may lead to near-death states (hence a possible connection with NDEs) and/or an intense psychological crisis (Halifax, 1991). Shamans have learned to control their "OBEs," whereas the vast majority of RISP experiencers, already subjected to an intense fear at the onset of a RISP episode, seem, to say the least, to be extremely puzzled if they access the tertiary features with the main feature being the "OBE" itself. In any case, the perceived OBE seems to be the main phenomenon linking full-blown RISP episodes to NDEs or to shamanic experiences.

Finally, some intriguing aspects of RISP that are also part of the tertiary features are the tingling or vibrating sensations that are experienced during some episodes, as well as bright flashes of light or sensations of explosion in the head. Such sensations match quite closely those that are described in the literature of Kundalini manifestations (Krishna, 1993). Kundalini experiences also include the feeling of leaving one's body, but they include also other phenomena that do not occur in RISP episodes. One important fact is that people who have what they consider Kundalini manifestations usually practice some form of Yoga that can produce changes in states of the mind. That is, they may be engaged in meditative practices, which can reach a point similar to the condition of falling asleep. When that happens, it is plausible that the phenomena the meditators experience might be similar to, if not the same as, those experienced in RISP. For example, let us consider the following account by Gopi Krishna (1993):

> During one such spell of intense concentration [on a shining lotus] I suddenly felt a strange sensation below the base of the spine, at the place touching the seat … The sensation extended upwards, growing in intensity … Suddenly, with a roar like that of a waterfall, I felt a stream of liquid light entering my brain through the spinal cord … The illumination grew brighter and brighter, the roaring louder. I experienced a rocking sensation and then felt myself slipping out of my body, entirely enveloped in a halo of light.

One should note here that Kundalini manifestations, as described in Gopi

Krishna's autobiography, as well in other references (Greenwell, 1990; Rieker, 1971), may have a lot more impact and consequences than the phenomena experienced by individual RISP episodes, in the sense that they have the potential for profound life transformations, just as a shamanic initiation. Meditation practices may increase the occurrence of RISP in an individual, with the associated OBEs, flashes of light, and vibrating sensations, but the meditator generally controls much more the Kundalini manifestations arising from the meditation than individuals who do not meditate control the phenomena experienced during their RISP episodes. In conclusion, if we consider that genuine OBEs are indeed possible during RISP episodes, one important issue to address is how then external visual/auditory stimuli (visual/auditory information from the actual world) are captured by the "non-physical body double" and how are they transmitted to the brain of the experiencer for interpretation and for future recall. Once again, until now it seems that no research has addressed this important issue. One interesting experiment could be to gather information on RISP experiences of blind subjects, especially subjects blind since birth, in order to assess the range and sense of reality perceived by such particular subjects—if there are any blind subjects who experience RISP.

6. Psychological and Socio-Historical Aspects of RISP: An Analysis of the RISP Experiences of a Sample Population

There is no doubt that sleep paralysis (whether ISP or RISP) has been documented since as early as Hellenistic times, but with a wide variety of interpretations throughout time that reflect the culture, folklore, and belief system of different populations or ethnic groups worldwide. The hypnogogic/hypnopompic hallucinations that are part of the secondary features of ISP/RISP have been interpreted as being:[12] indigestion ("hypocrites") for the Hellenic Greeks, guilt for the Romans and the Egyptians, witchcraft for the Mexicans, for the Yoruba people and for the Africans (Ohaeri et al., 1992),[13] demons for Medieval Europe, djinns for the Arabs, vampires for Europeans, hags (witches) for the Irish and Scottish (actually such hallucinations are still called "Old Hag" attacks in parts of the United States and of Canada, particularly in Newfoundland; Ness 1978), spectral foxes or spirit encounters for the Japanese, cats for the Chinese, ancestral ghosts for Southeast Asians (e.g., Wing et al., 1994; Adler, 1995), or the spirit of a dead unbaptized baby to different people in different parts of the world, etc.

In particular, the malevolent presence that is often perceived during ISP/RISP episodes, as well as the strong pressure on the chest (most often it is on the chest since sleep paralysis is also associated with lying in the supine position) were ascribed by the ancient Romans to incubus (incubare: to lie upon) and succubus attacks: incubi were believed to be male demons, and succubi female demons who were attacking the sleeper.[14] The Italian word "incubo" that is used nowadays to signify "nightmare" is derived from the Latin word "incubus." Similarly, the English word "nightmare," according to Webster's dic-

tionary, is derived from the words "night" and "mare," where "mare" signifies "demon," and a "nightmare" was formerly believed to be an evil spirit that would haunt and suffocate sleeping people. There are other traditions of hags (as mentioned above) and other instantiations of evil or grotesque creatures (fairies, gremlins, and assorted "little people") attacking, or trying to possess or abduct their helpless victim (Hufford, 1982).

The phenomenon of sleep paralysis has also been vividly depicted in the arts and in literature: For example, Henry Fuseli's famous painting *The Nightmare* (1781) seems more likely inspired by the notion of sleep paralysis rather than any sleeping dream (Schneck, 1969); Henri de Maupassant's *Le Horla* (published in 1887) contains a remarkably thorough and highly evocative account of sleep paralysis (Schneck, 1994;[15] it is interesting to note, with respect to the remark we made in Section 4 concerning autoscopic hallucinations, that de Maupassant, as well as apparently having experienced terrifying sleep paralysis episodes, often saw a double of himself or "Doppelgänger" when fully awake, and he was ultimately committed to a mental institution); and sleep paralysis has also been described in F. Scott Fitzgerald's *The Beautiful and Damned* (Schneck 1971), as well as in Herman Melville's *Moby Dick* a quarter of a century earlier than the first scientific description of the phenomenon by Silas Weir Mitchell in 1876 (Herman, 1997; Schneck, 1977). Despite the various interpretations, the descriptions of the hallucinations are remarkably similar. In the modern, Western industrialized world, especially in North America, most people have no connection with these ancient traditions (which also tried to explain these "demonic possessions" and developed rituals for exorcising the "possessions"), and so are quite mystified by the entire experience. Many of the descriptions of North Americans are rather vague and insubstantial in contradistinction to the more vivid and elaborate traditional descriptions. However, in North America, a new feature to explain the evil presence during sleep paralysis episodes is the phenomenon of so-called "alien abduction." Cheyne[16] presents an excellent sociological study of the similarity between the experience of sleep paralysis reported in the clinical and experimental literature and reports of alien abduction. Such similarity had already been theorized by popular writers of science such as the late Carl Sagan (1996).

In relation with the above socio-historical summary of the phenomenon of sleep paralysis, we now briefly present preliminary results of a statistical analysis of the reports of ISP/RISP episodes posted by a sample population of 250 individuals on the sleep web site of the University of California in Los Angeles (UCLA). We point out that the sample is biased in the sense that the vast majority of respondents are of Anglo Saxon origin. However, we think that most features of sleep paralysis have a sufficiently universal character that most statistical results are reasonably valid. The differences between this sample and a sample with a wide geographic distribution could really be significant in the interpretation of the sleep paralysis experience by the respondents, as the previous socio-historical summary seems to suggest.

First, the most common precipitating factors for ISP/RISP were found to be stress and/or disruption of sleep patterns, such as sleep interruption (in agreement with the clinical results presented by Takeuchi et al., 1992), oversleeping, or napping. It has been reported in the scientific literature that other precipitating factors could be jet lag (Snyder, 1983), intense meditation[17] or intense intellectual concentration, sensitivity to heat or to cold, or even possible variations in geomagnetic activity (Conesa, 1995): periods of relatively quiet geomagnetic activity would be significantly associated with an increased incidence of episodes. It has also been suggested that the hypnogogic/hypnopompic hallucinations (and possibly also the experiences associated with the tertiary features) could occur more often in highly creative individuals, but this has not been confirmed statistically.

Second, no sex difference has been found in the prevalence of ISP/RISP and there seem to be three patterns of recurrence: most commonly, sleep paralysis occurs only once or infrequently in an individual's life, with interval of several months or even years. Occasionally, an ensemble of several frequent episodes occurs, but lasting only a short period of time (a few weeks). More rarely, episodes occur recurrently, frequently, and over long periods of time spanning years, and "full-blown" episodes are predominant: such cases qualify as RISP.

Third, the hypnogogic/hypnopompic hallucinations experienced during sleep paralysis episodes induce fear, usually intense, in over 90% of the individuals. In particular, the sensed evil presence, followed by the paralysis itself, is a terrorizing experience. The vast majority of the respondents who experience intense fear do not reach the second phase of a RISP episode. For those who do, the fear diminishes somewhat and puzzlement when seeing the physical surroundings (the room, the physical body from above) increases. The other 10% of respondents do not experience fear (or at least they have "learned" to control their fear or gotten used to the episodes if they happen frequently), although they generally do not enjoy the hallucinations, but they tend to find pleasure, excitement, and enjoyment when experiencing the tertiary features.

Finally, about half of the respondents invoke a paranormal or a supernatural cause to explain their episodes (or at least, they have initially evoked such causes; upon learning about sleep paralysis, their belief tends to change to invoke natural, biological causes). Interestingly, none of the respondents has interpreted his/her episodes as alien abductions. Either individuals who report alien abductions have chosen not to post messages, or the association between sleep paralysis and alien abductions is not as strong as it is generally thought.

7. Suggested Strategies to Cope With RISP

Since RISP appears to be such a frightening experience to the vast majority of individuals with the condition and in some cases perturbs significantly day-to-day life, we briefly describe several different strategies that can be used to

mitigate its negative effects. We first enumerate different "conventional" medical treatments that have been applied in recent years with varying degrees of success, and then present a "psychological" approach that individuals may adopt in order to cope with RISP.

The medical treatment most commonly used to reduce the frequency of occurrence of RISP or even to stop RISP from occurring is the prescription of tricyclic antidepressants such as imipramine and clomipramine, and such a treatment is not limited to isolated sleep paralysis but is in fact widely used for narcolepsy, whose main symptoms are uncontrolled daytime sleepiness and cataplexy (sudden loss of muscle tone; Shimizu, 1998; Shapiro, 1975). As a matter of fact, imipramine is particularly effective in controlling cataplexy (Madea et al., 1995; Campbell, 1981; Dement et al., 1976) and may be significantly more so than in treating RISP (Snyder & Hams, 1982). Other treatments include serotoninergic agents such as L-tryptophan with or without amitriptyline (Snyder & Hams, 1982). More exotic forms of treatment have been applied, such as magnetic therapy, i.e., the application of weak time-varying electromagnetic fields (EMFs) (Sandyk, 1997; in the particular treatment described, AC-pulsed picotesla intensity EMFs of 5 Hz frequency were applied extracerebrally 1–2 times per week to a single patient), but the fact that episodes of sleep paralysis gradually diminished and abated completely over a period of 3 years in the case of a single patient does not necessarily imply that such a treatment would be successful for a majority of RISP experiencers. However, an interesting suggestion made by Sandyk (1997) is that the resolution of sleep paralysis in this particular patient by AC-pulsed applications of EMFs is related to enhancement of melatonin circadian rhythms and to cerebral serotoninergic neurotransmission, as pineal melatonin and monoaminergic neurons are implicated in the induction and maintenance of REM sleep and the pathogenesis of sleep paralysis. This also implies that taking melatonin at appropriate times for a better control of circadian rhythms may—at least—reduce the frequency of RISP episodes. Indeed, as pointed out in Snyder (1983), jet-lag seems to be a precipitating factor of isolated sleep paralysis, and the beneficial effects of melatonin in mitigating jet lag by correcting the resulting sleep pattern disturbances are well known. Therefore, a careful administration of melatonin may also have beneficial effects regarding RISP. However, until now we have not yet found any medical source confirming such a hypothesis. Finally, as stated by Dement et al. (1976), "No completely satisfactory treatment is available at the present time." This applies to narcolepsy as well as to RISP. Although we have described several medical approaches for treating RISP and have expressed the hypothesis that melatonin may have beneficial effects, we point out that we are not engaged in rendering medical advice or professional services and that RISP experiencers who have significant difficulties in coping with this condition should consult reputable sleep specialists and/or neurologists.

Since there is at present no proven medical treatment that would reduce the

frequency of RISP episodes, let alone eliminate RISP completely, for the vast majority of RISP experiencers, we may envisage various psychological approaches that could lead the experiencers to cope with RISP satisfactorily. At first, one should realize that RISP might occur during most of an individual's life—if not during all his/her own life—despite him/her leading a reasonable way of life (i.e., regular sleep patterns, mitigation of stress, an appropriate diet, reasonable exercise, etc.). In such a case, with time one can indeed learn to accept RISP as a "normal" part of one's life, and even obtain a better grasp of one's own individuality, by considering the following factors: first, consider the phenomenon as something to be explored. We have seen that RISP is not dangerous in general. The real problem to solve is the intense fear that is felt by most experiencers at the beginning of an episode. The wish to conquer the fear and to explore will lead to a radical psychological change, from a defensive stand to a courageous attitude of outward observation and learning. As an example, let us quote a message posted by a RISP experiencer on an electronic mailing list devoted to the "Awareness during Sleep Paralysis" (or ASP):[18]

I just wanted to let you all know about my first ever pleasant ASP! It happened on Saturday night, and I felt all the usual sensations (buzzing, being pushed down/dragged) but wasn't scared and just let it happen. After a while I felt like I was being lifted from the bed, and next thing I knew I was "floating" near the top of my wardrobe. I looked down at my bed and could see that it was unmade, but there was no one in it. I also remember being very excited, and pleased that this was such a non-threatening experience!

Second, When feeling a presence at the beginning or during a RISP episode, try to face it. Stephen LaBerge (1985) has suggested that, upon encountering an evil presence during a lucid dream, one should face it and try to transform it, or the unpleasant situation, into something good. There is a "forgiving attitude" in this suggestion, which might result in a transformation of the "evil presence" into something either neutral or outright friendly. Equivalently, a similar course of action can be used when having hypnogogic/hypnopompic hallucinations at the beginning of an episode involving the feeling of an evil presence. Other tactics might be to shout at the presence in order to conquer the fear, or prayers for religiously inclined individuals, which can help build a positive attitude. Finally, when hearing an increasingly loud buzzing sound, and/or feeling increasing pressure on the chest or inside the head, one might state repeatedly to oneself that RISP is not dangerous, so that one may proceed to the (generally more pleasant) next phase of the episode. An interesting approach to cope with RISP that is often used in Japan, where sleep paralysis is prevalent and is named "kanashibari" in Japanese (Fukuda et al., 1987), meaning "still-bound," is humor: RISP has been discussed repeatedly on various Japanese television programs, and jokes about RISP are often added to the serious discussions,[19] just as jokes about RISP are also included sometimes in television "durama" (dramas) in Japan.

Third, if these approaches or any other tactics used by a RISP experiencer fail, and if RISP continues to disturb one's daily activities, one can resort to professional psychological counseling with informed psychologists, eventually including also family counseling to inform the family of the experiencer about RISP and its benign, nonthreatening character.

8. Conclusions

To conclude, we can honestly say that most aspects of sleep paralysis, of RISP in particular, are still largely not understood, partly because of a lack of research of the phenomenon and also partly because of its "unsubstantial" nature. It is true that sleep paralysis has received increasing attention in the scientific community in recent years, particularly by psychologists in various universities or research centers, such as the departments of psychology at the university of Waterloo in Canada,[20] at the university of Edinburgh in the UK,[21] or at Fukushima university in Japan (Fukuda et al., 1987, 1991; Fukuda, 1993; Takeuchi et al., 1992, 1994), to name a few. It is also encouraging to see that the connections between consciousness and such states as lucid dreaming, or fields such as transpersonal psychology and parapsychology have been addressed recently in the Second Tucson Discussions and Debates on consciousness by world experts (Hameroff et al., 1998), although sleep paralysis has not (as yet) been included in the discussions. Also, the public at large in the United States was recently sensitized to the phenomenon of sleep paralysis, as well as to other sleep disorders, through a special television program entitled *Sleep Files*.[22] Therefore the tendency to explore in more depth such puzzling phenomena as sleep paralysis and their relationships with consciousness seems to increase. It is the hope of the authors that serious multidisciplinary research will in the near future unravel some of the mysteries of sleep paralysis and help develop better coping strategies for uninformed individuals whose lives are negatively affected by a phenomenon they perceive to be terrifying and that they cannot explain.

Disclaimer

This paper is the result of personal research by the authors and the first author officially declares that the subject matter of the paper has no relationship whatsoever with his research in the Human Information Processing Research Laboratories of ATR in Kyoto, Japan, and that his opinions do not necessarily reflect those of his employer.

Notes

[1] http://bisleep.medsch.ucla.edu/discussions/basic/msgs.html. The initial message was posted on January 23, 1996 and in it the words "sleep paralysis" were mentioned for the first time since the establishment of that site.

[2] A good example of psychological study is the survey on sleep paralysis es-

tablished on the web site of the department of psychology of the university of Waterloo, in Ontario, Canada, at: http://watarts.uwaterloo.ca/~acheyne/S_P.html.

[3] http://bisleep.medsch.ucla.edu/discussions/basic/msgs.html. The initial message was posted on January 23, 1996 and in it the words "sleep paralysis" were mentioned for the first time since the establishment of that site.

[4] In August 1999, Dr. L. Lin and Dr. E. Mignot's team (1999) from the Stanford University Sleep Research Center in California announced that they had identified the gene that causes narcolepsy in dogs. Simultaneously, Dr. R. M. Chemelli et al. (1999) from the Howard Hughes Medical Institute and the University of Texas Southwestern Medical Center announced that they had found a closely associated gene in mice. Quoting Dr. Mignot (1999), "Pinpointing the narcolepsy gene in dogs could open the gate to understanding human sleep disorders."

[5] Dr. G. Buzzi is an Italian neurologist practicing in Ravenna, Italy, and who specializes in sleep disorders. His e-mail address is gbuzzi@MAIL.LINKNET.IT. Just as the first author of this paper, he is a member of an electronic mailing list specifically devoted to patients affected with various forms of periodic paralysis (HypoKPP, HyperKPP, HypoKTPP, NormoKPP, etc.), where both are scientific observers. The address of the list is HKPP@maelstrom.stjohns.edu. To subscribe to the list, one should contact calexeditor@NUCLEUS.COM. Access to the list is strictly reserved to patients diagnosed with or suspected of being affected with one of the different types of periodic paralysis and to scientists interested in the PPs. The mailing list is associated with the Periodic Paralysis Association (PPA), a not-for-profit organization established and officially registered in the United States in 1998 that is devoted not only to an understanding of the different aspects of the PPs, but also to possible connections with other disorders, including sleep disorders. The URL address of the PPA is http://www.periodicparalysis.org. The first author of this paper is also a scientific advisor to the PPA, for sleep disorders.

[6] As mentioned above, the electronic address of the list is HKPP@maelstrom.stjohns.edu.

[7] The full results of the survey were posted on the list (HKPP@maelstrom.stjohns.edu) by Dr. G. Buzzi on January 19, 2000 and they can be retrieved from the archives of the list or obtained by contacting calexeditor@NUCLEUS.COM.

[8] As mentioned above, the electronic address of the list is HKPP@maelstrom.stjohns.edu.

[9] The first author of this paper has a mild anxiety disorder with agoraphobia in addition to RISP.

[10] The first author of this paper has been having permanent tinnitus since childhood. The intensity of the tinnitus increases significantly at the beginning of his RISP episodes.

[11] An extensive FAQ on the OBE phenomenon can be found at the following web site: http://www3. eu.spiritweb.org/Spirit/obe-faq.html.

[12] http://www.geocities.com/Paris/LeftBank/1700/hag.html

[13] Although the study is confined to Nigeria, it is probable that sleep paralysis is experienced in most other African countries, and the popular view in Africa is that it is caused by witchcraft.

[14] http://www.geocities.com/Paris/Leftbank/1700/hag.html

[15] An excellent explanation of sleep paralysis that includes the original text of de Maupassant's *Le Horla* describing a sleep paralysis episode can be found at http://watarts.uwaterloo.ca/~acheyne/LeHorla.html. See also the recently published journal paper Cheyne, Newbyclark, & Rueffer (1999).

[16] http://watarts.uwaterloo.ca/~acheyne/

[17] http://watarts.uwaterloo.ca/~acheyne/LeHorla.html

[18] ASP-mailing list ("*Awareness during Sleep Paralysis*" mailing list), (September 1998). To subscribe, send a message to: nightflyer@mindspring.com. (Warning: This is not a professional list. It simply contains personal accounts of individuals having RISP episodes.)

[19] The first author of this paper once saw a popular Japanese television program entitled "*Unbiribabo*" (meaning "*Unbelievable*"), animated by the famous television celebrity and Japanese actor Takeshi, where jokes about RISP where added to serious discussions on the subject: in a whimsical sketch, Mr. Takeshi was bound by large metal chains (hence referring to the paralysis), while sitting on a chair, and was frantically shouting for help (aired on August 8, 1998, Kansai-TV, channel 8, Japan).

[20] A good example of psychological study is the survey on sleep paralysis established on the web site of the department of psychology of the university of Waterloo, in Ontario, Canada, at http://watarts.uwaterloo.ca/~acheyne/S_P.html.

[21] M. Simon Sherwood is a PhD candidate in the department of psychology of the university of Edinburgh in the UK whose thesis will be a study of the parapsychological aspects of sleep paralysis. His email is sherwood@holyrood.ed.ac.uk.

[22] The television program entitled S*leep Files* was aired on the Discovery Channel (*Discovery Signature Series*) on June 21, 1998, at 10:00 p.m. (EST) and on June 22, at 1:00 a.m., as well as on June 28, from 3:00p.m. to 6:00 p.m. The name of the episode that covered sleep paralysis and Dr. David Hufford is *Altered States* (http://www.discovery.com).

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