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Sleep Paralysis and Locked-In Syndrome: An overview

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Abstract

The purpose of this review is to compare and contrast Locked-In Syndrome with Sleep Paralysis. These disorders share partially common symptomatology and therefore, the findings of one may shed light on unresolved issues of the other. This paper opens with the definitions of these syndromes. The most common causes or risk factors of these disorders are then thoroughly analyzed. Etiology and potential risk factors are followed by the methodology used in this review. The similarities as well as the differences between these disorders are presented in detail. The role of the brainstem in the occurrence of Sleep Paralysis, the extent to which cognitive functions are preserved and the issues raised around the hallucinations involved in these disorders are being discussed. Finally, suggestions about future research are made. This review aims to find the existing literature and compare findings about the life of people suffering from Locked-In Syndrome or Sleep Paralysis.

Keywords: Sleep Paralysis, Locked-In Syndrome, voluntary muscle paralysis, hallucinations, brainstem

Introduction

Defining Sleep Paralysis

Sleep paralysis (SP) is a sleep disorder characterized by temporary inability to move upon awakening or at sleep onset (Singh et al., 2018; Suni, 2022) with normal cognitive function (Malhotra & Avidan, 2012). It lasts from a few seconds to a few minutes (Hinton et al., 2005b) and for as long as it lasts no voluntary movement can take place (Denis et al., 2018a; Malhotra & Avidan, 2012; Singh et al., 2018) including speech (Ma et al., 2014) although people are conscious and aware of their current situation (Suni, 2022). They are aware of their immediate surroundings (Sharpless & Denis, 2017) as well as their inability to move (McNally & Clancy, 2005). They feel awake (Suni, 2022). In other words, the body is still asleep while the brain is active.

SP is in fact a prolongation of the muscle atonia present in the REM (Rapid Eye Movement) phase of sleep upon awakening (Singh et al., 2018). Therefore, it results in a mixed state of consciousness characterized by both REM sleep elements, such as sustained eye movement and breathing pattern (Malhotra & Avidan, 2012), and wakefulness elements (Denis, 2018; Sharpless & Denis, 2017; Suni, 2022) such as awareness. It is worth noting that this state is often accompanied by hallucinations (Sharpless, 2016; Sharpless & Denis, 2017; Suni, 2022) as well as difficulty in breathing or a feeling of pressure in the chest (Ma et al., 2014). People come out of this state automatically or thanks to an external stimulus (Malhotra & Avidan, 2012) such as someone else's voice or touch or by a great effort to move (Suni, 2022). When it does not co-exist with other medical conditions it is called Isolated Sleep Paralysis (Sharpless, 2016).

Defining Locked-In Syndrome

Locked-In Syndrome (LIS) is a rare neurological disorder which is characterized by paralysis of all voluntary muscles, with the exception of those regulating vertical eye movement and upper eyelid movements, and preserved consciousness (Farr et al., 2021). People who suffer from LIS have tetraplegia and anarthria but normal cognitive function

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and self-consciousness (Maiser et al, 2016). Besides tetraplegia, there is also paralysis of the facial, pharyngeal, tongue and palate muscles which result in anarthria and mutism (Golubović et al., 2013). Dysphagia and respiratory problems are basic clinical features as well (Pacheco-Hernández et al., 2017). LIS patients can communicate only through vertical eye movements and blinking, because they can't move anything else (Golubović et al., 2013; Young, 2014). Other important features of the syndrome are the dysfunction of horizontal eye movement and the normal pupillary light reflex (Barbic et al., 2012). More often than not a cutaneous sensation is preserved (Barbic et al., 2012). Although LIS patients seem to be in a vegetative state (Pacheco-Hernández et al., 2017) they can see and hear, they are self-conscious and aware of their surroundings (Pacheco-Hernández et al., 2017). Hallucinations have also been reported (Sarà et al., 2018). Finally LIS is an irreversible disorder with high mortality rates (McNair et al., 2019).

Etiology of Locked-In Syndrome

LIS is caused by a lesion to the ventral pons of the brainstem (Kumral et al.,2022; Law et al., 2018; Maiser et al., 2016; Young, 2014). The first most common cause of this lesion is ischemic stroke (Law et al.,2018) and the second is brainstem injury (León-Carrión et al., 2002). Ischemic stroke stems from occlusion of the basilar artery (Golubović et al, 2013; León-Carrión et al., 2002). Most of the times this occlusion happens due to thrombosis which in turn causes stroke and consequently damage to the pons (Golubović et al., 2013). Thrombosis in the basilar artery can derive from trauma (Golubović et al., 2013) and rarely from cocaine (Ali et al., 2019). The latter increases the chances of stroke and thus is included in the causes of LIS (Ali et al., 2019).

According to Cardwell (2013) in addition to thrombosis leading to stroke LIS can also stem from trauma, tumors that damage the pons of the brainstem, metabolic disorders, bleeding episodes, iatrogenic causes, infections and other causes. Young (2014) claims that another cause of LIS is polyneuropathy such as the one caused by Guillain-Barré syndrome. Considering the above, it is easy to conclude that LIS results from physical causes.

Etiology-Risk factors for Sleep Paralysis

Although SP has unknown causes some factors that increase the risk of developing SP have been identified (Denis et al., 2018a). Starting with physical causes, a possible cause of SP is dysfunction of the mechanism that regulates muscle atonia during REM sleep (Malhotra & Avidan, 2012). McNally and Clancy (2005) suggest that lack of synchronization between motor and cognitive mechanisms of REM sleep is responsible for SP that occurs during wakefulness and is accompanied by hallucinations. The electroencephalogram during SP indeed depicts a desynchronization, a mixed state of REM sleep and wakefulness which explains the intrusion of elements from both states of consciousness like actual perception of the environment and hallucinations (Sharpless & Denis, 2017).

SP episodes may occur due to defective regulation of cholinergic or other types of neurons during transitions from REM sleep to wakefulness (Sharpless & Denis, 2017). Sharpless and Denis (2017) argue that dysfunction of the cholinergic system and disorders of neurotransmitters like serotonin and norepinephrine in the transitional stages of sleep are responsible for SP episodes. This claim is based on the fact that with serotonin administration SP episodes gradually disappear or at least get better (Sharpless & Denis, 2017). The role of heredity and genes in the occurrence of SP is not negligible. It is believed that some genes related to circadian cycles are associated with SP (Sharpless & Denis, 2017). It is worth mentioning that in a study conducted in siblings and twins on the heritability of SP the results showed that SP is moderately heritable (Denis et al., 2015). Physical health is also associated with SP by increasing the chances of experiencing it when a person suffers from chronic pain that negatively affects his quality of life (Ohayon & Pakpour, 2022; Young et al., 2013).

Anxiety disorders and generally life stress and susceptibility to stress have also shown a strong correlation with SP (Denis & Poerio, 2017; Ramsawh et al., 2008; Sharpless et al.,2010). Specifically, Post Traumatic Stress Disorder (PTSD) is highly associated with SP (Denis et al., 2018a; Ramsawh et al., 2008; Sharpless et al., 2010). The correlation of SP with various traumatic events (Denis et al., 2018b) like those caused by child sexual abuse (McNally & Clancy, 2005) is reported by several studies. Another anxiety disorder that has been linked to SP is Panic Disorder (Ramsawh et al., 2008; Sharpless & Barber, 2011; Sharpless & Denis, 2017) although the association between them was not as strong as it was with PTSD (Denis et al., 2018a). Other anxiety disorders that are connected to SP are Generalised Anxiety Disorder and Social Phobia (Denis et al., 2018a).

A link appears to exist between SP and mood disorders as well (Sharpless et al., 2010). Bipolar disorder in particular was significantly related to SP (Ohayon et al., 1999) whereas depression did not have a strong connection to it (Denis et al., 2018a). However by some studies depression is considered an important predictor of SP (Sharpless & Denis, 2017; Sharpless et al.,2010) especially when it is major (Ohayon & Pakpour, 2022). Comorbidity between Anxiety Disorders and Mood Disorders is highly predictive of SP probably because it disrupts sleep to a great extent (Sharpless et al., 2010).

SP appears to have a significant relationship with psychiatric disorders (Sharpless & Barber, 2011) but there is little evidence on the role of psychiatric medication (Denis et al., 2018a; Sharpless et al., 2010). The frequency of SP was neither associated with antidepressants (Sharpless et al., 2010) nor with hypnotics and anxiolytics (Denis et al., 2018a). Nevertheless according to Larkin (1999) anxiolytic medication increase fivefold the likelihood of having a SP episode. It is worth noting though that the participants of this study who were taking anxiolytics also suffered from a mental disorder (Larkin, 1999). Therefore, doubts are raised concerning whether it was the mental disorder or the anxiolytics

that sharply increased the likelihood of a SP episode.

It is not clear whether SP is correlated with a particular substance (Denis et al., 2018a). Alcohol appeared to have a connection to SP without implying that alcohol alone could cause an episode (Denis et al., 2018a; Ma et al., 2014). Caffeine, on the other hand, had no connection to SP (Denis et al., 2018a). As for smoking, the odds of experiencing a SP episode were higher in people who smoked rendering smoking a potential risk factor (Denis et al., 2018a).

Other important risk factors are sleep disorders and poor sleep quality (Denis & Poerio, 2017; Ma et al., 2014). Sleep disorders that tend to co-occur with SP are Narcolepsy, Sleep Apnoea and Night Leg Cramps (Denis et al., 2018a). Moreover a high correlation appeared between SP and circadian rhythm disorders (Denis et al., 2018a). This was made evident by SP episodes in individuals working both day and night shifts and in individuals who had jet lag (Malhotra & Avidan, 2012; Singh et al., 2018). Regarding poor sleep quality a causal relationship was shown between SP and sleep deprivation (Sharpless & Denis, 2017; Singh et al., 2018), symptoms of insomnia (Denis et al, 2018b), daytime sleepiness, disturbed sleep at night with frequent awakenings during night (Denis et al., 2018a) and irregular sleeping patterns (Sharpless & Denis, 2017). Furthermore supine position appeared to increase the chances of SP occurrence (Sharpless et al., 2010) as did sleeping more than 9 hours and less than 6 hours at night and more than 2 hours during the day (Denis et al., 2018a). Midday napping was also reported by a study as a risk factor in vulnerable individuals (Sharpless & Denis, 2017). Those who tended to sleep routinely after midnight had also higher chances of experiencing SP (Denis et al., 2018a). Finally trying to sleep in a state of intense cognitive arousal was a predictive variable of SP (Sharpless & Denis, 2017).

Dreams are considered a potential cause of SP. There is a discrepancy between studies on whether lucid dreams and SP are connected as there are studies which claim that the frequency of SP episodes is not related to lucid dreams (Denis et al, 2018a) while others argue that the two of them are somehow linked to one another (Denis & Poerio, 2017). A common feature that predicted the presence of both lucid dreams and SP was dissociative experiences in the sense of feeling disconnected from oneself and one's environment (Denis & Poerio, 2017). One study speculated that SP that appears in REM sleep intends to inhibit movement during intense and vivid dreams (Torontali et al., 2014). Finally nightmares showed a high correlation with SP episodes (Denis,2018).

Personality traits in general did not appear to be a risk factor for SP (Denis et al., 2018a) with the exception of dissociative experiences (Denis & Poerio, 2017). More specifically people who display feelings of disconnection from themselves and their environment or beliefs that the world around them is not real tend to experience SP very often (McNally & Clancy, 2005). The same people often believe in metaphysical and supernatural phenomena which is also linked to SP (Ramsawh et al., 2008) both in terms of frequency and in terms of intensity (Denis et al., 2018a). Other similar beliefs that appeared to be influential were unusual ideas about what happens to people during sleep (Denis et al., 2018b).

From all of the above it can be easily deduced that SP has not only physical but also psychiatric-psychological causes. Although these variables are not always capable of causing SP alone when they act synergetically they increase the chances of experiencing this sleep disorder.

Methodology

An updated literature review was conducted whose overarching aim was to find out what research and other literature exists concerning SP and LIS. The data were collected from systematic searches using ScienceDirect, Google Scholar, PubMed and Scopus databases for relevant references. Search terms such as “Sleep Paralysis” , “Locked-In Syndrome”, “Sleep paralysis and hallucinations” “Sleep Paralysis and risk factors” and “Locked-In Syndrome and rehabilitation” were used. No language restrictions were applied. A total of 54 articles from years 1993-2022 fully met the criteria and were included in this review. All were peer-reviewed research articles. Two books and two valid websites were used as well.

The similarities between the two disorders are analyzed below. Their common characteristics have to do with the inability to move while maintaining consciousness, the impaired cognitive functions that often occur, the hallucinations that tend to accompany the syndromes and the role of the brainstem.

Voluntary Muscle Paralysis - Preserved Consciousnes

The most striking feature of both disorders is the paralysis of all voluntary muscles in conjunction with preservation of consciousness (Farr et al., 2021; Malhotra & Avidan, 2012). Despite the fact that one syndrome lasts for a few seconds or a few minutes (Hinton et al., 2005b) and the other is a permanent condition (McNair et al., 2019) both are characterized by inability to move with concurrent normal cognitive function and consciousness (Maiser et al., 2016; Malhotra & Avidan, 2012). Therefore people with either SP or LIS are aware of their inability to move but they can neither speak nor move to communicate the state they are in. Another common element is that eye movement is maintained in both syndromes (Maiser et al., 2016; Malhotra & Avidan, 2012). In LIS only vertical eye movement is preserved, which is voluntary (Pacheco-Hernández et al., 2017), whereas in SP eye movement is involuntary and is a feature of REM sleep (Malhotra & Avidan, 2012).

Altered Cognitive Functions

Another similarity is that the cognitive functions of both disorders often appear impaired (De Sa & Mota-Rolim, 2016; Farr et al., 2021; Kumral et al., 2022; Schnakers et al.,

2008). There is not much evidence concerning the cognitive functions of people who experience SP. As previously reported, during SP people generally have normal cognitive function and awareness (Malhotra & Avidan, 2012) of themselves and their surroundings (Sharpless & Denis, 2017). However according to another study due to this mixed sleep and wakefulness state perceptual and cognitive functions in SP episodes appear to be altered (De Sa & Mota- Rolim, 2016).

This is a partly common characteristic with LIS for which a recent study reported that in the early period when LIS was caused by a stroke patients had altered cognitive functions (Kumral et al., 2022).

In particular the cognitive functions of perception, concentration and attention span, but also other abilities such as spatio-temporal orientation, language skills and visual tracking appeared to be impaired (Kumral et al., 2022). It is important to clarify that this was more obvious in patients who had secondary lesions, apart from the lesion in the pons, such as damage to the midbrain (Kumral et al., 2022). However, recovery did occur in one year. Farr and her colleagues (2021) mention that both after stroke and after injury leading to LIS for a short period of time various cognitive functions such as memory and attention are impaired. According to Schnakers et al. (2008) cognitive functions are indeed impaired in the first period following brainstem damage but if the patient has no secondary lesions after six months they are restored. In another survey though it is stated that attention and cognitive functions in LIS remain intact (Bensch et al., 2014).

Hallucinations

Another noteworthy similarity is that hallucinations occur in both syndromes (McNally & Clancy, 2005; Sarà et al., 2018). These illusions stem from neither a neurological disease nor from medication (Hinton et al., 2005a; Sarà et al., 2018). In both syndromes visual as well as movement hallucinations (sometimes linked to pleasant emotions) occur (Sarà et al., 2018; Sharpless & Denis, 2017). Furthermore when people are informed by a professional about the illusions they experience they tend to relax and show signs of improvement (Sarà et al., 2018; Sharpless, 2016).

According to Suni (2022) the hallucinations that occur in SP fall into three categories and they bear no resemblance to classical dreams. The first type is a feeling of suffocation and chest pressure accompanied by thoughts of impending death (Sharpless & Denis, 2017). The second type involves the sensation of a dangerous person/presence invading the room (Suni, 2022). The third type includes movement sensations such as circular and flotation movements as well as the feeling of getting out of bed and starting to walk (Sharpless & Denis, 2017) or the sensation of flying (Suni, 2022). Additionally, many people have stated that they felt as if they got out of their body and saw themselves sleeping (Sharpless & Denis, 2017). The hallucinations of chest pressure and threatening presence often occur together (Suni, 2022). These three illusion categories have been reported by people of different cultures (Sharpless & Denis, 2017) and the majority of them are accompanied by feelings of intense fear (Suni, 2022). As for people with LIS they have reported visual hallucinations of people and objects wandering around hospital wards, sensations of limb movements and other delusions (Sarà et al., 2018).

Brainstem Involvement

As previously reported, LIS is caused by damage to the pons of the brainstem (Law et al., 2018). Surprisingly this part of the brain also appears to be associated with SP (Hishikawa & Shimizu, 1995; Torontali et al., 2014). It is found that dorsolateral parts of the pons are home to nerve cells that are primarily responsible for muscle atonia and other elements of REM sleep (Hishikawa & Shimizu, 1995). In general, brainstem regulates sleep cycles (Varthalitis, 2019). More specifically, cells have been identified in a portion of the brainstem that contribute to the occurrence of SP (Torontali et al, 2014). The activation of neurons in this region either by medication or by electrical stimulation induces a particular type of REM sleep in which the brain is alert while muscle atonia is present which is exactly what happens during SP (Torontali et al., 2014). This region is a nucleus located on the dorsal pons of the brainstem and it is called "sublaterodorsal nucleus" or "subcoeruleus" (Torontali et al., 2014).

Directly related to the brainstem is the neurotransmitter serotonin which is released from neurons based in this part of the brain (Kinney et al., 2009). Serotonin appears to be involved in both LIS and SP as it is often included in patients' medication (Farr et al., 2021; Sharpless & Denis, 2017). Serotonin dysfunction is in fact a possible cause of SP as mentioned above (Sharpless & Denis, 2017).

Differences between SP and LIS

Despite the similarities that these two disorders share they are fundamentally different. Their differences are discussed below in detail. Prevalence, gender, ethnicity, age, duration of the syndromes, hallucinations, the diagnostic procedure used and treatment plan vary besides etiology.
SP is a disorder that occurs in episodes (Denis et al., 2018a) which last from a few seconds to a few minutes (Hinton et al., 2005b). People come out of this state either by an external stimulus or automatically (Malhotra & Avidan, 2012). The great effort people often make to move can also end an episode (Suni, 2022). On the other hand LIS is a permanent condition as it is caused by a stroke or a lesion in the brainstem (León-Carrión et al., 2002).

Prevalence, Gender, Nationality and Age of people with SP or LIS

SP appears to be quite common in the general population (Ohayon & Pakpour, 2022; Olunu et al., 2018; Sharp-

less & Barber, 2011) while LIS is an extremely rare neurological condition (Cardwell,2013; Maiser et al., 2016). It is estimated that there are approximately ten thousand people with LIS worldwide (McNair et al., 2019). Regarding gender SP appears to be more common in women than in men (Ma et al., 2014; Sharpless, 2016) whereas LIS occurs with equal frequency in both genders (León-Carrión et al., 2002). Interestingly SP occurs more frequently in minorities (Sharpless & Barber, 2011) and in people of Asian or African descent than in Caucasians (Jalal & Hinton, 2013; Ohayon & Pakpour, 2022; Olunu et al., 2018) although it is not a cultural phenomenon (Sharpless & Denis, 2017). On the other hand LIS is not related to ethnicity as it is a neurological disorder mainly caused by stroke (León-Carrión et al., 2002). Concerning age SP is more common in younger ages and particularly in adolescence and in the first years of adulthood (Jalal & Hinton, 2013; Ohayon & Pakpour, 2022; Suni, 2022) contrary to LIS which is more common in middle age and specifically in the 40s (León-Carrión et al., 2002; Svernling et al., 2019).

Diagnosis

The diagnosis of the two syndromes is made in a completely different way. The only thing that the two syndromes have in common in the diagnostic process is the electroencephalogram (Kotchoubey & Lotze, 2013; Malhotra & Avidan, 2012). According to the international classification of sleep disorders, as stated by Sharpless and Denis (2017), to get a diagnosis of SP , one must meet four criteria. These criteria are not being able to move the trunk and limbs just before waking up or just before falling asleep, the episodes must last from a few seconds to a few minutes, they must cause negative emotions such as anxiety and fear about sleep, and they must not be better explained by another sleep disorder, by medication or substance use, by a medical problem or even by a mental disorder. The international classification of sleep disorders does not mention a specific number of episodes that a person must experience in order to be diagnosed with SP (Sharpless & Denis, 2017). Besides history taking, an overnight polysomnogram is necessary to diagnose SP or any other parasomnia correctly and this may include an electroencephalogram to rule out other psychiatric or sleep disorders (Malhotra & Avidan, 2012).

The diagnosis of LIS is done in a completely different way as it requires neither history taking nor polysomnogram. However electroencephalography is a common diagnostic method between these two syndromes (Kotchoubey & Lotze, 2013; Malhotra & Avidan, 2012). The diagnosis of LIS is a great challenge as this syndrome is very similar to and therefore confused with various disorders of consciousness such as vegetative state, minimally conscious state (Kuehlmeyer et al., 2012) and coma (Farr et al., 2021). It is easy to misdiagnose LIS with vegetative state or akinetic mutism because the patient exhibits the same behavior in these three disorders (Kotchoubey & Lotze, 2013; Laureys et al, 2005).

To avoid confusion with other disorders a thorough neurological examination must be initially performed and especially examination of oculomotor function (Barbic et al., 2012) because this can determine whether cognitive functions are preserved or not. LIS diagnosis includes Magnetic Resonance Imaging (MRI), a computerized tomography (CT), electroencephalogram and other measurements such as assessment of the amount of oxygen consumed by the brain or the blood that flows in the latter (Kotchoubey & Lotze, 2013). To avoid giving a patient with LIS the wrong diagnosis of coma it is important to observe the sleep-wake cycles (Bensch et al, 2014). The average time until a patient is diagnosed with LIS is approximately two and a half months and more specifically 78 days (León-Carrión et al., 2002). A delayed diagnosis reduces the chances of a better outcome and survival of the patient as opposed to immediate diagnosis (Johnson et al., 2018).

Treatment

The treatment of the two syndromes is diametrically opposed as SP is a psychologically based syndrome that occurs in short episodes while the other is a purely physical, neurological and permanent syndrome. In SP no motor rehabilitation is needed as individuals automatically regain control of voluntary muscles as soon as the episode ends unlike patients with LIS who need motor rehabilitation. The only common way of treatment between these syndromes is serotonin administration (Farr et al., 2021; Sharpless & Denis, 2017).

No large clinical trials have been conducted to find the most appropriate treatment for SP. The most common ways of treatment are psychotherapeutic interventions, changes in daily habits and medication (Sharpless & Denis, 2017). It should be noted that people with SP receive treatment only if episodes cause great discomfort and significantly affect their functionality (Sharpless, 2016). Antidepressants is one way of treating SP using medication (Sharpless & Denis, 2017). These pills often worsen the severity of the episodes and when abruptly discontinued they cause episodes to relapse (Sharpless & Denis, 2017). Another issue that arises with medication is when it should be taken as many times episodes do not occur consistently but show a spike for a period of weeks or days and then are completely absent (Sharpless & Denis, 2017). A study showed that taking serotonin through the antidepressant fluoxetine suppressed in a patient SP episodes as well as the hallucinations that accompanied it (Koran & Raghavan, 1993).

Sometimes replacing unhealthy sleeping habits with new healthier ones and reducing daily stress can be effective treatment options (Sharpless & Denis, 2017). Consistent sleeping habits such as going to bed and waking up almost at the same time every day is reported as a good therapeutic option for treating SP (Ma et al, 2014; Sharpless, 2016). Other healthy sleeping tactics include reducing caffeine and alcohol in the late afternoon (Sharpless, 2016), limiting light and noise, relaxing and removing all electronic devices before bedtime and having an appropriate mattress and pillow (Suni, 2022). The above may prove to be effective treatment options since some of the causes of the disorder are poor

sleeping habits, disturbed and fragmented sleep (Sharpless, 2016) as well as daily stress (Denis & Poerio, 2017).

Regarding the psychotherapeutic interventions used to treat SP some of them are hypnosis, psychoanalysis and cognitive-behavioral therapy (Sharpless & Denis, 2017). The latter includes various techniques such as methods of relaxation and disruption of episodes, ways of managing hallucinations, rehearsing effective episode management and discussing negative thoughts (Sharpless, 2016). Part of psychotherapeutic interventions are techniques for preventing and disrupting SP episodes (Sharpless & Denis, 2017). Prevention strategies include stopping caffeine intake, exercise, relaxation exercises, dietary changes, avoiding stressful topics and changes in sleeping habits (Sharpless & Grom, 2016). Strategies to disrupt episodes involve attempting to move limbs or other parts of the body, attempting to shout, engaging in the hallucination, feeling angry or assertive and attempting to relax (Sharpless & Grom, 2016).

It should be stressed that sometimes the only treatment needed is to inform and reassure people about what this sleeping disorder is (Malhotra & Avidan, 2012; Sharpless, 2016). This is enough for some individuals who are ashamed of the incidents they experience because they think that they are mentally unstable and thus education and reassurance about the phenomenon of SP may cause a reduction in episodes (Sharpless, 2016). Larkin (1999) believes that complacency is the most appropriate way to handle SP if no comorbidity with another disorder is present.

LIS on the other hand is not a curable condition as patients never fully recover (Papadopoulou et al., 2019). Svernling et al. (2019) in a study reviewed by Cerasa and Becker on LIS in Sweden stress that there are ways of treatment and rehabilitation methods that help patients to achieve the greatest possible improvement and thus a good quality of life. Patients with LIS can show a remarkable improvement (Kumral et al., 2022). The rehabilitation of LIS is similar to the rehabilitation of any severe stroke (Farr et al., 2021) and depends on the extent of the damage (Kumral et al., 2022). Assessment of the individual's needs and abilities should precede any treatment method and be repeated at different stages of rehabilitation (Schjolberg & Sunnerhagen, 2012). Intensity of treatment, multidisciplinary cooperation and early initiation of rehabilitation play a major role in rehabilitation by reducing the chances of demise and by paving the way for improvement (Casanova et al, 2003).

Another important issue in the treatment of patients with LIS is their capability of making decisions regarding their own rehabilitation (Maiser et al., 2016). This is because many patients who have other lesions in the brain besides the lesion in the pons, such as damage in the midbrain, have great difficulties in perception, concentration, and other cognitive functions (Kumral et al., 2022). Patients who have normal cognitive functions are required to participate in the decision making process for their rehabilitation (Maiser et al., 2016) by first expressing whether or not they accept some kind of treatment (Laureys et al., 2005).

Rehabilitation methods and ways of coping with LIS include physiotherapy, speech and language therapy, cognitive therapy (Kumral et al., 2022), occupational therapy, movement therapy, eye movement exercises (McNair et al., 2019), medication, eye care, feeding and breathing care, bladder and bowel care, pain management and of course ensuring ways of communication (Farr et al., 2021). Assistive Technology and specifically Alternative and Augmentative Communication systems are essential to ensure the patients' communication as only with these can a patient with LIS communicate (Farr et al., 2021). In particular an eye-based word processor, speech synthesizers (Laureys et al., 2005), picture boards, low-pressure touch surfaces and computer brain interface systems can be used (Farr et al., 2021).

Physiotherapy is used because it tones body muscles and reduces spasticity (Casanova et al., 2003). Although it does not always result in regaining mobility it can improve the patients' quality of life (Pacheco-Hernández et al., 2017). Another essential rehabilitation method is motor therapy which includes changing posture and turning the patients' head to improve its mobility, a tilt table which tones neck and trunk muscles (Farr et al., 2021) and use of technological devices such as «Armeo Spring robotic upper limb» and FES (Functional Electrical Stimulation) cycling (McNair et al., 2019). Finally medication is used among others to treat pain (Bonin et al., 2022) and to regulate the pathological crying and laughing that patients with LIS often display by administering serotonin (Farr et al., 2021).

Hallucinations

Differences between the two syndromes can also be found in the incidence of hallucinations. In LIS hallucinations are rarely reported (Sarà et al., 2018) while the hallucinations that accompany SP are very frequent as they appear in 75% of SP episodes (Suni, 2022). Incidence of hallucinations in patients with LIS may actually be higher than the recorded because patients either have no way of expressing it or because they would rather communicate more urgent physical needs (Sarà et al., 2018). Regarding the content of illusions in SP people experience visual, auditory and tactile hallucinations whereas patients with LIS only report visual and movement illusions (Sarà et al., 2018; Sharpless & Denis, 2017). Contrary to SP in LIS feelings of chest pressure, illusions of grasping and whispering and feelings of a demonic or threatening presence have not been noted.

Another difference lies in the etiology of hallucinations. SP illusions may stem from social anxiety, traumatic experiences and particularly childhood sexual abuse, dissociative experiences as well as from faith in metaphysical phenomena (Denis et al., 2018a). They may also derive from intrusion of fantasy that characterizes dreams that occur in REM sleep into a wakefulness state of the brain (Sharpless & Denis, 2017). LIS hallucinations on the other hand are physically explained. They may either result from various secondary lesions in the cerebral cortex following damage to the pons or from reduced volume of some brain regions (Sarà et al., 2018). This brain atrophy is present only in patients with LIS who experience hallucinations and not in all patients with LIS (Sarà et al., 2018). LIS hallucinations have been linked to midbrain lesions that some patients have (Andrews et al., 2016). As for movement hallucinations Sarà and his col-

leagues (2018) hypothesize that lesions in the prefrontal cortex of the brain combined with dysfunction of corticocerebellar pathways probably lead to an inability to perceive the actual movements that patients can make which in turn results in patients thinking that they can move their limbs. The most likely explanation for LIS illusions is that they emerge due to the synergy of several dysfunctional areas linked to one another and not because of a single pathological region (Boes et al., 2015). It has become clear that explanations for LIS hallucinations are primarily neurological, although not firmly established, whereas in SP are more psychological.

Discussion

From the above literature review some notable issues emerge that need to be discussed. The two syndromes share the commonality of paralysis of all voluntary muscles while maintaining consciousness (Farr et al., 2021; Malhotra & Avidan, 2012). What mainly differs is that LIS is a rare disorder (Cardwell, 2013) leading to a permanent condition with high mortality rates (McNair et al., 2019) while SP is quite common in the general population (Ohayon & Pakpour, 2022) occurs in sporadic episodes (Denis et al., 2018a) and lasts only a few seconds or minutes (Hinton et al., 2005b).

Their causes are also very different. LIS has purely physical-neurological causes (Kumral et al.,2022; Law et al., 2018) while SP has some physical causes and interpretations (Malhotra & Avidan, 2012; McNally & Clancy, 2005; Sharpless & Denis, 2017) but is being triggered mainly by psychological causes (Denis et al., 2018a; Sharpless & Barber, 2011). The latter may explain the higher incidence of SP in younger people (Suni, 2022) and in women (Ma et al., 2014; Sharpless, 2016) as adolescence and young adulthood is a period of change and instability accompanied by intense stress for many people. Women experience anxiety disorders more frequently than men (McLean et al., 2011). Therefore they also experience SP episodes more frequently (Ma et al., 2014; Sharpless, 2016) since SP episodes are often triggered by stress (Ramsawh et al. 2008; Sharpless et al., 2010). Given that the causes of the two syndromes are entirely different (Denis et al., 2018a; Kumral et al., 2022), distinct duration, diagnosis and treatment are to be expected (Farr et al., 2021; Hinton et al., 2005b; Kotchoubey & Lotze, 2013; Malhotra & Avidan, 2012; Sharpless & Denis, 2017). Electroencephalography and serotonin administration are the only exceptions as the former helps in diagnosing both disorders (Kotchoubey & Lotze, 2013; Malhotra & Avidan, 2012) and the latter is a common way of treatment in both syndromes (Farr et al., 2021; Sharpless & Denis, 2017). The reason why SP appears at a higher rate in Asians and black people remains unanswered (Jalal & Hinton, 2013; Ohayon & Pakpour, 2022).

The literature shows that in the brain the pons of the brainstem appears to be involved in states of simultaneous body paralysis and brain wakefulness as it is linked to both disorders (Law et al., 2018; Torontali et al., 2014). Regarding LIS the lesion in the pons of the brainstem causing it is clear (Law et al., 2018) unlike the involvement of the brainstem in SP which raises a lot of questions (Torontali et al., 2014). One of these questions is whether the cells in the brainstem that contribute to the occurrence of SP (Torontali et al., 2014) are present in the general population or only in those who experience SP and whether these cells are capable enough of causing SP on their own. If these cells are found in the general population they probably act synergetically with other factors rather than causing SP on their own. Otherwise everyone would suffer from this sleep disorder. It has also not been investigated whether the activation of neurons in the brainstem nucleus that produces sleep similar to that of SP (Torontali et al., 2014) is responsible for the episodes or it happens after the episode has started. If this was responsible a way of suppressing the stimulation of that area could be found. Therefore it needs to be investigated if SP is ultimately the result of a transient dysfunction of the pons of the brainstem which regulates sleep cycles (Varthalitis, 2019).

The involvement of the neurotransmitter serotonin is also noteworthy to mention as increasing its levels is included in the treatment of both syndromes (Farr et al., 2021; Sharpless & Denis, 2017). The state of simultaneous brain awareness and body paralysis appears to be connected to serotonin (Farr et al., 2021; Sharpless & Denis, 2017). This neurotransmitter is released, as mentioned above, by neurons of the brainstem (Kinney et al., 2009). Hence, since this area of the brain is affected in LIS (Young, 2014), and possibly in SP (Torontali et al., 2014), the normal amount of serotonin may not be produced causing people with any of these syndromes need medication to increase its levels (Farr et al., 2021; Sharpless & Denis, 2017). In SP it may work the other way around as low serotonin levels may be responsible for the dysfunction of the brainstem leading to SP episodes (Sharpless & Denis, 2017). In any case it is an issue that needs to be further investigated.

Another issue arising from this review is the unexpected presence of hallucinations in both disorders. This combination of inability to move and brain activity appears to be frequently accompanied by hallucinations (Sarà et al., 2018; Suni, 2022). Although several possible causes of hallucinations have been put forward they have not been scientifically proven and it has not been clarified whether their causes are physical or psychological (Denis et al., 2018a; Sarà et al., 2018). LIS hallucinations are neurologically explained (Sarà et al., 2018) while SP illusions are mostly psychological rather than physical (Denis et al., 2018a). It needs to be further investigated if there is a specific lesion in any part of the brain responsible for the incidence of hallucinations in both syndromes. The brainstem which is involved in both disorders (Law et al., 2018; Torontali et al., 2014) may be somehow related. It has not been studied if the reduced volume of some brain regions found in patients with LIS and hallucinations (Sarà et al., 2018) is also present in people with SP and hallucinations.

Depression, anxiety and disturbed sleep cycles may also affect illusions. Perhaps mental instability with its accompanying negative emotions or dissociative experiences can explain the incidence of hallucinations (Denis et al., 2018a) in both disorders. It may even be that the hallucinations in LIS are inextricably linked to this mixed state of consciousness consisting of REM sleep and wakefulness features, as

in SP (Sharpless & Denis, 2017). It is not known whether patients with LIS experience illusions when they are awake, asleep or in a sleep-wake transition state or vice versa, as in SP (Sharpless & Denis, 2017).

Whatever the etiology illusions in SP and possibly in LIS are usually accompanied by strong negative emotions (Suni, 2022). It is possible that they are frightening and thus experienced as negative experiences because they reflect peoples' awareness of their inability to move whereas movement hallucinations are more often accompanied by positive emotions (Sarà et al., 2018; Sharpless & Denis, 2017) because they reflect a strong desire for physical movement. It is still no coincidence that the same type of hallucinations in SP have been reported by individuals of different cultures making it clear that this is not a cultural phenomenon (Sharpless & Denis, 2017).

As for the hallucinations of people with LIS they have not been adequately researched as they are mentioned only by one study (Sarà et al., 2018). There may be many people with LIS experiencing hallucinations who have no means of communicating their illusions and it is also likely that they do not share their illusions because there is an urgency to express more primary needs such as pain relief (Sarà et al., 2018). Lastly some ways of preventing and disrupting episodes of SP may also be beneficial in treating hallucinations of people with LIS.

Regarding cognitive functions it remains unanswered to what extent they are maintained in SP beyond awareness of the immediate surroundings and self-awareness (De Sa & Mota-Rolim,2016). To elaborate it is not known how long-term and short-term memory, attention span, selective attention and range of attention as well as auditory perception and discrimination are affected. During SP episodes it is reported that cognitive functions appear altered without specifying which functions are impaired and in what way (De Sa & Mota-Rolim, 2016).

As for LIS it has been found that when it stems from stroke some cognitive functions and other abilities are impaired in the early period following stroke but that they are restored within a year (Kumral et al., 2022) when there are no additional lesions besides the lesion in the pons (Schnakers et al., 2008). It is of interest to the reader to know what accelerates or hinders the recovery of cognitive functions. It is possible that this happens automatically when balance is restored in the brain and new static patterns of brain function are established after the stroke (Conrad et al., 2021). Another hypothesis is that some specific rehabilitation methods contribute to regaining cognitive functions (Kumral et al., 2022). Finally it would be interesting to further investigate how specific cognitive functions such as auditory and visual perception and different types of memory such as visual and auditory, long-term and short-term memory are affected (Schnakers et al., 2008). It is important to clarify the above in order to optimize or design new ways of communicating with LIS patients (Schnakers et al., 2008) and new rehabilitation methods but also to understand if and when they can participate in decision making concerning the prolongation of their lives and their rehabilitation.

Future directions

The above literature review reveals some research gaps that need further investigation. These gaps concern the hallucinations of the two disorders, the role of the brainstem in SP and the cognitive functions of the two syndromes. It would be useful if future research compared the hallucinations of the two disorders to identify the cause of them and to find out which way of treatment is the most effective in reducing or eliminating them. The comparison would be helpful because a potential cause of SP hallucinations may also be a cause of LIS hallucinations and vice versa , or an effective way of managing illusions of one disorder may seem beneficial for the other. Identifying their etiology would probably shed light on what the best treatment is. The above aim to eliminate hallucinations for a better quality of life of people who either suffer from LIS or from SP. It would also be beneficial if future research continues to investigate the role of the pons, the brainstem and the neurotransmitter serotonin in the occurrence of SP. It is important to find out whether this sleep disorder is the result of a dysfunction of specific brain structures or neurotransmitters because this may lead to an appropriate medication treatment and ultimately to the prevention of SP incidence.

Finally future studies should explore the cognitive functions of people with LIS or with SP as well. This would be particularly helpful for patients with LIS. Factors that delay or accelerate the recovery of cognitive functions and whether they are impaired in all patients with LIS or not should be studied . Furthermore it would be interesting to study for how long cognitive impairment lasts, on what this recovery depends and whether all cognitive functions recover or only a part of them. These would show whether and when these patients are able to participate in decision-making about their rehabilitation and prolongation of their lives. It would even help to optimize or find new ways of communicating with them. For instance if visual processing and visual perception were found to be impaired communication methods using mainly auditory stimuli could be designed or if patients were found to have poor judgment in the first six months after LIS they could be interviewed after these months to participate in medical decision making. Concerning the cognitive functions of SP it would be interesting to look into them in the future to see the effect of sleep on cognitive functions and how these functions are affected by this rare state of wakefulness and REM sleep consciousness. More research is needed to better understand the underlying processes and structures involved in LIS and SP.

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