REVIEW ARTICLE

Parasomnias: A review with approach to patients in the clinic and management principles

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Abstract

Parasomnias are characterized by undesirable motor, verbal or experiential phenomenon / behaviors occurring in relation to sleep and sleep wake transition phases. They are believed to occur due to incomplete arousal from different sleep states, abnormal intrusions of wakefulness into different stages of sleep as well as de-afferentation of generators of locomotion from generators of sleep. They are classified as sleep wake transition parasomnias, NREM (non-rapid eye movement), REM (rapid eye movement) parasomnias and miscellaneous group.Accurate diagnosis of various parasomnias is important as they are associated with distinct group of predisposing factors.Associated conditions that require different treatment considerations which may be as innocuous as identification and removal of predisposing factors and reassurance. Correct diagnosis also invokes right differential diagnostic considerations and further investigations. Understanding of the underlying pathophysiology, semiological features and natural history of the particular parasomnia aids in counseling the patient and the parents, safety precautions to be undertaken and addressing psychosocial complications which are one of the most distressing factors for the patients. Correct diagnosis can be established fairly accurately based on careful and detailed clinical interview (including account from parents and bed partner), age of onset, time of occurrence, polysomnographic (PSG) studies and rarely an extended montage to study electrographic characteristics for differentiation with an ictal event.

Keywords: Parasomnia, sleep start, confusional arousal, nightmares, sleep bruxism. rhythmic movement disorder, sleep walking, sleep paralysis, sleep enuresis, sleep talking, sleep terror, REM Sleep Behavior Disorder, nocturnal paroxysmal dystonia, nocturnal leg cramps, impaired sleep related penile erections, catathrenia, sleep related eating disorder

Abbreviations: REM – rapid eye movement, NREM – non-rapid eye movement, PSG – Polysomnography, EEG – electroencephalography, CNS – central nervous system, ICSD – international classification of sleep disorders, EMG – electromyography, BXD – benzodiazepines, TCA – tricyclic antidepressant, PTSD – post-traumatic stress disorder, ISP – isolated sleep paralysis, RISP – recurrent isolated sleep paralysis, RBD – REM sleep behavior disorder, OSA – obstructive sleep apnea, NPD – nocturnal paroxysmal dystonia, UTI – urinary tract infection, NES – nocturnal eating syndrome, RLS – restless leg syndrome, PLMS – periodic limb movement of sleep, RDI – respiratory disturbance index, SPECT – single photon emission computerized tomography, MAO – monoamine oxidase, SRED – sleep related eating disorder, CPAP – continuous positive airway pressure.

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Introduction

Parasomnias are a diverse group of sleep disorders which can be broadly defined as undesirable events or behaviors occurring at various stages of sleep and sleep wake transitions phases[1, 2]. These diverse undesirable events may be motor, verbal or experiential and are manifestations of CNS activation, involving activation of systems (autonomic nervous system) or programs (eg: motor, behavioral, cognitive)[1,3]. In this review we will discuss various parasomnias, their clinical features, brief note on treatment of each individual parasomnias, approach to evaluation of parasomnias, and broad management principles.

There are two schemes used for classification of parasomnias - International classification of sleep disorders, the fourth iteration of which was published in2012 (ICSD II) dividing parasomnias in four categories (arousal, sleep wake transition disorder, parasomnias associated with REM sleep and miscellaneous group)[1] (Table 1) and classification based on Diagnostic statistical Manual of Mental Disorders, Vth edition (nightmare disorder, sleep terror disorder, sleepwalking disorder, and parasomnias not otherwise specified). In the ensuing sections, we will divide parasomnias in four categories (sleep wake transition disorder, disorders of arousal during NREM sleep, REM sleep disorder and miscellaneous group) to better illustrate the underlying manifestations, etiopathogenesis and neurophysiological features[4, 5, 6].

A. Wake sleep transition disorders occur during the transition from wakefulness to sleep and include sleep starts, rhythmic movement disorder, sleep talking, and nocturnal leg cramps[1, 5,7,11].

Sleep starts, also referred to as hypnagogic jerks, hypneic jerks or pre-dormital myoclonus, occur at the transition from wake to sleep state and involves isolated, bilateral, brief jerks, often awakening the person[5, 7, 8]. These usually involve the legs but can also affect the arms and head, can be asymmetric and are often multiple, occurring in succession. They are sometimes associated with various sensory phenomenon such as flashing lights, vivid dreams, hypnagogic hallucinations and subjective sensation of falling and sometimes aggressive and violent behavior.

These are believed to be secondary to imbalance between hypnagogic and wakefulness structures as the person is falling asleep thereby leading to various sensory

Table 1: Classification of Parasomnias

Sleep Wake Transition Disorders	NREM Sleep Disorders (Arousal Disorders)	REM Sleep Associated Parasomnias	Other Parasomnias
Sleep Starts	Confusional Arousals	Nightmares	Sleep Bruxism
Rhythmic Movement Disorder	Sleep Walking	Sleep Paralysis	Sleep Enuresis
Sleep Talking	Sleep Terrors	REM Sleep Behavior Disorder (RBD)	Nocturnal Paroxysmal Dystonia
Nocturnal Leg Cramps		Impaired Sleep Related Penile Erections	Sleep Related Abnormal Swallowing Syndrome
		Sleep Related Penile Erections	Sudden Unexpected Nocturnal death Syndrome
		REM Sleep Related Sinus Arrest	Primary Snoring
			Infant Sleep Apnea
Hypoventilation Syndrome			Congenital Central
			Sudden Infant Death Syndrome
Myoclonus			Benign Neonatal Sleep
			Other Parasomnias NOS
			Sleep Related Eating Disorder (SRED)
			Sleep Related Groaning (Catathrenia)

and motor phenomenon described above. As would be expected, anything that disrupts this balance such as excessive stimulants (alcohol, coffee), excessive exercise before sleep and stress would lead to worsening of this normal physiologic event. Sleep starts occurs at all ages and no gender bias has been noted.

Electrophysiology studies show that sleep starts occur during drowsiness or stage 1 sleep with vertex waves during the jerks[9]. There are no epileptiform sharp waves preceding those jerks differentiating it form various pre-dormital epilepsies. Gastaut and Broughton studied EMG characteristics and noticed 75-250 msec high amplitude potentials occurring bilaterally over affected regions[10].

Differential diagnostic considerations includes fragmentary partial myoclonus, hyperekplexia syndrome (pathologic response to sensory stimulus during wakefulness), brief epileptic myoclonus, periodic movement of sleep (are longer, occur in all stages of sleep and have pseudo-periodic reps), restless leg syndrome

Diagnosis of sleep starts is straightforward being based on history, semiology and associated features[11].

Treatment involves avoidance of precipitating factors such as stress and stimulants such as exercise and coffee, and irregular sleep wake pattern leading to instability of the structures mentioned above. In severe cases, benzodiazepines such as clonazepam and diazepam can be used.

Rhythmic Movement Disorder, also known as jacatatio capitis nocturna, rhythmie du sommeil and multiple other synonyms, is characterized by repetitive, rhythmic stereotyped movements involving large body areas such as head and neck or entire torso exemplified by head banging, body rocking, body rolling occurring prior to sleep onset or in light sleep[2,4,5,6,11,12,13]. It usually lasts about for few seconds, can occur in long clusters and is sometimes associated with rhythmic chanting or vocalizations.

It is typically considered a disorder of infancy and early childhood (mean age of onset is 6 months) with spontaneous resolution during adolescence. However it can occur at any age and may persist in adulthood. It is more common in males with M: F ratio of 3:1. If it appears later or persists during adulthood, it is often associated with underlying organic brain disease and mental retardation, autism and may lead to various physical as well as psychological issues[14]. It is presumed to have some genetic component as well it is more frequent in twins.

EEG recordings are dominated by movement artifacts (Fig 1), usually in stage 1 or stage 2 of sleep[10, 15, 17, and 18]. If the patient has long clusters they can persist in or sometimes even occur exclusively in REM sleep[16] or deep slow wave sleep state. A characteristic finding that aids in differentiation from other rhythmic movement disorders is no EEG changes indicating arousal during or after this complex motor behavior.

Stereotypical behavior such as bruxism, thumb sucking behavior, periodic movement of sleep and epileptic phenomenon are relatively easy to separate based on semiology and EEG changes.

Most cases subside by adolescence, however if it persists, workup for underlying psychiatric or neurological disorders is warranted. Sometimes

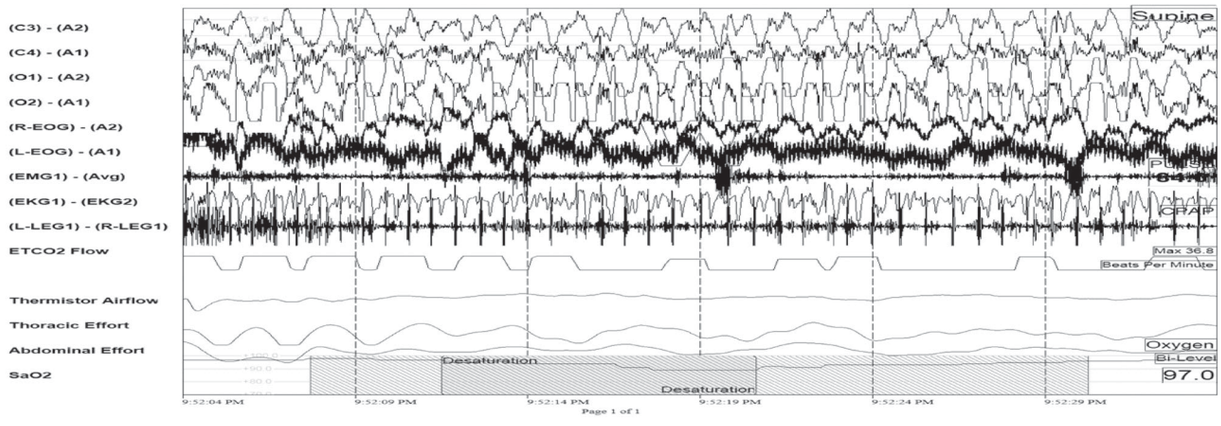

Figure 1: PSG epoch in a child with rhythmic movement disorder. Note the rhythmic EEG artifact.

considering the nature of movements, protective precautions such as helmet or padding might be necessary. Various behavior modification therapies are being increasingly used for severe or persisting symptoms. Benzodiazepines and tricyclic antidepressants have been tried with variable amount of success[18, 5, and 6].

Sleep talking also known as somniloquy is characterized by talking out loud during sleep or "utterances of speech or sounds during sleep without simultaneous subjective detailed awareness of the event"[1, 21]. It is a relatively common disorder that runs a self-limited benign course lasting days or months and occasionally years with some element of heredity suggested by various studies[20]. Sleep talking is usually precipitated by a stressful condition such as emotional stress or febrile illness or anxiety but may be seen in association with apneic events among those with obstructive sleep apnea. It happens during partial wakefulness during sleep stage transitions and can occur in all stages of sleep (NREM, REM and slow wave sleep) and frequently occurs in association with various NREM and REM sleep behavior disorders.

Nocturnal leg cramps are relatively common nocturnal, unpleasant, painful sensation of recurrent muscle tightness/cramping of the calf or foot muscles secondary to involuntary muscle contractions lasting few seconds to minutes and frequently wakes up people from sleep[1, 22]. It is a very common disorder with at least 60% of total population and 16% of healthy population having experienced it one time or the other in life with no familial or sex preponderence[1, 22]. It is associated with a host of physiological states (late pregnancy, dehydration, fatigue) , pharmacological agents (intravenous sucrose, diuretics, statins, long acting beta agonists, conjugated estrogens, raloxifene, naproxen, teriparatide), electrolyte abnormalities and disease processes (restless leg syndrome, myositis, lumbar stenosis, peripheral neuropathy). There should be no underlying medical disorder for satisfying the criterions for diagnosis of sleep related nocturnal leg cramps. It is a benign, self-limited disorder which is usually relived by massaging the muscles, leg shaking or walking around and in severe cases can lead to insomnia and daytime fatigue. Polysomnographic studies show non-periodic bursts of EMG activity in the affected muscles[24]. There is limited evidence for treatment of severe cases with magnesium supplements, vitamin B12, calcium channel blockers, or carisoprodol[22].

B. NREM Parasomnias or disorders of arousal are by far the most common type of parasomnias. They occur because of partial or impaired arousal during the transition from deep slow wave sleep (Stage 3 and 4 of NREM sleep) to lighter stages of sleep or intrusion of the waking stage in NREM sleep.

Confusional arousal is characterized by marked confusion, slow mentation, disorientation and perceptual impairment during and after arousal which is oftentimes accompanied by inappropriate and/or aggressive behavior, lending it various names or eponyms such as nocturnal sleep drunkenness, excessive sleep inertia, l'iveresse du sommeil, schlaftrunkenheit[1]. It may last for minutes to hours with the patient having complete amnesia for the eventusually occurs in the first third of the night.

Confusional arousals are ubiquitous during early childhood[1, 25] with gradual resolution over few years with prevalence rate of 4.2% in adult general population[5, 626, and 27]. There is often a family history of sleep disorders. Predisposing factors include any factors that impairs awakening and predispose to deep sleep such as sleep deprivation such as alcohol, CNS depressants, sedatives hypnotics and tranquilizers; medical factors affecting sleep and arousal mechanisms such as fever, toxic and metabolic encephalopathy's; circadian rhythm sleep disorders and sleep deprivation, various hypersomnia's and sleep apnea to name a few.

PSG and EEG recording shows that it is most frequent during the first third of night during slow wave sleep. Less frequently it arises from lighter stages of NREM sleep and rarely during REM sleep and daytime naps[10, 27]. Proposed etiologic mechanisms and pathophysiology: EEG during the episode shows remnants of sleep patterns such as slow wave sleep, stage 1 theta pattern, poorly reactive alpha rhythm and periods of micro sleep[10, 27].

Differential diagnostic considerations include sleep walking, night terror, REM sleep behavior disorder and nocturnal complex partial seizures. Of note, it frequently occurs with sleep walking and sleep terrors.

No treatment is necessary considering benign course. Avoidance of precipitating factors is recommended and in individuals with serious impairment mild stimulants, behavioral and safety measures might be tried.

Sleepwalking or somnambulism is characterized by recurrent episodes of complex automatic behaviors during slow wave sleep (N3, N4 NREM) during first third of the night[1, 5, and 6]. It can involve a whole range of complex

behaviors such as a child arising from deep sleep and walking to the parent's bedside to adults getting up and walking around the house or outside, trying to get out of the window, indulging in complex task such as preparing meals, eating food, talking to oneself or even driving. Owing to their lack of / or altered awareness, subjects may injure themselves during the event or may become very aggressive and violent on attempts to restrict their mobility. It restricts by itself or on forced awakening with subjects expressing complete amnesia of the event and marked confusion upon awakening.

It is a very common childhood parasomnia that can start as soon as the child learns to start walking with peak incidence between 4-8 years of age[1, 25]. It is less frequent in adolescence and adults with prevalence in general population of 1-15%[1, 26]. There is often a strong family history of similar disorders and the incidence increases with parents or first degree relatives being affected[28]. Precipitating factors include factors that either deepens sleep or disrupts sleep such as sleep deprivation, stress, pain, fever, sleep apnea or distended bladder, medications (CNS depressants, lithium, thioaridazine, prolixin, perphenazine, chloral hydrate,desipramine)[1]. More recently, GABA-B agnostic such as Zolpidem with their increasing use have been implicated in causing this parasomnia. Somnambulsim may be associated with sleep apnea in children.

PSG and EEG recordings show sleep walking to occur most frequently during first third of night during stage 3 and 4 sleep, particularly the end of 1st or 2nd episode of slow wave sleep[10]. During the event, EEG shows lightening to stage 1 pattern or abnormal diffuse non-responsive alpha rhythm and frequent micro arousals with no autonomic activation on other channels[29].

Based on its characteristics, it is easy to differentiate this entity form other sleep disorders, frontal lobe epilepsy and complex partial seizures.

No therapy is indicated in children. Parental reassurance with advice to take safety precautions to prevent harm is advised. Hypnosis and psychotherapy has been tried with variable results. Among children with sleep apnea, treatment of the apneas may result in improvement of somnambulism[67]. Benzodiazepines such as diazepam, clonazepam and oxazepam as well as tricyclic antidepressants such as clomipramine, imipramine and amineptine have been tried with modest success.

Sleep terrors sometimes referred to as night terrors, pavor nocturnus (in children), incubus attacks (in adults) ischaracterized by sudden arousal from deep sleep usually during the first third of the night with patient emitting a piercing scream or blood-curdling cry and is associated with varying degrees of autonomic, behavioraland motoric manifestations of intense fear[1,5,6]. They are distinct from nightmares or terrifying dreams occurring during REM sleep in last third of the night. Patients usually haveno or little recollection of the whole dream sequence, show autonomic hyperactivity manifested as tachycardia, flushing, diaphoresis, mydriasis associated with increased muscle tone and sometimes vocalizations or involuntary micturition[1].

It often begins in childhood with peak incidence in the 4-12 year age range and prevalence of with rough incidence of 6% in children and 1% in adults[1, 25, 26]. In children, it is considered benign and tends to disappear later in adolescence. However in some cases it may persist or appear in adulthood. In case of adult onset, there is higher incidence of underlying psychopathology. Various epidemiological studies have also shown male preponderance and familial predeisposition[1, 26, 28].

Polysomnographic studies shows that sleep terrors usually arise during slow wave sleep (N3, N4 of NREM sleep) in first third of the night with evidence of autonomic hyperactivity in EKG leads and EMG artifacts.

As mentioned earlier, they usually run a benign course with disappearance later in life and no specific therapy apart from reassurance is necessary in most case. Benzodiazepines and tricyclic antidepressants have been tried with variable success rates.

C. Parasomnias of REM Sleep:

REM sleep disorders have some unifying underlying pathophysiologic mechanisms and manifestations. They usually occur in last half of the night during REM sleep stage with age of onset in late middle ages, have strong male preponderance, commonly awakening the person from sleep with almost complete recollection of the dream sequences and are associated with lack of motor features.

Nightmare Disorder also referred to as terrifying dreams, dream anxiety attack or REM nightmares is distinct from sleep terrors described above and represents "recurrent episodes of awakening from sleep with recall of intensely disturbing dream mentation, usually involving fear or anxiety, but also anger, sadness, disgust, and other

dysphoric emotions"[1], occurring during second half of the night during REM sleep leading to awakening[1, 2, 5, 6]. In contrast to sleep terrors nightmares can be associated with mumbling, vocalizations or movements prior to awakening with minimal autonomic over activity, preserved sensorium following awakening and detailed recollection of the dream content.

Nightmares are quite common in childhood with estimated prevalence of 10 to 50% in children aged 3 to 6 yrs. and occasional occurrence in 40 to 50% of adults with female preponderance in adult population groups[1,5,30]. Nightmares may appear or persist in adolescence or adulthood, can be recurrent, and in about50% of such cases they are frequently associated with underlying psychopathology or characteristics such as difficult childhood, prior psychological traumatic event, vulnerable and artistic people borderline personality disorder, schizophrenia, PTSD and stress[1, 31, 32]. In majority of patients the frequency and severity decreases over time. Medications such as dopamine agonists, beta blockers, withdrawal from REM suppressant medications such as tricyclic antidepressants (TCA) and monoamine oxidase (MAO) inhibitors, alcohol, stimulants and various sedatives and hypnotics may worsen or induce nightmare's[1].

PSG recording shows occurrence during REM sleep associated with increases eye movement and eye twitches and mildincrease in autonomic activity. Nightmares associated with prior trauma may occur in stage 1 and 2 of NREM sleep.

Distinction from other sleep disorders is relatively easy based on aforementioned characteristics.

Treatment is necessary only in severe and disabling cases and involves a multidisciplinary approach including cognitive behavioral therapy, psychotherapy, stress reduction, sleep hygiene, withdrawal of predisposing drugs and rarely use of REM sleep suppressants such as TCA and MAO inhibitors[1,5,65].Alpha blocker such as Prazosin has been shown to be particularly effective for PTSD associated nightmares[65].

Recurrent isolated Sleep paralysis as the name suggests is characterized by transient inability to perform voluntary motor movements occurring at sleep onset (hypnagogic or pre-dormital) or upon awakening (hypnapompic or post-dormital)during sleep wake transition stages[1, 5]. At the time of its occurrence subject cannot move or speak but the eye movements and voluntary breathing activities are preserved. It can be associated with fragmentary recollection of the dream sequences or hypnagogic and hypnopompic hallucinations. It is usually the complete inability to move that leads to a frightening experience. It lastsone tofew minutes and is aborted spontaneously or on stimulation by the partner.

Sleep paralysis is a very common occurrence having occurred atleast once in lifetime of 40-50% of normal individuals with a prevalence of around 6% in the general population[33]. It exists in two forms: isolated sleep paralysis (ISP) oras recurrent isolated sleep paralysis (RISP) and as part of narcolepsy-cataplexy tetrad (cataplexy, sleep paralysis, hypnagogic hallucinations and excessive daytime sleepiness). There has been no sex predilection reported.Rare familial form of sleep paralysis is chronic and x linked with female preponderance[34].

Pathophysiologically it is considered as persistence of REM sleep associated atonia in wakefulness which occurs during REM/NREM sleep transition periods.Consequently, it is seen more often in association with irregular sleep wake cycle such as seen in shift workers, people flying between time zones, subjects with circadian rhythm disorders. Sleep paralysis have also been associated with anxiety, depression, bipolar disorders, migraine, and sleep apnea[33].

PSG recording shows characteristic EEG pattern of wakefulness including blinking, ocular movements with EMG demonstrating absence of movement artifacts and loss of H reflex during nerve conduction studies in the setting of relatively normal MSLT and the recordings demonstrating occurrence of events after direct transition into or from REM sleep.

Differential diagnostic considerations include narcolepsy, cataplexy,familial forms, hysteria, hypokalemic periodic paralysis and atonic seizures. Differentiation is relatively easy based on clinical characteristics and PSG findings.

Treatment involves sleep hygiene and withdrawal of the drug in chronic cases associated with other syndromes REM suppressants such as, clomipramine andother anti-depressants[35]. MAO inhibitors such as phenelzine can be used in resistant cases.

REM Sleep Behavior Disorder: is manifested by absence of normal atonia of REM sleep followed by dream mentation and dream enactment manifested as explosive motor activity characterized by sudden limb jerks, violent

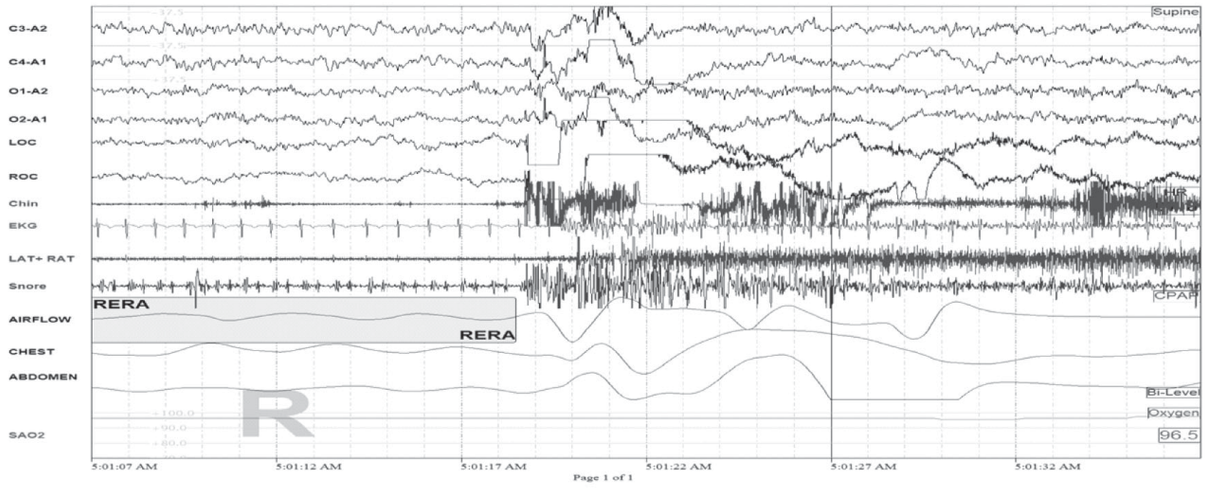

Figure 2: PSG Epoch in a patient with RBD. Note the sudden loss of EMG tone in REM sleep and over motor activity.

movements, complex behaviors such as gestures, waving, running into walls or furniture to name a few[1,36,37]. The episodes may occur relatively infrequently or many times a night occurring in cyclical pattern of every 90 minutes. Subjects have vivid recollection of the dream and their violent behaviors are manifestations of themselves reacting to the unpleasant dream. RBD may be acute in cases associated with drugs or abrupt drug withdrawal or may be chronic especially when idiopathic or precipitated by other disorders.

Sixty percent of the cases are idiopathic with the remainder being associated with underlying pathology such as various neurodegenerative disorders and dementias, olivopontocerebellar atrophy, multiple sclerosis, stroke, pontine lesions and in chronic alcoholics, withdrawal from sedative hypnotic drugs, tricyclic antidepressants and biperiden[1, 38, 39]. Etiology is generally believed to be organic despite the test results and is presumed to be secondary to dysfunction of brain stem mechanisms responsible for maintaining atonia during REM sleep[5]. It is more prevalent in males over 60 years of age but can occur at any age. While onset is often in the middle age, RBD may be seen in much younger population with a female preponderance, in association with antidepressant use[68,69]. There is a high degree of association with neurodegenerative disorders associated with α-synuclein pathology such as Parkinson's disease, dementia of Lewy body type and multiple system atrophy[39].

PSG shows both tonic and phasic abnormalities of muscle tone on EMG leads during REM sleep (Fig 2)associated with elaborate motor activity with dream mentation[40]. Moreover, RBD episodes may be associated with apneas in REM sleep although REM behaviors without loss of atonia may be seen at the termination of an apnea in REM sleep epochs among patients with severe sleep apnea. History and PSG studies are usually characteristic aiding differentiation from other rhythmic movement disorders of NREM and REM sleep, nocturnal seizures, episodic nocturnal wandering, nocturnal psychogenic dissociative disorders, PTSD, and nocturnal delirium to name a few.

Treatment involves taking safety precautions with first line pharmacologic therapy being benzodiazepines, specifically clonazepam[41, 66]. In patients with cognitive dysfunction and safety issues and when co-existent with OSA, melatonin is used in place of clonazepam[41, 66]. Desipramine has been tried with variable amount of success[5]. Imipramine, carbamazepine, levodopa, pramipexole, donepezil, triazolam, quetiapine and clozapine have been tried with variable success rate but limited evidence exists for the use of same[65, 66].

Impaired Sleep Related Painful Erectionsis also known as erectile dysfunction, organic impotence, impaired nocturnal penile tumescence refers to inability to sustain a penile erection that is large or rigid enough to engage in a sexual intercourse in the presence of normal sleep architecture[1]. It is important to take into consideration the presence of normal sleep architecture including sufficient REM sleep which is associated with phenomenon of involuntary penile erections and also patient's age in making assessment about organic impotence. It is associated with multiple other vascular, endocrine, and neural diseases as well as underlying

mental illnesses and sleep disorders which need to be taken into consideration while formulating a treatment plan. Prolonged PSG studies with strain gauge devices are important to assess sleep architecture including REM sleep duration and penile tumescence features.

Sleep Related Painful Erectionsrepresent painful penile erections during sleep associated with vivid dreams, are usually idiopathic occurring in adults over 40 years of age but can be sometimes associated with underlying organic causes such as phimosis or Peyronie's disease[42]. PSG is performed with various strain gauge devices to show multiple REM awakenings associated with erections and over the long term can lead to anxiety, irritability, insomnia and daytime somnolence. It usually remits spontaneously and REM suppressant medications are seldom required. Surgical treatment of underlying organic cause is performed if present.

REM Sleep Related Sinus Arrestor nocturnal asystole refers to sinus arrest or a prolonged period of asystole during REM sleep in an otherwise healthy individual. PSG studies show prolonged period of asystole (>2.5 sec) in EKG leads occurring repeatedly during REM sleep in the absence of OSA and other sleep related disorders and may be associated with vague chest discomfort or even lightheadedness, syncope or loss of consciousness if awakened[1,43].

D. Other Parasomnias

Includes a diverse group of parasomnias that cannot be otherwise grouped elsewhere with sleep wake transition disorders, disorders of arousal and REM sleep disorders (Table 1). Only a few of these are discussed below.

Bruxism is a relatively common sleep disorder characterized by repeated stereotyped teeth grinding during sleep and is commonly associated with underlying dental or orofacial abnormality which can lead to secondary wear and tear tissue injury and periodontal and jaw pain. The etiology of sleep related bruxism is different from bruxism during wakefulness when it is more likely to be associated with movement disorders such as tics.

Although bruxism can appear at any age from infancy to adulthood, it is most commonly seen in children, adolescents and young adults. Approximately 90% of population has had bruxism at some point in their life with almost 5% presenting with bruxism as a clinically significant condition[1]. No gender differences have been reported. It is often associated with underlying stress, anxiety and emotional turmoil in life, various orofacial abnormalities such as malocclusion of teeth and temporomandibular joint disorders, and is more common in children with cerebral palsy and mental retardation[1, 5,44,45,46].

PSG recordings shows characteristic rhythmic, bursts of EMG motor artifacts occurring in bursts in NREM stage 1, 2 and REM sleep. Bruxism may be associated with obstructive apneas among adults.

Treatment involves stress reduction, sleep hygiene, psychotherapy and in chronic severe cases, use of mouth guard to prevent injury. Treatment of underlying anatomical pathology as discussed above is recommended if present.

Nocturnal Paroxysmal Dystonia (NPD) consists of stereotyped, repeated dystonic or dyskinetic movements characterized by dystonic posturing such as flexion or extension adductor limb posturing, truncal rotation, ballistic or choreoathetoid movements during sleep with open and unresponsive eyes and occasional vocalizations[1, 5, 47]. Nocturnal paroxysmal dystonia may present as brief 15-60 episodes with frequent recurrence during the night sleep, may be associated with episodic nocturnal wandering or exist as a prolonged form lasting up to an hour and occur almost every night[47].

The subject can awaken or remain asleep during the episode and in contrast to other parasomnias usually are not confused and have no difficulty going back to sleep. They might have excessive daytime sleepiness and fatigue secondary to lack of restful sleep, cause significant interference with the sleep of their bed partner and can even injure to themselves or their partner.

They can begin in childhood, adolescence, middle age or late middle ages with no gender difference. Rare familial forms have been reported[48]. There has been intensive investigation in the last decade with regards to underlying pathophysiology, associated disorders and PSG and EEG findings with its differentiation into the short and prolonged from and the ones associated with nocturnal episodic wandering. Although there has been no conclusive evidence of NPD being an ictal phenomenon (lack of epileptic discharges during the episodes), there has been many reports of co-occurrence of frontal lobe seizures and generalized tonic clonic seizures in patients with NPD[49, 50]. Moreover, as frontal lobe seizures often have a poor EEG correlate,there has been a growing consensus that some of the cases of NPD

(short duration) with episodic wandering may represent nocturnal frontal lobe epilepsy[47, 49].

Neurophysiologic studies shows occurrence in NREM sleep, usually in stage 2 but they can occur in stage 3 and 4 with preceded by EEG pattern of arousal indicated by desynchronization which is later on obscured by superimposed motoric artifact[5, 47]. In case of brief spells associated with coexisting epilepsy, no ictal EEG discharge is noted during the spell indicating it to be a non-epileptic event[47].

Thorough investigation with PSG and EEG studies should be performed for possibility of coexisting nocturnal frontal lobe epilepsy.

Treatment is usually warranted with carbamazepine being the drug of choice.

Enuresis Nocturna, nocturnal bedwetting or sleep enuresis involvesrecurrent, unconscious, involuntary micturition during sleep. It can be grouped into primary or secondary and symptomatic or idiopathic form. Primary form involves bedwetting before the child has acquired micturition control in the absence of any underlying disorder, whereas secondary form involves occurrence after the child has acquired control and has been continent for three to six months, usually after 6 years of age. Symptomatic form is associated with underlying organic genitourinary pathology and recurrent UTI. It can involve post event incorporation of the enuresis stimuli in dream sequences, particularly during the first third of the night.

Etiology as mentioned above can be because of underlying pathology, genitourinary malformations, recurrent UTI, irritable bladder and allergies. Obstructive sleep apnea, milk allergies with irritable bladder and many metabolic and endocrine disorders predispose to persistent enuresis[52]. Bedwetting has higher prevalence in institutionalized children of low socioeconomic group. Positively family history is frequently reported in males.

The events can occur during any stage of sleep. Diagnostic workup involves detailed history taking, workup for identifying underlying structural, endocrine disorders PSG is usually performed with sleep cystometry to reflect the bladder reactivity and instability.

Treatment for secondary form involves treating the underlying etiology. For primary form, various non-pharmacologic (bladder exercise, avoiding fluids at bedtime, bed alarms, minimizing stress and maximizing support) measures should be tried first. In resistant cases, pharmacotherapy with desmopressin and anticholinergic agents such as imipramine is considered to be first line agent[51]. TCA's have also been tried with variable amount of success. Most cases usually remit with aforementioned interventions. Among children with associated apnea, treatment of the underlying apnea may result in improvement/resolution of enuresis.

Sleep Related Eating Disorder (SRED) involves recurrent episodes of nightly eating or drinking occurring during sleep occurring in the absence of other distinct parasomnia, medical or neurological problems or secondary to medications or substance abuse[1]. SRED patients consume a major portion of their daily intake during these nightly episodes with complaints of morning anorexia, non-restorative sleep and daytime fatigue. It may involve sleep related injury, dangerous behaviors during pursuit of food, ingestion of toxic or inedible material and adverse health consequences of excessive eating. It is more common in patients with underlying eating disorder (9-17%) as compared to prevalence of 5% in general population, predominantly in young, overweight (40%) females (75%) [53,54,55,56]. PSG studies with actigraphy shows frequent awakening during NREM sleep associated with frequent chewing and swallowing and is frequently associated with somnambulism. Differnital diagnosis includes nocturnal eating syndrome (NES), kleine-levin syndrome, dissociative disorder, bulimia nervosa with nocturnal eating and binge eating disorder. Clinical history, demographic profile, occurrence during NREM sleep, PSG findings along with the features mentioned above aids in the diagnosis and distinction from NES. GABA-B agonists may predispose patients to sleep related eating disorder as well even in patients with no prior history of an eating disorder.

Treatment involves evaluation for and identification of other co-existing underlying disorder (OSA, RLS, PLMS and somnambulism) and its treatment and safety precautions to avoid injury from food as well as activity. Topiramate therapy has been demonstrated to be the most effective therapy for SRED[57].

Sleep Related Groaning or nocturnal groaning or Catathrenia (derived from two greek words kata - below and threnia - lament) is a parasomnia characterized prolonged inspiration followed by end inspiratory apnea and subsequent desaturation leading to partial arousal and prolonged expiration with expiratory groaning with

a morose quality or sexual connotation posing a significant problem for the sufferer as well as his/her bed partner[1]. Little is known about the exact epidemiology, pathophysiology and associated features and disorders of this newly classified disorder. Various literature sources mention that it occurs predominantly during the REM sleep but can also occur in NREM sleep with evidence of some degree of expiratory obstruction and abnormal respiratory disturbance index (RDI) during PSG study[58]. Various case reports and case series have shown excellent response to continuous positive airway pressure therapy[58].

E. Approach to a patient with parasomnia:

Historical details are important in diagnosing Parasomnias (Table 2). Age of onset of the events in question is first. Most NREM parasomnias have an early age of onset. Night terrors, somnambulism and

Table 2: Sleep Wake Transition Disorders & Other Parasomnias: Distinguishing Features & Various

		Age of Onset	Time of Occurrence	Sensory / Behavioral Symptoms	Event Recall	Significant Autonomic Symptoms	Motoric Phenomenon	DDx Considerations	Associations / Other Considerations
Sleep Wake Transition Disorders		**Variable**	**Sleep wake transition periods**	**Infrequent**					
	Sleep Starts	Variable	Pre or post-dormital	Falling sensation, vivid dreams, hypnagogic hallucinations	Partial	No	Yes	Myoclonus, hyperekplexia, epileptic myoclonus, nocturnal frontal lobe epilepsy	Excessive stimulants and stress
	Rhythmic Movement Disorder	**Infancy or early childhood**	**First 3rd of the night, NREM 1,2**	**No**	**No**	**No**	**Yes**	**Periodic movement of sleep, epileptic event**	**Underlying neurological or psychiatric disorders if it persists in adulthood**
	Sleep Talking	Variable	During sleep transition phases in all stages of sleep	No	No	No	No		Precipitated by emotional stress, febrile illness, OSA
	Nocturnal Leg Cramps	**Variable**	**Variable**	**Painful sensation of muscle tightness / cramping**	**Yes**	**No**	**No**		**Frequently associated with various physiological states, electrolyte abnormalities and disease processes**
Other Parasomnias		Variable	Variable						
	Sleep Bruxism	**Variable**	**NREM 1,2 and REM sleep**	**No**	**No**	**No**	**Stereotyped teeth grinding**	**Underlying orofacial and TMJ abnormalities**	**Stress, Poor sleep hygiene, Developmental disorders in children, OSA**
	Sleep Enuresis	Childhood	Any stage		Occasional incorporation in dream sequences	No	No	Secondary form associated with underlying genitourinary abnormality and recurrent UTI	OSA, genitourinary abnormalities
	Nocturnal Paroxysmal Dystonia (NPD)	**Variable**	**First 3rd of night, usually NREM 2**	**Occasional vocalizations, episodic wandering**	**No**	**No**	**Stereotyped, repeated, dystonic or dyskinetic movements**	**Should be differentiated from various epilepsies, particularly nocturnal frontal lobe epilepsy**	**Association with frontal lobe epilepsy**
	Sleep Related Eating Disorder (SRED)	Young adults	Usually first half of night during NREM sleep	Can be associated with injury to self	No	No	Nightly eating or drinking activity	NES, Klein-Levin syndrome, dissociative disorder, bulimia nervosa	Association with OSA, GABA-B agonists
	Sleep Related Groaning (Catathrenia)	**Unknown**	**Usually during REM sleep**	**Make morose or lamenting expiratory noise / groaning**	**No**	**No**	**No**	**Other parasomnias**	**Association with OSA**

confusional arousals are most often seen in children. These NREM parasomnias may persist into adulthood and infrequently begin after adolescence. When they begin in childhood, they often tend to decline as the child enters adolescence and adulthood. REM parasomnias are more often seen in adults. Onset after the age of 40 is common especially for REM sleep behavior disorder. RBD episodes tend to decline in frequency with advancing age. Clinical features described above can be helpful in the diagnosis. Parasomnias can be distinguished from frontal lobe seizures based on the stereotyped nature of the latter whereas the former are generally non-stereotyped. REM sleep behavior disorder and somnambulism may be associated with injuries, and so do frontal lobe seizures with hypermotor features whereas most other parasomnias are not associated with injuries. Sleep walking episodes may result in self injury whereas bed partners report injuries in cases of RBD. Amnesia to the events is common parasomnias that are associated with delta wave sleep and in patients with frontal lobe seizures. Timing of the events is also a useful distinguishing feature. NREM parasomnias tend occur in the first third of the night whereas REM parasomnias have a predilection to the later third of the night. Frontal lobe seizures tend to occur shortly after falling asleep. Parasomnias generally do not occur during daytime naps whereas frontal lobe seizures can occur during daytime naps as well. Lastly precipitating factors are important as many of these have precipitating factors. Duration of the event and any confusion during the event or post event can be helpful in distinguishing them. Frontal lobe seizures are often brief (less than a min), whereas most NREM parasomnia episodes tend to be longer. REM parasomnias can be of variable duration. Nocturnal seizures can have postictal confusion whereas in frontal lobe epilepsy postictal confusion is extremely brief.

There are a number of known precipitants for parasomnias and information regarding this can be helpful for a physician in the clinic. Alcohol is a common precipitant for RBD episodes, whereas sleep deprivation is a common trigger for NREM parasomnias. Intercurrent febrile illness, menstrual periods or psychosocial stressors can precipitate both REM and NREM parasomnias. Other precipitants include medications. GABA agonists can precipitate somnambulism or other NREM parasomnias whereas abrupt withdrawal of antidepressant medications can precipitate REM parasomnias such as RBD acutely. RBD may be precipitated by antidepressant use in younger population with female preponderance as mentioned previously. Moreover, coexistent sleep disorders such as sleep apnea can precipitate parasomnia episodes wherein patients can have either a NREM or a REM parasomnia episode at the termination of an apnea. Some parasomnias are associated with other neurological conditions and patients should be evaluated for those as well. For example the chronic form of RBD is associated with Parkinson's disease or other synucleopathies that manifest with extrapyramidal symptoms. Acute form of RBD may be caused by a number of other acute neurological insults such as those due to pontine strokes, multiple sclerosis and subarachnoid hemorrhage to name a few.

While a polysomnogram (PSG) is not always necessary in diagnosing parasomnias, there are specific situations where it should be considered. First and foremost is when patients are suspected to have coexistent sleep disordered breathing or periodic limb movements of sleep. Secondly PSG should be considered when history is atypical or when parasomnias of both NREM and REM sleep are suspected in the same patient. EEG or prolonged video EEG monitoring should be considered in cases where frontal lobe epilepsy is suspected.

Pathophysiology: Pathophysiology of parasomnias is not well understood. They are believed to occur due to incomplete arousal from different sleep states, abnormal intrusions of wakefulness into different stages of sleep as well as deafferentation of generators of locomotion from generators of sleep. While generators of locomotion are known to be active during wakefulness, those at the spinal or supraspinal level are inhibited during sleep states to varying degrees and a central pattern generator hypothesis leads to the notions that there is some dissociation between the two that results in abnormal locomotion[59, 60].

There is a better understanding into the pathophysiology of RBD. The disorder occurs as a result of the loss of the normal atonia seen during REM sleep as well as abnormal augmentation of locomotor influences during REM. The cholinergic neurons in pedunculopontine nucleus and lateral dorsal tegmental area normally exert an excitatory input to the nucleus reticularis magnocellularis and gigantocellularis in the medulla which in turn exhibit inhibitory influences over the spinal motor neurons. Degeneration of these pontine centers that control REM sleep has been implicated in the loss of the atonia[61]. Studies using SPECT in patients

with RBD also showed abnormalities in striatal dopamine transporters as well as reduced striatal dopaminergic innervation although precise way by which these findings contribute to abnormal locomotion during REM sleep is not well understood[62, 63].

General Principles of Management:

In broadly stated terms, management of parasomnias involves following components:

- Accurate diagnosis
- Identification of and removal of predisposing factors
- Addressing associated conditions and co-morbid sleep disorders
- Environmental safety precautions
- Reassurance and counseling of the people affected (patients, parents, spouses and bed partners) including psychosocial counseling
- Pharmacologic therapy

Refer to Table 2 and the detailed description in prior sections for details.

Sleep wake transition disorders occurs because of relative instability or imbalance between hypnagogic and wakefulness structures. Consequently, it is frequently associated with factors that disrupt this balance such as excessive stimulants, exercising before sleep, emotional stress, febrile illness, anxiety or obstructive sleep apnea. As it runs a benign course, sleep hygiene, avoidance of the precipitating factors and reassurance and counseling of the patients, parents and bed partners usually suffices. Sleep starts and rhythmic movement disorders can be associated with injury requiring environmental safety measures such as removing bedside objects or furniture and protective measures such as helmet or padding. Rhythmic movement disorder persisting or appearing late in adulthood warrants investigations into associated underlying organic brain disorder, physical or psychological issues. Also, as stated above nocturnal leg cramps are frequently associated with various physiological states, electrolyte abnormalities and disease processes as mentioned earlier which should be addressed. An inkling about an associated condition should be evident on detailed history taking. Pharmacologic therapy is rarely necessary for sleep wake transition disorders. In particularly severe and resistant cases, benzodiazepines such as clonazepam and TCA's such as imipramine can be tried.

Disorders of arousal or NREM parasomnias occur because of partial or impaired arousal during NREM transition stages. Factors that predispose to frequent cortical arousals such as circadian rhythm sleep disorders are sleep deprivation, stress, pain, fever, CNS depressants, sedatives hypnotics and tranquilizers. Various hypersomnia's, sleep apnea, and occasionally stimulus such as distended bladder are implicated of precipitating factors and remediation of associated conditions such as sleep apnea treatment is sufficient along with reassurance and counseling. Environmental safety precautions is of paramount concern in confusional arousal and sleep walking which carry a significant potential for injury to self or others. All bedside furniture and objects of potential harm should be removed, bedroom doors and windows should be locked, firearms should be removed or locked and door and window alarms can be used to alert others. In resistant cases benzodiazepines such as clonazepam and diazepam and TCA such as imipramine can be tried. TCA can exacerbate and should be used with caution in sleep walking. Paroxetine has been shown to be particularly effective in sleep terrors in various case reports but should be used with caution in sleep walking[65]. Non-pharmacologic therapy with cognitive behavioral therapy, hypnosis and psychotherapy has been tried with variable amount of success. In childhood NREM parasomnias anticipatory awakening just before the onset of a typical episode is frequently used.

REM parasomnias can be associated with supposedly benign precipitating factors or factors readily amenable to treatment such as secondary to medications (nightmares), circadian rhythm disturbances (sleep paralysis), underlying psychopathology (nightmares, sleep paralysis) or sleep apnea or it may be associated with an underling neurodegenerative disorder (RBD) and comorbid sleep disorders (narcolepsy-cataplexy triad in recurrent isolated sleep paralysis). Various associations are described in detail in preceding sections.

Management is along the same underlying principles as described above including safety precautions, psychosocial and non-pharmacologic interventions. Initial treatment of nightmares involves sleep hygiene, stress reduction, CBT, psychotherapy and in resistant cases REM suppressants such as TCA and MAO inhibitors. MAO inhibitors should be used with caution in RBD as it may induce or aggravate it. Clonazepam

and melatonin are the first line agents for RBD with level B evidence for use of pramipexole and little evidence for use of paroxetine or L-DOPA[66]. In resistant sleep paralysis cases, REM suppressants such as imipramine, antidepressants and MAO inhibitor phenelzine can be used.

Other parasomnias are a diverse group with no unifying underlying pathophysiologic mechanism and sleep stage association. Bruxism is usually associated with underlying stress and anxiety with treatment focused along the same lines with use of mouth guard in severe cases. Treatment of underlying orofacial abnormality is warranted if present. Nocturnal paroxysmal dystonia is sometimes difficult to separate from associated epilepsy, particularly nocturnal frontal lobe epilepsy, warranting detailed PSG and EEG studies. Carbamazepine is the most effective treatment. Enuresis treatment again revolves around behavioral and non-pharmacologic approach with treatment of underlying pathology in secondary cases. First line pharmacologic agent in resistant cases is desmopressin with imipramine falling out of favor. SRED is frequently associated with bulimia, OSA, RLS, PLMS and somnambulism which should be addressed concurrently along with safety precautions. Topiramate is the most effective drug therapy for SRED. Catathrenia is frequently associated with sleep disordered breathing and abnormal RDI with excellent response to CPAP in various case reports.

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