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CASE REPORTS

Three cases of parasomnias similar to sleep terrors occurring during sleep-wake transitions from REM sleep

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Sleep terrors are a non-rapid eye movement (NREM) sleep–related parasomnia characterized by abrupt terror with a frightening scream. However, it remains unknown whether sleep terrors occur only from NREM sleep. We evaluated 3 cases of a sleep terrors–like parasomnia that occurred exclusively during arousals from rapid eye movement (REM) sleep. These parasomnia events occurred at REM sleep-wake transitions, manifesting with screaming or crying, similar to sleep terrors, without alertness or clear orientation. The patients were all young-adult females without notable medical conditions. REM sleep behavior disorder and nocturnal frontal lobe epilepsy were not detected based on their video-polysomnographic findings. These 3 cases should be provisionally diagnosed as "Parasomnia, Unspecified" according to the *International Classification of Sleep Disorders,* third edition; however, the phenomenological diagnosis is proposed to be "Disorders of Arousal from REM Sleep." Our reported cases indicate that sleep terrors may also arise from REM sleep.

Keywords: sleep terrors, nightmare disorder, RBD, clonazepam, V-PSG, parasomnia

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INTRODUCTION

Human consciousness consists of 3 essential states of being: awake, non-rapid eye movement (NREM) sleep, and rapid eye movement (REM) sleep. These 3 states of being are not necessarily exclusive and appear in various combinations with fluid boundaries.[1] Parasomnias are the result of such sleep state dissociation.[2] Disorders of arousal manifest as parasomnias that occur during the sleep-wake transition from NREM sleep, especially from slow-wave sleep. This disorder is classified into 3 categories: confusional arousals, sleepwalking, and sleep terrors, according to the *International Classification of Sleep Disorders,* third edition (ICSD-3).[2] These 3 behavioral patterns have a common neurophysiological basis with regard to the dysfunctional transition from NREM sleep to wakefulness and may be found in the same individual as a mixed state or may shift from one to another. Sleep terrors are characterized by emotional behavior in the form of abrupt terror accompanied by symptoms of frightening screams with obvious autonomic involvement, including mydriasis, tachycardia, tachypnea, and diaphoresis.

However, it remains unknown whether disorders of arousal occur only from NREM sleep. There has been 1 case report in which confusional arousals occurred from REM sleep.[3] In this case, confusional arousals arising from REM sleep were documented during video-polysomnography (V-PSG) in a 5-year-old girl who was referred for suspected sleep apnea on account of a 2-week history of snoring triggered by tonsillitis. The girl had a history of rare nightmares, but no history of sleepwalking or confusional arousals. The V-PSG showed mild sleep apnea with an apnea-hypopnea index of 2.7 events/h. However, the V-PSG also showed a typical confusional arousal arising from the third REM cycle, which was not triggered by an apnea or nightmare. REM atonia was well preserved and REM sleep behavior disorder (RBD) was excluded. This case suggested a possibility that arousal disorders can also occur from REM sleep. There have also been reported cases of sleep-related eating disorder[4] and sexsomnia[5] emerging from REM sleep, both of which are recognized as NREM parasomnias in the ICSD-3.[2] Recently, we evaluated 3 cases of parasomnias that showed sleep terror symptoms, including screaming or crying without clear orientation and alertness, during arousals from REM sleep. We herein present these 3 cases and discuss how sleep terrors can occur during arousals from REM sleep.

REPORT OF CASES

Case 1

This is a case of a 34-year-old Japanese female office worker who presented to our sleep clinic with a complaint of nightly screaming during sleep that had begun a few months earlier.

She had no history of parasomnia or any other sleep disorders until the age of 12 years. She stated that sleep paralysis–like symptoms began to occur frequently at the age of 13 years. Cataplexy-like antigravity muscle weakness also began to occur almost simultaneously. She stated that this muscle weakness symptom was induced mainly by laughing and

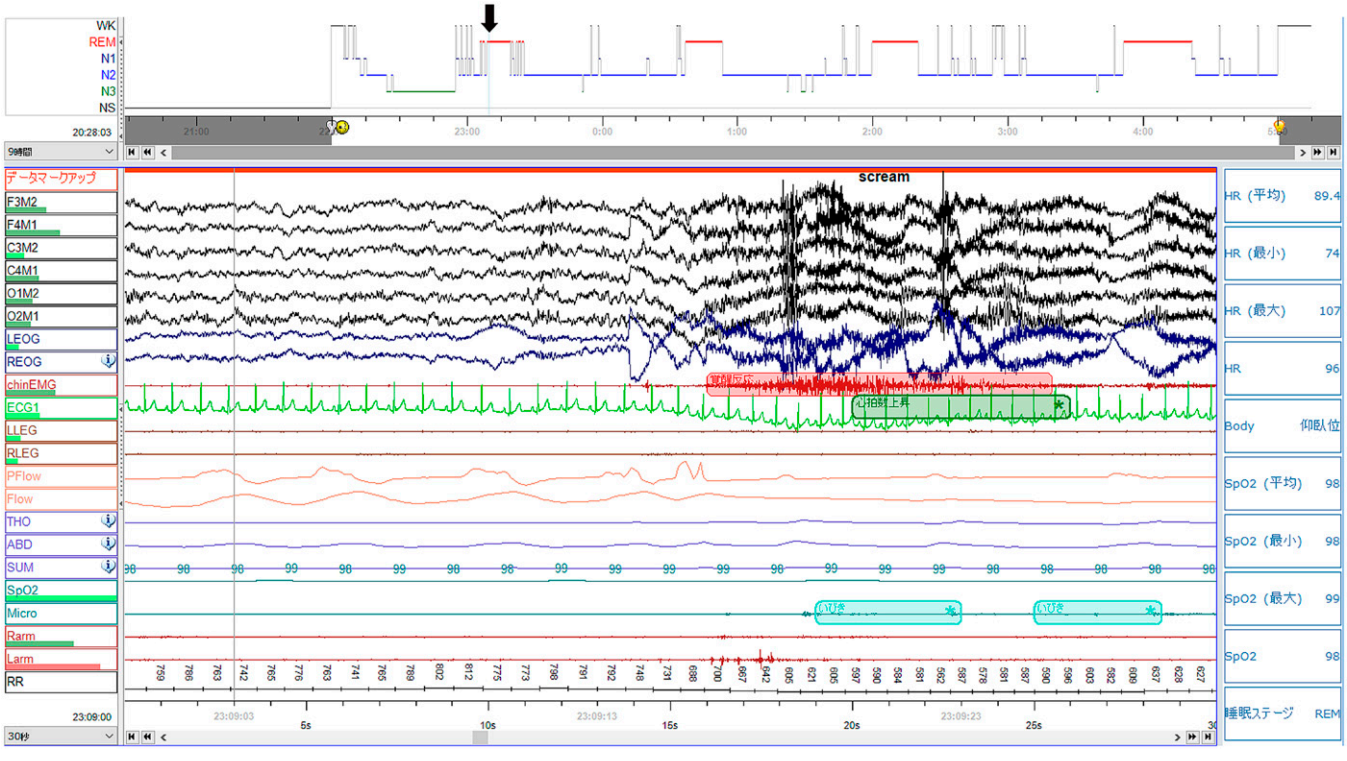
lasted a few seconds. However, she did not have other narcoleptic symptoms, such as excessive daytime sleepiness or hypnagogic hallucinations at that time. These symptoms gradually decreased with age, and the symptoms of weakness disappeared completely within several years, but sleep paralysis persisted after reaching adulthood.

A few months before presenting to our clinic, she began to scream while sleeping at night. The events occurred almost every night without any significant inducement. Her family members stated that the patient suddenly screamed fearfully without words during her sleep, and that the episodes lasted a few minutes, and shortly thereafter the patient fell asleep. The patient also stated that the frequency of sleep paralysis increased a few months before presentation to our clinic along with an increased frequency of screaming. When her family members awakened the patient during the episodes, she sometimes recalled nightmares and occasionally had symptoms of sleep paralysis. These episodes often occurred in the first third of the night, without accompanying body movements.

Her body mass index was 19.5 kg/m^2 at the time of presentation. She had no medical or psychiatric disorders and had not taken any medications. Despite her history of REM sleep–related symptoms, such as sleep paralysis and cataplexy-like symptoms, narcolepsy was excluded because excessive daytime sleepiness was completely absent by patient and family report. There was no family history of sleep disorders including parasomnia apart from her brother, who had been diagnosed with idiopathic hypersomnia. She usually slept from 1 AM to 6 AM on weekdays and from 2 AM to 8 AM on weekends. She also did not smoke or drink, and physical and neurologic examinations did not reveal any abnormal findings on her first visit to our clinic.

V-PSG revealed that she suddenly woke up screaming fearfully for the first 30 seconds intermittently and then crying sadly for the last 30 seconds without any words in the first cycle of REM sleep. Electroencephalography (EEG) revealed an alpha-dominant pattern without body movement, while electrocardiography showed tachycardia with a heart rate of 100 beats per minute (**Figure 1**). Shortly thereafter, her sleep returned to typical REM sleep with the occurrence of rapid eye movements and atonia. There were neither epileptiform EEG activities nor any episodic arousals from NREM sleep. Sleep paralysis also did not occur throughout the night. The patient could not recall the parasomnia episode and had no recollection of dreaming throughout the night in the interview the next morning. Sleep architecture did not show any remarkable findings: stage N1 sleep, 5.1%; stage N2 sleep, 65.4%; stage N3 sleep, 8.6%; and stage R sleep, 20.8%. Total sleep time was 397.5 minutes and sleep latency (SL) was 5.0 minutes. Sleep efficiency was 94.5% and the arousal index was 11.0 events/h. The apnea-hypopnea index was 0.5 events/h, the periodic limb movement (PLM) index was 0 events/h, and a sleep-onset REM period (SOREMP) was not observed. REM without atonia (RWA)/REM (%) was 1.21% throughout the night but was observed only in the fourth REM

Figure 1—Nocturnal video-polysomnography of case 1.



In the first cycle of rapid eye movement sleep, she suddenly woke up screaming and crying intermittently for 60 seconds at 23:09. The hypnogram (top row) shows the point at which the parasomnia occurred with black arrow.

sleep cycle without accompanying parasomnia events. The initial therapeutic intervention for this patient consisted of optimizing sleep hygiene and increasing total sleep time; shortly afterward, she reported the complete disappearance of her parasomnia.

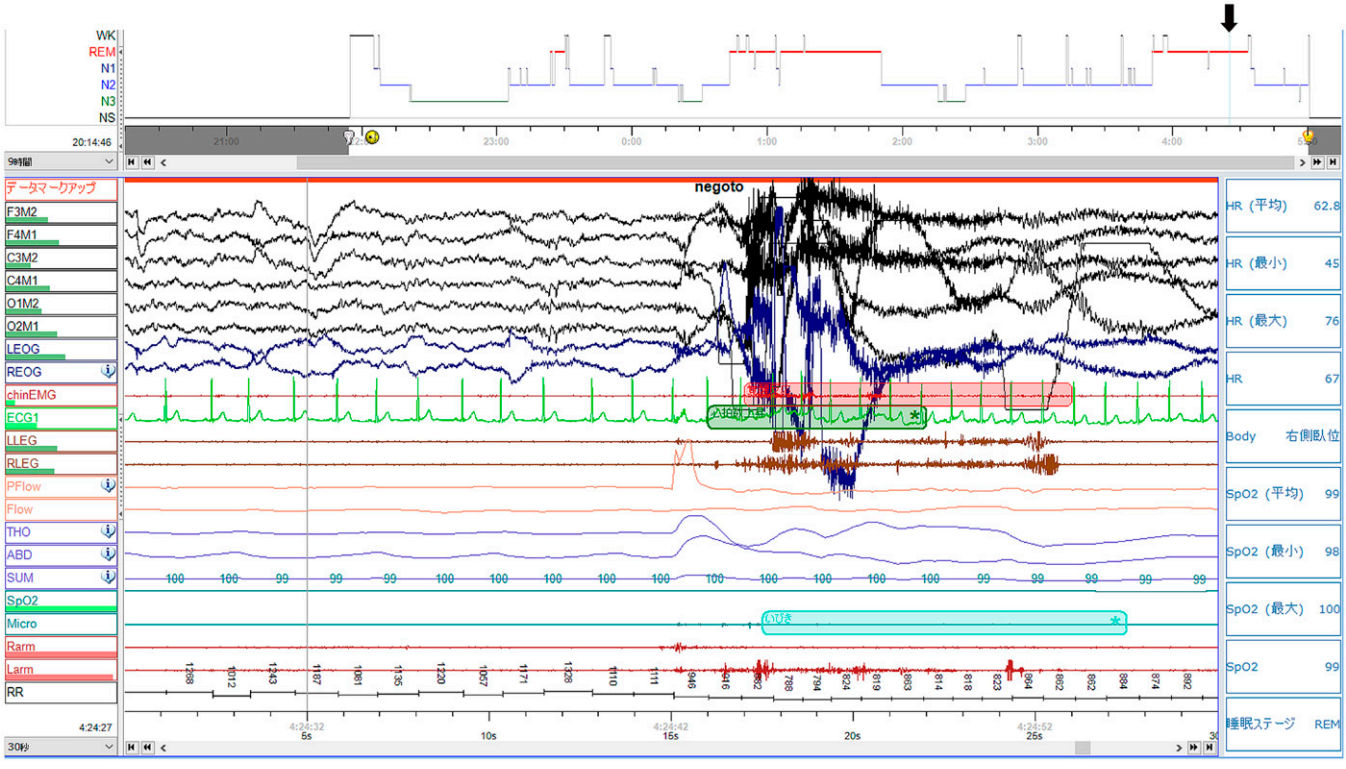
Case 2

Case 2 was a Japanese 26-year-old female office worker who presented at our sleep clinic with a complaint of nightly screaming for 5 years after she had started working. She had no history of sleep disorders. Her family noticed that the patient suddenly woke up while screaming or crying during nocturnal sleep. Episodes lasted for only a few seconds, and the patient fell asleep soon after these events. She felt that the stress of long working hours may have reflected upon the occurrence of parasomnia events. She hardly remembered these episodes; however, she sometimes woke up with her own loud voice during the episodes and could recall nightmares at these times. These events occurred approximately twice or three times per week, especially during a stressful week with a heavy workload. Sometimes she brushed her arms away during the parasomnia events and once hit her arm on the wall during an episode in which she remembered the content of her dreams. The symptoms persisted without any changes until she presented herself to our clinic.

The patient had atopic dermatitis and took an antihistamine drug (bepotastine besilate 20 mg daily) regularly at the time of presentation. However, she had no other medical or psychiatric history or any significant family history of sleep disorders, including parasomnia. She slept from 2 AM to 9 AM on weekdays and from 1 AM to 10 AM on weekends. She did not have a habit of smoking or drinking alcohol, and her body mass index was 17.1 kg/m^2. On her first visit, physical and neurologic examinations revealed no abnormal findings, and psychiatric symptoms were not detected.

This patient underwent V-PSG while taking the medication bepotastine besilate. A parasomnia episode occurred during an arousal from the third cycle of REM sleep when she suddenly woke up and brushed her arms away while she looked like she was murmuring something for about 10 seconds; however, we could not understand what she said from the V-PSG recording. At that time, EEG revealed an alpha rhythm and electrocardiography revealed a stable heart rate of approximately 60 beats per minute (**Figure 2**); however, the sleep stage soon returned to normal REM sleep. There was no epileptiform EEG activity or episodic arousal from NREM sleep. The patient could not recall the parasomnia event at the episode; however, she recalled a nightmare of being strangled by someone during the night at the interview the next morning. Sleep architecture did not show any remarkable findings: stage N1 sleep, 3.9%; stage N2 sleep, 52.9%; stage N3 sleep, 16.2%; and stage R sleep, 27.0%. Total sleep time was 401.5 minutes and sleep latency was 10.5 minutes. Sleep efficiency was 94.2% and the arousal index was 9.0 events/h. The apnea-hypopnea index was 1.0 events/h and

Figure 2—Nocturnal video-polysomnography of case 2.



In the third cycle of rapid eye movement sleep, she suddenly woke up and brushed her arms away while murmuring something for about 10 seconds at 4:24 AM. The hypnogram (top row) shows the point at which parasomnia occurred with a black arrow.

the PLM index was 0 events/h. REM atonia was well preserved with RWA/REM (%) showing 0%.

We chose clonazepam (0.5 mg) at bedtime to treat her parasomnia symptoms. Her husband reported that the symptoms disappeared immediately after starting the treatment. However, the patient complained of feeling a hangover the next morning, and the dose of clonazepam was reduced to 0.25 mg. Thereafter, mild sleep-talking episodes without screaming appeared at times, but the symptoms of the next morning hangover disappeared. Nightly administration of clonazepam (0.25 mg) was continued due to its ongoing benefit.

Case 3

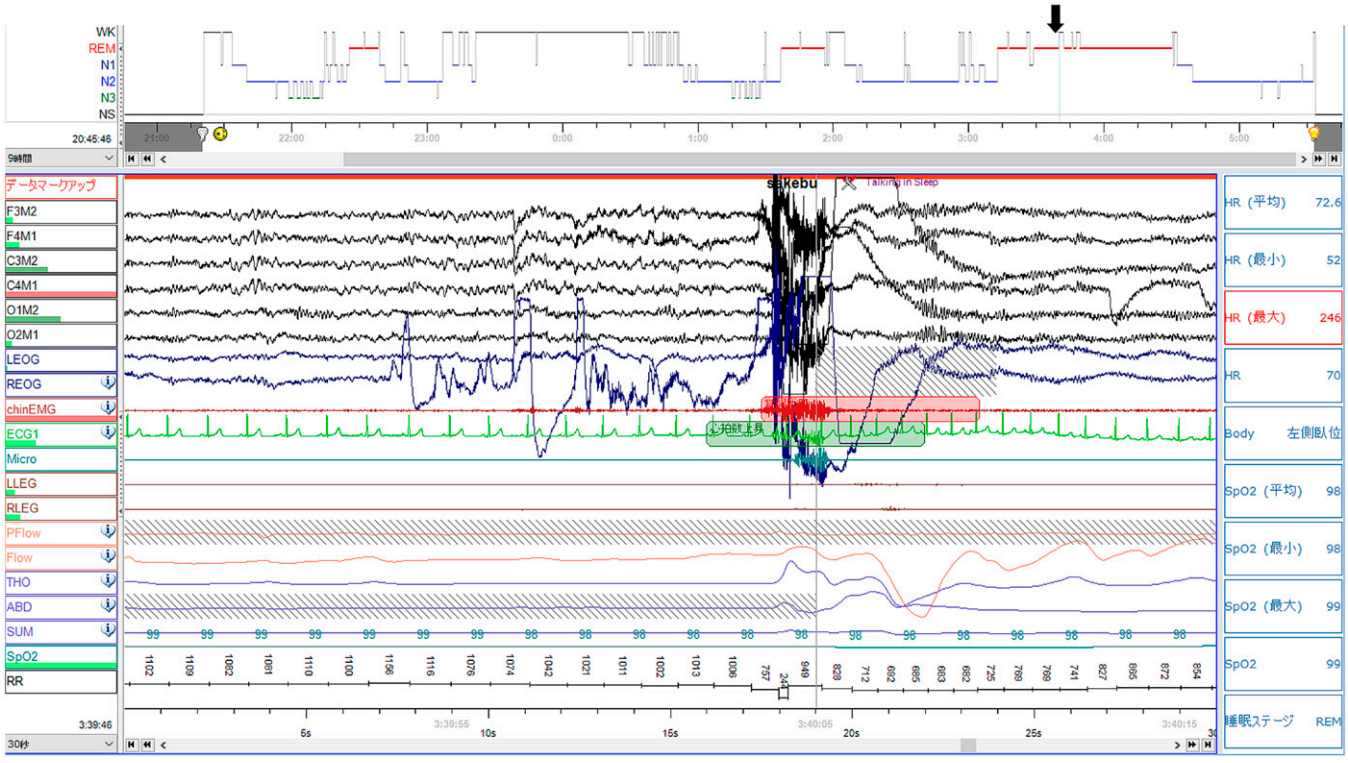
This is a case of a 26-year-old Japanese woman who was revealed to scream angrily during the transition from REM sleep with atonia to arousal on V-PSG. She visited our sleep clinic with a complaint of nightly screaming during sleep that had lasted for 13 years.

Her first parasomnia history began at the age of 13 years, without a history of any other sleep disorders. One of her family members stated that she suddenly screamed angrily or laughed loudly during night sleep. The episodes lasted for only a few seconds, and the patient fell asleep again soon after the events. The patient could not recall the episodes and denied the coexistence of dreams. The events occurred around dawn or during the last third of the night, twice or 3 times a week, without any kind of inducement. Sometimes, the patient quickly moved her arms and legs during parasomnia events. Symptoms persisted continuously and showed no change until the time she presented herself to our sleep clinic.

Regarding her medical history, she had gone to a smoking-cessation clinic to receive a prescription of varenicline tartrate at a dose of 2.0 mg per day orally for a few months before visiting our clinic. However, her family members felt that the administration of varenicline tartrate and quitting smoking did not affect her parasomnia episodes. The patient did not have any other medical or psychiatric history. There was also no family history of sleep disorders, including parasomnia. She did not habitually ingest alcohol and had continued to quit smoking for a few months before she presented at our clinic. She was unemployed but slept on a regular sleep schedule from 1 AM to 9 AM at that time. Her body mass index was 19.5 kg/m^2, and physical and neurologic examinations revealed no abnormal findings. The patient did not exhibit any psychiatric symptoms.

This patient underwent V-PSG while taking the medication varenicline tartrate. An episode of screaming during an arousal from REM sleep was observed; she suddenly woke up while screaming angrily for a second just before 4:00 AM in the third cycle of REM sleep (**Figure 3**). After this episode, she remained lying silently, with the EEG revealing a predominant 10-Hz alpha rhythm for 2 minutes and the electrocardiography revealing a stable heart rate at about 70 beats per minute.

Figure 3—Nocturnal video- polysomnography of case 3.



In the third cycle of rapid eye movement sleep, the patient suddenly awoke while screaming angrily for a second at 3:40 AM. The hypnogram (top row) shows the point at which parasomnia occurred with a black arrow. An artifact is observed on the right electrooculogram.

The alpha rhythm on the EEG gradually attenuated and was replaced by a low-voltage mixed-frequency (4–7 Hz) EEG within 1 minute, returning thereafter to normal REM sleep. There was no epileptiform EEG activity or episodic arousal from NREM sleep. Although frequent neck myoclonus[6,7] was observed during REM sleep throughout the night (the index was 42.0 events/h in REM sleep) in this patient, no myoclonic event occurred at least 3 minutes before the parasomnia event. The patient had no memory of the parasomnia episode and could not recall any dream experience during the night at the interview the next morning. Sleep architecture and other findings were generally unremarkable, apart from increased N1 sleep, decreased N3 sleep, and increased arousal index: stage N1 sleep, 16.3%; stage N2 sleep, 51.8%; stage N3 sleep, 4.3%; and stage R sleep, 27.5%. Total sleep time was 368.5 minutes and sleep latency was 8.0 minutes. Sleep efficiency was 74.7% and the arousal index was 17.3 events/h. The apnea-hypopnea index was 1.1 events/h and the PLM index was 0 events/h. REM atonia was well preserved, and RWA was not observed.

Clonazepam was chosen as the treatment drug for her unspecified parasomnia. After a dose of 0.5 mg clonazepam at bedtime, the patient noted immediate effects on the reduction in the frequency of parasomnia episodes, from about 3 times to 1 time or less per week. The same nightly medication of the drug was continued due to its ongoing benefits.

DISCUSSION

To the best of our knowledge, this is the first case series showing sleep terror–like symptoms that appear during arousals from REM sleep. These cases were found to have neither RWA (or very few, if any, episodes of RWA, without relation to the parasomnia event) nor epileptiform EEG activity on V-PSGs. These 3 cases were provisionally diagnosed as "Parasomnia, Unspecified" according to the ICSD-3;[2] however, the phenomenological diagnosis should be "Disorders of Arousal from REM Sleep." The clinical characteristics of the patients are summarized in **Table 1**.

The symptomatic features of these cases were quite similar to those of sleep terrors because their parasomnia episodes showed screaming or crying without any recall of the episode. However, the most notable conflict for the diagnosis of sleep terror is that these episodes arose not from NREM sleep but from REM sleep. Furthermore, the parasomnia events of cases 2 and 3 did not show autonomic symptoms such as tachycardia, which is a cardinal feature of sleep terrors. Typically, during REM sleep, the heart rate fluctuates more prominently compared with NREM sleep since normal REM sleep is characterized by relative activation of sympathetic function.[8] We do not know why cases 2 and 3 did not show autonomic symptoms; however, as we state below, REM-related parasomnias (such as RBD and nightmare disorder) show relatively minor autonomic changes, in contrast to the emotional dream content.[2] In general, they had characteristics in common with sleep terrors in that clonazepam[9] showed a clear treatment response in cases 2 and 3, and sleep deprivation (case 1) or job stress (case 2) was thought to contribute to the occurrence of the episodes.[2,9] Thus, it is possible that these cases form part of a disease continuum with sleep terrors.

The parasomnia episode of case 1 was notable in that tachycardia was observed, which was not seen in cases 2 and 3. Furthermore, the parasomnia events of this case arose from the first REM sleep period while those of cases 2 and 3 arose from the third cycle of REM sleep, similar to a study on a case of confusional arousals from REM sleep.[3] Case 1 might have had a vulnerability that caused sleep state dissociation[1] considering her family history of idiopathic hypersomnia. Case 1 also had other symptoms of sleep state dissociation, including sleep paralysis and cataplexy, both of which are considered as sleep state dissociation involving REM sleep and wakefulness.[1] Case 1 was also chronically sleep deprived, which might have been a

Table 1—Clinical characteristics of the 3 cases.

	Case 1	Case 2	Case 3
Chief complaint	Screaming during sleep	Screaming during sleep	Screaming during sleep
Age (y)/sex	34/Female	26/Female	26/Female
Age at onset (y)	34	21	13
V-PSG finding during the episodes	Arousal from REM sleep with crying and screaming	Arousal from REM sleep with murmur	Arousal from REM sleep with screaming
RWA/REM sleep (%)	1.2%	0.0%	0.0%
Psychological stress	–	+	–
Body movement at the episodes	–	+	±
Autonomic symptom	+ (tachycardia)	–	–
Medical history and medication	Mild asthma with no medication	Antihistamine drug for atopic dermatitis	Varenicline tartrate for smoking cessation
Family history	Idiopathic hypersomnia	–	–
Effective treatment	Extension of nocturnal sleep time	Clonazepam 0.25 mg at bedtime	Clonazepam 0.5 mg at bedtime

REM = rapid eye movement, RWA = rapid eye movement sleep without atonia, V-PSG = video-polysomnography, + symbols means present, – symbols means absent, ± symbols means inconsistent (sometimes present and sometimes absent).

predisposing factor for her sleep paralysis.[10] Moreover, we should consider the possibility that sleep deprivation induced several forms of sleep state dissociation, including the proposed "Disorders of Arousal from REM Sleep," because the extension of nocturnal sleep duration clearly improved the parasomnia symptom in this case. A case report has shown that clonazepam improved prolonged sleep paralysis and sleepwalking,[11] which suggests the possibility that clonazepam improves sleep state dissociation involving both REM sleep and NREM sleep. Clonazepam is used as an effective treatment for both RBD and "Disorders of Arousal from NREM Sleep."[12] Clonazepam also showed a clear treatment response in the present cases of "Disorders of Arousal from REM Sleep" in cases 2 and 3.

Among the candidate diseases for differential diagnoses of these cases, nightmare disorder is characterized by recurrent, highly dysphoric dreams, which are disturbing mental experiences that generally occur during REM sleep and that often result in awakening.[2] However, patients with nightmare disorder do not usually scream, in contrast to RBD. Typically, on awakening from dysphoric dreams, individuals with nightmare disorder rapidly become oriented and alert, which is in conflict with the present cases showing episodes of abnormal vocalization without alertness or clear orientation. Another difference is that patients with nightmare disorder often complain of difficulty in returning to sleep after awakening from a vivid dream, while these cases returned to sleep quickly after the parasomnia event. Moreover, in nightmare disorder, highly disturbing dream content frequently contrasts strikingly with relatively minor autonomic changes.[2] Nightmares could be induced by medication, including varenicline[13] and an antihistamine drug,[2] both of which were used in the present cases. However, varenicline did not change the symptoms or the frequency of the parasomnia event in case 3. Case 2 also stated that she felt the parasomnia events were induced by her heavy work-related stress, and not by the antihistamine drug medication. Reportedly, more than two-thirds of individuals affected with sleep terrors and sleepwalking have NREM dreams or unpleasant dreamlike mentation during their parasomnia episodes.[14] Cases 1 and 2 also sometimes partially recalled their nightmare. Accordingly, it is possible that a certain type of nightmare affected their behaviors during the parasomnia episodes in these cases.

RBD, a disorder showing dream enactment behavior and vocalization during REM sleep, should also be considered as a differential diagnosis for these cases. In particular, patients with RBD showed an attenuated heart rate response during REM sleep similar to our cases due to reduced sympathetic activity.[15] The present cases did not fulfill the criteria for RBD because of a lack of significant RWA, a physiological background of dream-enactment behaviors. Furthermore, parasomnia episodes in these cases occurred during the transition to awake from REM sleep, but not during REM sleep. In addition, RBD typically manifests in a person's 50s or thereafter, and the disorder has a clear male predominance; however, the present cases were all young women with symptom onset in their 10s to 30s. Reportedly, REM-related parasomnias mimicking RBD could be triggered by REM sleep–disruptive events such as respiratory events or PLM activities.[16] In these cases, neither sleep apnea nor PLM activities were observed. Case 3 experienced frequent neck myoclonus[6,7] during REM sleep; however, the contribution of myoclonus to her parasomnia symptoms was ruled out due to an absence of myoclonic movements just prior to the parasomnia event.

Sleep-related dissociative disorder (SRDD)[17,18] should also be considered in differential diagnosis. SRDD is one of the variants of psychogenic dissociative disorders. It is often problematic to differentiate SRDD from arousal disorders arising from NREM sleep. However, SRDD could be differentiated by the V-PSG findings, since disorders of arousal occur just after arousal from sleep but SRDD occurs from a state of EEG wakefulness.[18] SRDD could also be differentiated from arousal disorders by the response to clonazepam therapy and sleep deprivation.[18] The symptoms of arousal disorders improve by clonazepam therapy and worsen by sleep deprivation while those of SRDD do not show much change by the response to clonazepam therapy and sleep deprivation.

Sleep-related epilepsy that frequently accompanies vocalization and motor events should be considered in differential diagnosis of our cases. In this category, nocturnal frontal lobe epilepsy[19] has been reported to occur during or on arousal from sleep. Patients with nocturnal frontal lobe epilepsy are likely to show motor events or parasomnia-like behavior events, including vocalization with frightened expression and fear. Most of the seizures appear mainly in the NREM sleep stages and rarely emerge from REM sleep.[2] In contrast, in the present cases, parasomnia events occurred not from NREM sleep but from REM sleep without showing epileptiform EEG activity on V-PSGs. However, epilepsy could not be completely excluded in the present cases, since we did not perform long-term video-EEG monitoring with multiple electrodes. Additionally, the EEG findings during the attacks are uninformative in almost half of the cases with nocturnal frontal lobe epilepsy because the discharges are located deep in the frontal lobe.[19] Although epilepsy and parasomnia are different diseases, they have common semiologic features in that complex behaviors arise in the absence of conscious wakefulness without awareness. Such similar features are explained by the activation of the same neural network of "central pattern generators."[20] Central pattern generators are located at the subcortical level mainly in the brain stem and spinal cord, and they produce stereotypical rhythmic motor sequences based on primitive and instinctual behaviors such as eating/alimentary, attractive/aversive, locomotor, and nesting habits. The concept of central pattern generators explains the behavioral patterns of epileptic seizures and parasomnias in 3 forms: (1) oro-alimentary automatisms, bruxism, and biting; (2) ambulatory behaviors, ranging from pedaling activity or PLMs to somnambulism; (3) various sleep-related events such as ictal fear, sleep terrors, nightmares, and violent behaviors, which were observed in the present cases.

In summary, the present cases suggest that sleep terrors may occur from REM sleep, which supports the hypothesis that arousal disorders may arise from REM sleep in line with the reported cases of confusional arousals,[3] sleep-related eating disorder,[4] and sexsomnia.[5] These disorders of arousal from REM sleep can be either spontaneous, or can be triggered by another sleep abnormality, and can emerge exclusively from REM sleep or from both REM sleep and NREM sleep (the case of sexsomnia[5] from REM sleep showed masturbation triggered by sleep bruxism during both REM sleep and NREM sleep).

Based on the concept of central pattern generators, these reported arousal disorders from REM sleep can be classified into a confusional behavior pattern (confusional arousals), an appetitive and sexual behavior pattern (sexsomnia), and a shouting with fear pattern (sleep terrors in the present cases). However, ambulatory behavior patterns arising from REM sleep have not yet been reported. Sleepwalking, as another disorder of arousal from REM sleep, awaits future documentation.

ABBREVIATIONS

EEG, electroencephalogram
NREM, non-rapid eye movement
PLM, periodic limb movement
RBD, REM sleep behavior disorder
REM, rapid eye movement
RWA, rapid eye movement without atonia
SRDD, sleep-related dissociative disorder
V-PSG, video-polysomnography

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DISCLOSURE STATEMENT

All authors have read and approved the manuscript. Work for this study was performed at the Japan Somnology Center, Neuropsychiatric Research Institute, Tokyo, Japan. Dr. Futenma reports personal fees from Eisai Co, Ltd, and from MSD, outside the submitted work. Dr. Inoue reports personal fees from Eisai Co, Ltd, Otsuka Pharmaceutical Co, Ltd, Takeda Pharmaceutical Co, Ltd, Astellas Pharma, Inc, and MSD and clinical trials for Idorsia Pharmaceuticals Japan, Ltd, outside the submitted work. Dr. Inoue also reports a grant from Philips Japan Co, Ltd, outside the submitted work. Dr. Takaesu reports personal fees from Eisai Co, Ltd, MSD, Otsuka Pharmaceutical, Meiji Seika, Eli Lilly, Mitsubishi Tanabe Pharma, Yoshitomi Pharmaceutical, and Takeda Pharmaceutical, outside the submitted work. Dr. Takaesu also reports grants from Eisai Co, Ltd, MSD, Otsuka Pharmaceutical, and Meiji Seika, outside the submitted work. The other authors report no conflicts of interest.