Title

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Journal

Journal of veterinary internal medicine, 37(1)

ISSN

0891-6640

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Publication Date

2023

DOI

10.1111/jvim.16585

Received: 8 September 2022 | Accepted: 18 November 2022

DOI: 10.1111/jvim.16585

CASE REPORT

Journal of Veterinary Internal Medicine Open Access

ACVIM American College of Veterinary Internal Medicine

Electroencephalography of rapid eye movement sleep behavior disorder in a dog with generalized tetanus

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Abstract

Case Summary: A 3-month-old Airedale dog with clinically diagnosed generalized tetanus was investigated for the occurrence of excessive paddling and chewing movements when sleeping. Electroencephalogram (EEG) with time-locked video over 31 hours determined occurrence of the abnormal movements to be within 20 to 180 seconds of the onset of rapid eye movement (REM) sleep, but not at any other stage of wakefulness or sleep. No epileptiform activity was noted. Clinical signs of generalized tetanus resolved over 8 weeks with antimicrobial and symptomatic treatment, and sleep-associated movements resolved 6 weeks after presentation.

Clinical Relevance: Rapid eye movement sleep behavior disorder (RBD) has been suspected in dogs with generalized tetanus but not confirmed by correlation of repeated episodes of vocalization or motor behaviors or both with REM sleep defined by an EEG. The case further defines RBD in dogs with tetanus, and highlights the value of EEG to differentiate among different parasomnias and epileptiform activity.

KEYWORDS

Clostridium tetani, EEG, polysomnography, RBD

1 | INTRODUCTION

Parasomnias are undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousal.[1] Parasomnias may be related to the nonrapid eye movement (NREM) sleep phase, such as sleep walking and sleep terrors or related to REM sleep. Rapid eye movement sleep behavior disorder (RBD) in humans is defined by repeated episodes of sleep-related vocalization, complex motor behaviors or both documented by polysomnography to occur during REM sleep, typically associated with dream enactment. Ideally, loss of atonia documented by electromyography (EMG) also should be identified.[1]

Rapid eye movement sleep behavior disorder in humans can be idiopathic, or more often associated with comorbidities including neurodegenerative diseases, narcolepsy, ponto-medullary brainstem lesions, autoimmune disease, antidepressant medications, alcohol withdrawal, and autonomic dysfunction.[2-4] Sleep-associated abnormal movements are reported uncommonly in the veterinary literature, but REM- and NREM-associated conditions have been described. Idiopathic RBD, defined by electroencephalogram (EEG), has been reported in a Labrador puppy[5] and RBD was suspected in Nova Scotia Duck Tolling Retrievers with a degenerative encephalopathy.[6] A variety of REM and non-REM movement disorders with both idiopathic and central nervous system (CNS) lesion-related disorders were reported in 5 cats and 3 dogs based on observational and EEG documentation,[7] and suspected RBD in 13 dogs and EEG-defined RBD in 1 dog were reported with minimally defined etiologies.[8]

Abbreviations: CRI, continuous rate infusion; EEG, electroencephalogram; EMG, electromyogram; NREM, nonrapid eye movement; RBD, REM sleep behavior disorder; REM, rapid eye movement; SLD, sublaterodorsal nucleus; TeNT, tetanus neurotoxin; VMM, ventromedial medulla.

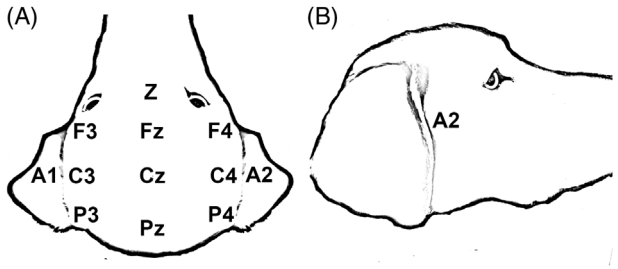

FIGURE 1 Dorsal (A) and lateral (B) view of the montage used in this case. F = Frontal; C = central; P = parietal; A = aural (placed just ventral to the zygomatic arch); Z = ground. Odd numbers = left side, even numbers = right side, z = midline

Rapid eye movement sleep behavior disorder has been suspected clinically in dogs during the course of disease associated with *Clostridium tetani* intoxication,[9-12] but not confirmed by correlation of repeated episodes of vocalization or motor behaviors or both with REM sleep defined by an EEG. We utilized continuous video-EEG to document RBD in a puppy with generalized tetanus.

2 | CASE SUMMARY

A 3-month-old intact female Airedale dog was presented to the University of California, Davis Veterinary Medical Teaching Hospital (VMTH) for evaluation of rapid-onset generalized increased muscle tone and abnormal facial expression. On neurological examination, the dog had normal mentation, was ambulatory when placed in a standing position, with marked generalized extensor rigidity and intermittent muscle spasms, trismus, risus sardonicus, miosis oculus uterque (OU), and a clinical diagnosis of generalized tetanus was made. Multiple small grass awn lesions with suppurative discharge were noted on the paws and digits, and were suspected to be the likely source of infection. The dog was admitted to the hospital and treatment for tetanus was started. In the first 24 hours of hospitalization, the dog received 10 000 IU tetanus antitoxin IV once, metronidazole 10 mg/kg IV q12h, ampicillin sodium/sublactam (Unasyn, Pfizer, New York, NY) 50 mg/kg IV q8h, midazolam constant rate infusion (CRI) 0.2-0.5 mg/kg/h IV, dexmedetomidine CRI, 1-2 μg/kg/h IV, lactated Ringer's solution 60 mL/kg/d IV, and was anesthetized for endoscopic placement of a gastrotomy feeding tube. Phenobarbital 4 mg/kg IV q8h subsequently was started, both for additional sedation as well as an anticonvulsant, because the dog exhibited several episodes of twitching, limb thrashing, chewing movements, and vocalizing that were interpreted as possible seizure activity.

The dog was nonambulatory with marked extensor rigidity, opisthotonus, and trismus for the duration of its hospitalization. Over the next 17 days of management, feedings and medications were progressively managed through the gastrotomy tube, and IV CRIs of sedating medications were tapered gradually as the muscle spasms decreased in frequency and severity. Episodes of limb thrashing, chewing, and twitching continued to be noted, but no overt generalized tonic-clonic seizure activity was ever documented.

On the 18th day of hospitalization, an EEG (Cadwell Arc Alterna EEG, Kennewick, Washington) unit was placed on the patient for continuous EEG and time-locked video recording. Subdermal 25-gauge wire electrodes (Ives EEG Solutions, Newburyport, Massachusetts) were placed in a limited montage (Figure 1) connected to an electrode harness, and the dog's head and electrodes were gently wrapped in bandage material to secure them in place. No additional sedation was administered for electrode placement. The amplifier and recording unit were placed in the cage with the dog; the video camera was placed to monitor the cage and dog, and normal patient care continued throughout the recording. The EEG was removed after 45 hours, and the recording was downloaded for review. During the EEG recording, dexmedetomidine was the only IV medication administered as a CRI at 1.25 μg/kg/h. All other medications, as well as food and water, were given per stomach tube: phenobarbital 6 mg/kg q8h, trazodone 5 mg/kg q8h, methocarbamol 50 mg/kg q6h, baclofen 0.5 mg/kg q8h, acepromazine 1 mg/kg q6h, metronidazole 12.5 mg/kg q8h, enrofloxacin 13.6 mg/kg q24h, and amoxicillin/clavulanic acid 37.5 mg/kg q8h. The dog was discharged for continued management at home on day 21 with instructions for passive range of motion exercises. Medications consisted of phenobarbital 6 mg/kg q8h, acepromazine 1 mg/kg q6h, baclofen 0.5 mg/kg q8h, methocarbamol 50 mg/kg q6h, metronidazole 12.5 mg/kg q8h.

At a reevaluation 2 weeks after discharge, the dog had generalized muscle atrophy, but overall muscle tone was considered normal, and the dog had resumed eating and drinking normally. The paddling and thrashing events during sleep continued to occur, but were lessening in severity, and over time, became limited to excessive movements of the head and face. At this visit, all medications were discontinued other than phenobarbital 6 mg/kg PO q12h. Abnormal head and face movements ceased 3 weeks after discharge. Physical therapy was increased to include passive range of movement and weight-shifting exercises and the dog was ambulatory 4 weeks after discharge. The gastrotomy tube was removed 5 weeks after discharge.

3 | ELECTROENCEPHALOGRAM FINDINGS

Forty-five hours of continuous EEG were recorded, however at 31 hours 22 minutes, the video unit stopped recording; only EEG with concurrent video was considered for manual review. No electrodes were lost during the recording, and the quality of the recording was considered subjectively to be excellent with minimal artifact and easily defined sleep/arousal stages. No epileptiform features were noted. Episodes of arousal, non-REM/slow wave (NREM) sleep, and REM sleep were documented, with distinct background rhythms. Awake/arousal was relatively high frequency (15-25 Hz), high amplitude (30-50 μV) background activity, most consistent with superimposed muscle artifact (Figure 2). The NREM sleep had low frequency (1.5-2 Hz), high amplitude (30-60 μV) slow waves, with normal sleep transients including sleep spindles and K complexes (Figures 2 and 3). The NREM sleep transitioned to REM sleep, characterized by high

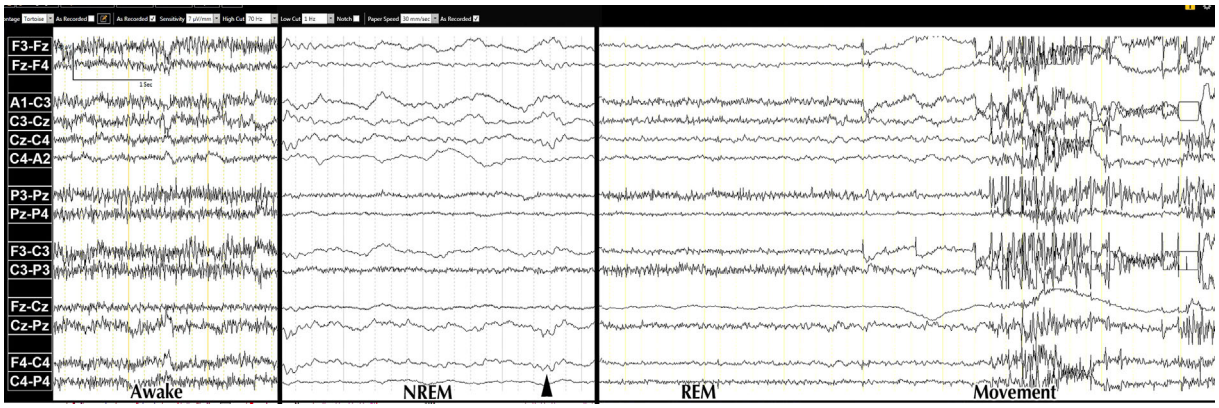

FIGURE 2 Electroencephalogram recording examples of the dog's awake background (left), nonrapid eye movement (NREM) sleep (middle), and REM sleep (right) with marked movement/muscle artifact toward the end of the epoch when the patient thrashed her thoracic limbs. A NREM-associated sleep spindle is highlighted (arrowhead). Bipolar montage; recording parameters are identical for all epochs: sensitivity 7 μV/mm, high filter 70 Hz, low filter 1 Hz, paper speed 30 mm/s

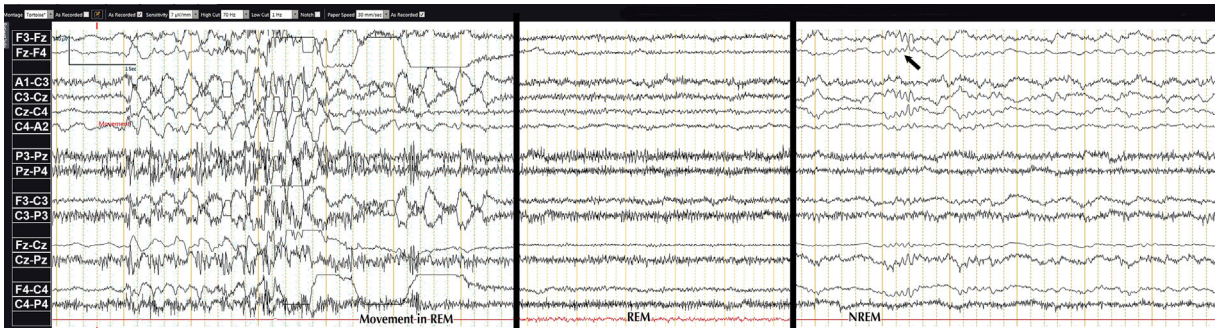

FIGURE 3 Electroencephalogram recording example of rapid eye movement (REM)-associated movement progressing back into REM sleep followed by nonrapid eye movement (NREM) sleep (NREM-associated sleep spindle indicated by arrow). Postmovement REM and NREM recordings are within a 3-minute period after cessation of movement. Bipolar montage; recording parameters are identical for all epochs: sensitivity 7 μV/mm, high filter 70 Hz, low filter 1 Hz, paper speed 30 mm/s

frequency (15-20 Hz), low amplitude (10-20 μV) background activity (Figures 2 and 3). A low level of presumed muscle artifact was present during sleep stages, which can be seen in routine EEG and may have been increased secondary to the tetanic state.

Twenty-one cycles of REM sleep were recorded, with durations ranging from 56 seconds to 10 minutes. In 17/21 instances of REM sleep, within 20-180 seconds of the transition from NREM to REM sleep, the dog exhibited vigorous movements on the video (Video S1), including paddling, head-thrashing, and chewing, reflected in the EEG by marked movement artifact (Figures 2 and 3). The EEG returned to the high-frequency/low-amplitude REM background and subsequently transitioned back to NREM sleep after 1-10 minutes of REM in 16/17 instances (Figure 3). In 1 instance, the patient woke up directly from REM sleep after the movements. Because of the marked clinical signs associated with the generalized tetanus, wakefulness was determined primarily based on the distinctive EEG activity (Figure 2). In the other 4/21 cycles of REM sleep, no movements were noted. In 3 instances, the dog awoke directly from REM sleep, and in 1 instance transitioned back into NREM sleep. The only time during the recording the dog exhibited marked limb movements or chewing was during REM sleep. When awake, the only movement noted was elevation of the head from lateral recumbency.

4 | DISCUSSION

In humans, RBD is 1 of several disorders that can manifest with violent, sleep-related, and dream-related behavior.[1] The American Academy of Sleep Medicine describes mimics of RBD in humans to include sleepwalking, sleep terrors, obstructive sleep apnea (OSA), nocturnal seizures, sleep-related movement disorders, sleep-related dissociative disorders, hypnopompic hallucinations, and post-traumatic stress disorder.[1] Diagnosis of several of these disorders requires verbal description of associated dreams and phenomena making definitive diagnosis in animals challenging. Defining the state of consciousness (arousal, NREM sleep, REM sleep) however is a key component of diagnosis and, although experienced observers may be able to define sleep stages relatively consistently in dogs,[13] EEG is required for definitive determination. Sleepwalking, sleep terrors and confusional arousals result from an admixture of wakefulness and NREM sleep inconsistent

with the repeatable REM-associated activity in this dog. Sleep terrors in humans are not dream-associated, can have associated motor behavior, and are differentiated from nightmares that typically arise out of dream-associated REM sleep but result in awakening with no associated motor behavior.[1,14] Obstructive sleep apnea-induced arousal from REM sleep and hypnopompic hallucinations that arise on awakening from REM sleep may mimic RBD. Hallucinations may result in patients jumping out of bed in terror, unlike RBD where patients' movements generally reflect acting out their dreams.[1,15] Verbal-based differentiation is not possible in animals, but the absence of observable apneic episodes and the transition from movement events directly back into REM and subsequently NREM sleep (Figure 3), rather than waking, in 16/17 documented episodes in our case makes apnea or hallucination-related disorders unlikely. Epileptiform activity on EEG was not observed, and loss of identifiable sleep stages, typical of status dissociatus, was not present.[1] Although RBD can be associated with periodic limb movements in addition to more complex RBD-associated movements, sleep-related movement disorders such as periodic limb movement disorder or sleep-related bruxism typically involve simple and stereotyped movements rather than the complex and variable limb paddling and chewing observed in our case.[16] Periodic limb movements in movement disorders also usually are absent during REM sleep and are frequent during arousal or the N1-3 stages of NREM sleep.[16]

Rapid eye movement sleep in normal dogs is characterized by loss of postural muscle tone (atonia) with preservation of movements of small distal muscles (extraocular, facial, digital, and tail) and diaphragm.[13] Control of REM sleep and associated postural atonia is incompletely understood,[17-19] but based on data from primarily rodent and cat models, REM sleep atonia is governed by neural circuitry in the pons and medulla. Sublaterodorsal nucleus (SLD) glutamatergic/gamma amino butyric acid (GABA)-ergic (predominantly glutamatergic) neurons in the dorsal, rostral pons project directly to synapse on inhibitory glycinergic/GABA-ergic spinal interneurons or indirectly on glycinergic/GABA-ergic premotor neurons in the ventromedial medulla (VMM) that subsequently project to directly inhibit spinal and brainstem motor neurons, resulting in postural atonia.[17-20] Atonia-controlling cells in the VMM have been specifically defined in narcoleptic dogs associated with atonia of REM sleep and cataplexy.[21] The relative contribution of these 2 pathways to REM-associated atonia is not clear, but SLD input is both necessary and sufficient to produce atonia. Stimulation of the SLD region causes loss of postural tone, and experimental animals or human RBD patients develop REM without atonia.[17,20] Medullary lesions also can result in REM without atonia, but pontine (SLD) glutamatergic input appears to be necessary for medullary-induced muscle tone suppression.[17,20]

Experimental and clinically documented lesions in humans strongly suggest that damage to REM sleep circuitry contributes to RBD,[17-19] however the mechanism for tetanus neurotoxin (TeNT)-induced RBD in dogs has not been defined. Although skeletomotor neurons and their associated inhibitory interneurons are the primary targets for TeNT,[22] the toxin has been shown experimentally to have effects on other neuronal subtypes including neocortical, hippocampal, excitatory, and cholinergic and adrenergic autonomic neuronal elements[22-28] Targeting of *C. tetani* neurotoxin (TeNT) in dogs to noncanonical targets such as glycinergic VMM neurons, or excitatory glutamatergic neurons of the SLD could result in loss of REM atonia and RBD. However, TeNT appears to preferentially affect inhibitory (glycinergic) rather than excitatory (glutamatergic) synapses,[22,25,27] which would support effects on the VMM inhibitory neurons as a more likely target in dogs with tetanus and RBD. In support of this mechanism, a recent study identified RBD-like activity with loss of muscle atonia in a Cre-Lox mouse model expressing tetanus toxin light chain in glycinergic neurons of the VMM.[20]

Although RBD-like activity has been identified in a tetanus toxin rodent model,[20] RBD secondary to tetanus has not been described clinically in species other than dogs, even in species that appear to have a greater sensitivity to TeNT such as humans.[29] Binding, internalization, and transport of TeNT is incompletely understood, and involves many cellular receptors and transport mechanisms.[22] Species differences in receptor and transport profiles in canonical (motor neuron/inhibitory interneuron) versus noncanonical (REM-atonia pathway) target neurons may underly the apparently discordant sensitivity of dogs and humans to spasticity and RBD-associated phenotypes.

Limitations of our report include the presumptive diagnosis of tetanus based on history and clinical signs and the absence of diagnostic procedures to investigate additional potential underlying causes other than tetanus.[2-4] . Although culture of the suspected infection site was not attempted, diagnosis of tetanus is by necessity often presumptive,[9-12] and the clinical and drug exposure history, and resolution of clinical signs would not be consistent with progressive intracranial disease or drug-related RBD. For comprehensive definition of RBD, REM sleep without atonia ideally should be identified on by EMG during polysomnography,[1,30] and REM sleep without atonia in the absence of clinically apparent RBD can occur.[1,30] Loss of muscle tone in our dog was defined visually during video-EEG recording based on video-recorded overt movement and associated EEG muscle artifact recordings, however, it is possible that additional small motor events may have been overlooked. As with all but a single previously reported tetanus-associated case,[9-12] our dog had a generalized disease presentation, and resolution of RBD was seen with resolution of the underlying tetanus-associated spasticity with no specific treatment. This outcome further supports the absence of additional underlying causes of RBD, because RBD in dogs with documented or suspected CNS disease frequently persisted or progressed with variable improvement with medical treatment.[5-8]

The recumbent nature of our dog, because of severe generalized disease, enabled acquisition of high quality, prolonged EEG data consistently defining all sleep stages through multiple sleep cycles. This information allowed definitive association of paroxysmal motor behaviors with REM sleep and rational differentiation of RBD from common differential diagnoses reported for human patients with suspected RBD. This case highlights the value of EEG in differentiation of paroxysmal disorders including different types of parasomnias and epilepsy, and further defines the clinical presentation of RBD in dogs with *C. tetani* intoxication.

ACKNOWLEDGMENT

No funding was received for this study. Neurology/Neurosurgery residents (Colleen Embersics, Kelly O'Connell, Wojciech Panek), students (Julie O'Neill, Elisabeth Sevy, Jasmine Huynh, Bradley Whelchel), and VMTH Clinical Patient Care Staff – provided exceptional care for this dog.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Knipe M, Embersics C, Dickinson P. Electroencephalography of rapid eye movement sleep behavior disorder in a dog with generalized tetanus. *J Vet Intern Med*. 2023;37(1):277-281. doi:10.1111/jvim.16585