

DISSOCIATIVE DISORDERS
PRESENTING AS
SOMNAMBULISM:
POLYSOMNOGRAPHIC,
VIDEO AND CLINICAL
DOCUMENTATION
(8 CASES)

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An earlier version of this paper was presented at the Association of Professional Sleep Societies, Second Annual Meeting, San Diego, June 11-15, 1988.

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ACKNOWLEDGEMENTS: This work was supported in part by a grant from Hennepin Faculty Associates.

ABSTRACT

A polysomnographic (PSG) and clinical study of 150 consecutive patients presenting to a sleep disorders center during a 7.5 year period for evaluation of repeated sleep-related injury (ecchymoses, lacerations, fractures) identified 5.3 percent (N=8) with Dissociative Disorders (DDs) as the cause of the injuries, and whose presenting diagnosis was somnambulism. 87.5 percent (7/8) were female, and the mean age at referral was 29.5 (+/-SD 6.1) years. Two patients fulfilled DSM-III-R criteria for Multiple Personality Disorder (MPD). The six other patients were diagnosed as Dissociative Disorders Not Otherwise Specified, but were strongly suspected to have MPD. One patient had an exclusively nocturnal, animalistic DD: a 19 year old male who had acted like a large jungle cat twice weekly for 4 years. PSG studies were diagnostic for nocturnal DD in 50 percent (4/8) of the cases (including that of the jungle cat), with distinctly altered, complex, repetitive and lengthy behaviors emerging suddenly from sustained electroencephalographic wakefulness. PSG studies supported the diagnosis of DD as the cause of nocturnal injury in the other 50 percent (4/8) of the cases: i) by not detecting seizure activity, NREM/REM sleep motor abnormality or sleep breathing disturbance, and ii) when correlated with the clinical history of chronic daytime DDs.

The original diagnostic Classification of Sleep and Arousal Disorders includes Psychogenic Fugue as the only Dissociative Disorder (DD) in the differential diagnosis of Somnambulism or Sleepwalking (SW), which is defined as a disorder arising from non-REM (NREM) sleep (Roffwarg, 1979). One reported case of presumed SW was found to arise

from electroencephalographic (EEG) wakefulness during a formal overnight polysomnographic (PSG) study which, when correlated with the clinical history, established the diagnosis of a DD (Fleming, 1987). The paucity of PSG data in DDs is in contrast to a considerably more extensive psychological understanding of them (Kluft, 1988; Putnam 1989; Nemiah, 1989). We now will report on a PSG and clinical study which identified 8 patients with nocturnal DDs who had been referred to our sleep disorders center with the provisional diagnosis of injurious SW. The differential diagnosis of dangerous sleep-related behaviors will be addressed in relation to our findings.

METHODS

Seven patients with DDs (Patients 1 and 3-8) were identified in a series of 100 consecutive adult patients presenting clinically over a 6 year period with a chief complaint of repeated nocturnal injury (ecchymoses, lacerations, fractures) (Schenck, Milner, Hurwitz, Bundlie, & Mahowald, 1989). Patient 2 was identified when our series was expanded to 150 patients with the same complaint, gathered over 7.5 years. All 150 patients underwent: 1) Overnight PSG evaluations; 2) Clinical sleep/wake evaluations consisting of interviews structured by a standard questionnaire which also covered past history, review of systems, 31 questions on medication and substance use, and 9 questions from the Michigan Alcoholism Screening Test; 3) Clinical psychiatric interviews conducted by the authors (CHS, TDH) before the PSG studies to determine the presence of DSM-III Axis 1 disorders. Past psychiatric and medical records were obtained and relevant information was elicited from the patient's psychiatrists, psychotherapists, and family members. DSM-III-R criteria were ultimately utilized for assigning DD diagnoses.

PSG data were obtained by protocol, utilizing standard methods for recording and scoring (Rechtschaffen & Kales, 1968). Patients slept overnight in the laboratory during their usual hours. PSG monitoring included an electrooculogram, EEG, electromyogram (chin, bilateral anterior tibialis muscle and extensor digitorum muscle), electrocardiogram, and nasal-oral thermocouple air flow. Full respiratory monitoring was utilized in any patient demonstrating altered airflow. A 9-channel scalp EEG montage was used, with a paper speed of 15 mm per second and also of 30 mm per second for 5 minutes every hour. The subjects were continuously videotaped. Any psychotropic medications were discontinued at least 10 days prior to PSG study, apart from 4

patients who were continued on maintenance pharmacotherapy (one each: lithium carbonate; phenelzine; thiothixene; carbamazepine/amitriptyline). Urine toxicology screens were obtained at the time of PSG studies.

RESULTS

DDs were diagnosed as the cause of repeated nocturnal injury in 8 patients (5.3%) from the series of 150 consecutive patients, on the basis of positive and of exclusionary PSG findings, described below, which were correlated with the psychiatric findings. Urine toxicology screens were negative for illicit substance use. Case histories and PSG data will now be presented.

Exclusively Nocturnal Animalistic Dissociative Disorder

Case 1. A 19 year old male presented with a 4 year history of stereotypic spells recurring 1-2 times weekly, but appearing exclusively at night. These episodes typically began 1-2 hours after retiring, when suddenly he would leave the bed (Figure 1A) while growling, hissing, crawling, leaping about, and biting objects in the manner of a large jungle cat for periods of up to 1 hour, terminated by an abrupt collapse and unresponsiveness while perspiring profusely. Although amnesic for his actions, the next morning he invariably recalled a specific recurrent "dream" of being a lion or tiger let out of his cage by a woman zookeeper whom he then followed down a path. She held a "piece of raw meat," but he could not leap to snatch it "because of an invisible force field" which then made him feel "disappointed" and "frus-

trated." The "dream" always ended with "someone shooting a tranquilizer gun at me" and then he would fall down and become unconscious.

The patient considered the "dream" action, which closely mirrored his actual behaviors (for which he was ostensibly amnesic), to be "very vivid and real," and expressed interest in recreating the dream by making a movie of it. Although the "dream" plot was always identical, the face of the woman zookeeper varied from episode to episode and was never familiar. He emphasized that during this "dream" he felt completely like an animal and had no awareness of being human.

His family had never heard the patient speak nor interact verbally with them during these spells. His postures and behavioral repertoire (including an impressive transformation of hands into facsimiles of paws) were consistently those of a large cat. He successfully navigated the environment, as when he commonly opened the refrigerator with his mouth, put uncooked bacon between his teeth and then proceed to prowl around the house.

His family commented on his repeated feats of "superhuman strength," such as leaping far from his bed, lifting a mattress with his jaws and dragging it across a room (Figure 1B), or lifting a marble table with his jaws. He frequently left imprints of his teeth on the furniture during these nocturnal episodes, but never left the house, nor did he sit or stand up: his movements were constantly quadrupedal. He typically remained unresponsive to people during and immediately after these spells, despite having the family talk, shout, shake him or splash cold water on his face. However, oppositional

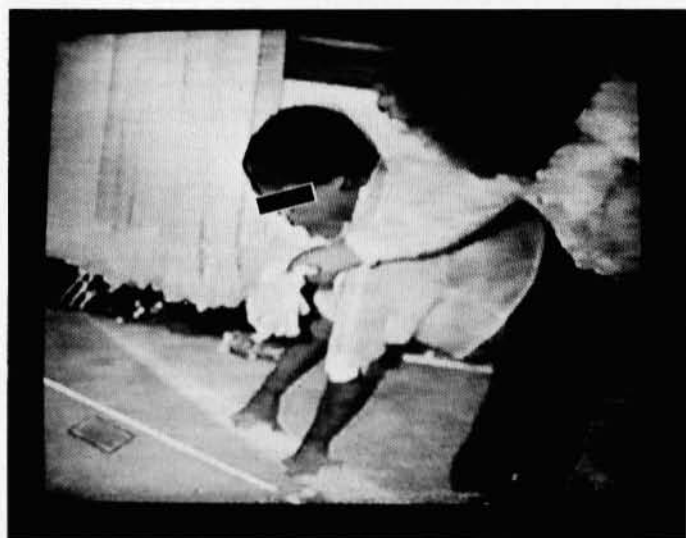


Figure 1A

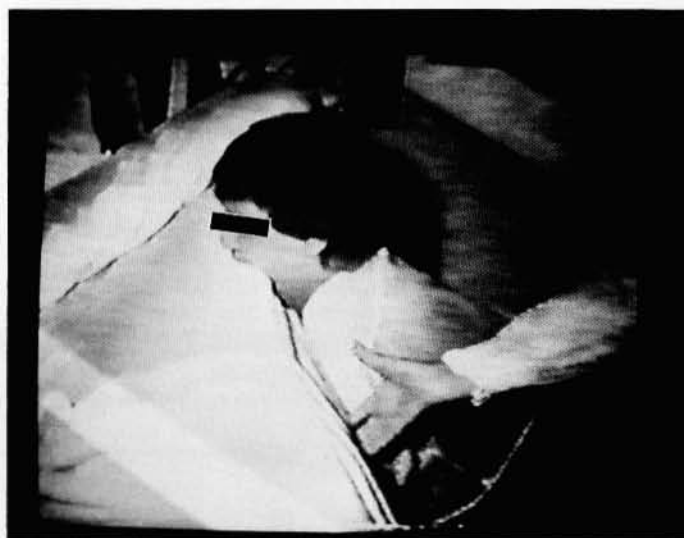


Figure 1B

FIGURES 1A-1B
Video Prints of a Nocturnal Dissociative Episode

Still frames from a home movie record how a 19 year old male crawls around his room while assuming the identity and behaviors of a large jungle cat. He is seen to clamp his teeth on a towel offered by his mother (1A). He then drags a mattress across the room with his teeth as his mother attempts to intervene (1B). Two identical episodes were documented to arise from electroencephalographic wakefulness during overnight polysomnographic study with continuous audio-videotaping.

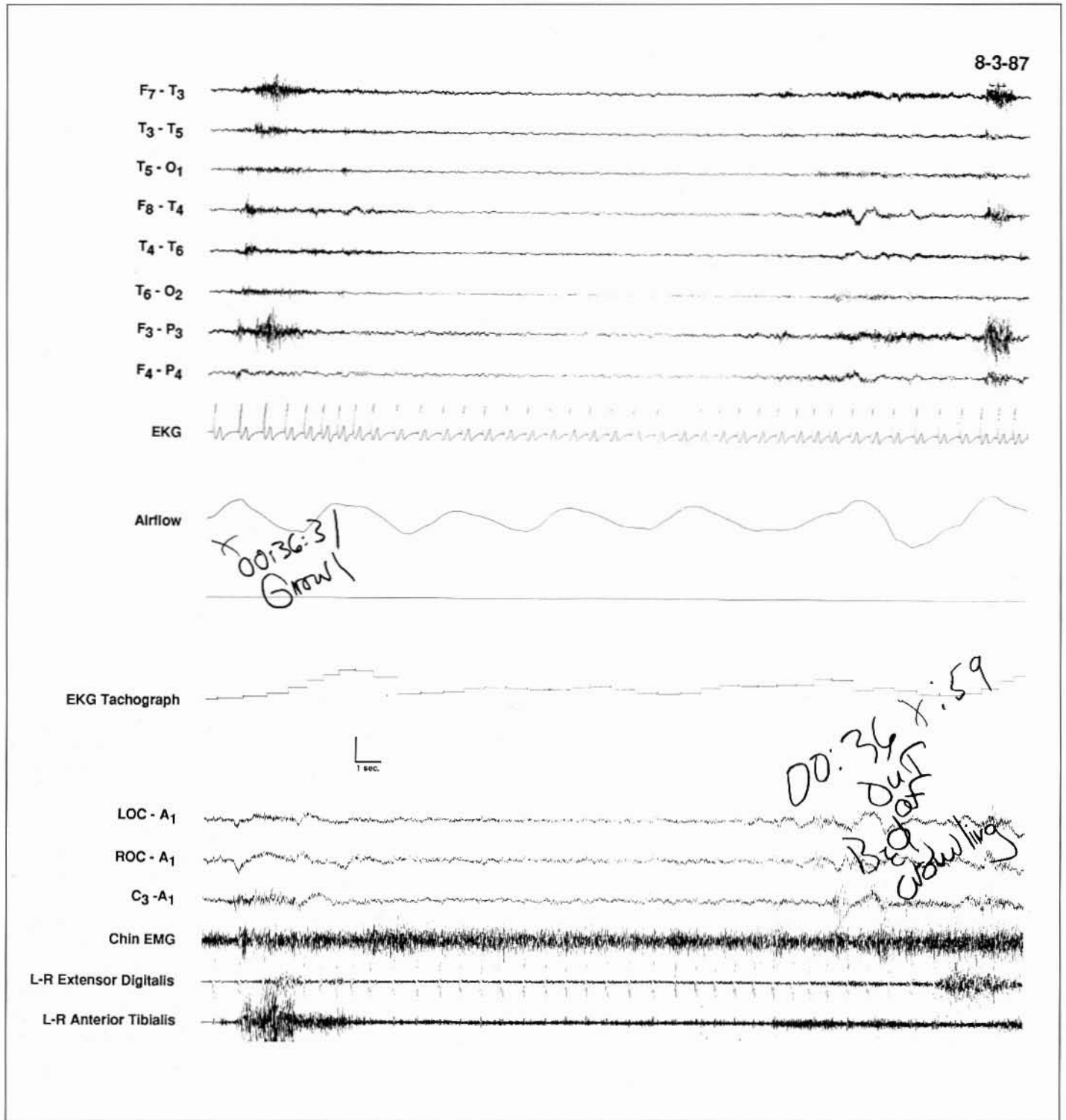


FIGURE 2
Nocturnal Polysomnogram of an Animalistic Dissociative Episode

53 minutes after sleep onset, a 19 year old male suddenly begins to growl and then leave his bed and crawl away in the manner of a large jungle cat, as noted by the sleep laboratory technician. A 9 channel electroencephalogram (EEG), (the top 8 channels and C3-A1), indicates a corresponding wakeful state, characterized by desynchronized, low-voltage, fast activity ("activated EEG"). The chin electromyogram (EMG) shows a moderate level of tone throughout the tracing, and there is intermittent twitching of the extensor digitorum and anterior tibialis EMGs. Minimal eye movements are present (LOC-A1; ROC-A1).

behavior was observed on occasion, as when he actively avoided or resisted his mother's attempts to wipe his perspiring brow. Whenever his father held him during an episode, he would engage in prolonged continuous four-legged, animalistic, ambulatory behaviors while being held upright. Urinary or fecal incontinence was never noted, nor was there any history of tonic-clonic movements.

He had injured his lips and gingiva on numerous occasions from biting sharp objects, and also had sustained ecchymoses and lacerations on his trunk and limbs. Excessive daytime sleepiness also had been present throughout the 4 year course of his nocturnal disorder. His family brought a videotape — which they had recorded at home — to the initial consultation to help convince physicians of the reality of these chronic peculiar events. The patient was observed to weep in disbelief upon viewing his videotaped nocturnal behaviors, and his family openly referred to his "good" (i.e. diurnal) and "bad" (i.e. nocturnal) "sides."

The onset of this disorder had no recognized precipitant, nor did any particular episode have an identifiable trigger. Shortly after the onset, he was hospitalized for 1 month on a psychiatric unit for evaluation and treatment of his sleep-related problem. He was diagnosed as having a major depressive episode, but continued to have his typical nocturnal spells in the hospital despite treatment with various anti-depressant medications and also behavioral and psychotherapeutic interventions. Over the ensuing years, long-term family therapy and several courses of individual psychotherapy failed to control his nocturnal spells. Within the year prior to referral to our center, therapeutic trials with tranylcypromine and later with clonazepam, 0.5 mg h.s. had aggravated his condition.

The patient was born prematurely and developed a post-partum respiratory distress syndrome (hyaline membrane disease). He was adopted at age 10 months. During infancy and childhood he was noted to have consistently prolonged sleep latencies and excessively restless sleep which had prompted the parents to place fishnets around his bed and later lock him in the room with a screen door during the night. A learning disorder was first detected in kindergarten and an Attention Deficit Disorder (ADD) was diagnosed at the age of 8 years. Comprehensive evaluations were completed at the ages of 8, 10, and 18 years (involving neurologic, neuropsychologic, psychological, speech and language, occupational therapy, physical medicine and vocational assessments). Multiple severe impairments were found: 1) gross motor delays; 2) visual receptive deficit severely affecting reading, spelling and arithmetic performance; 3) visual motor deficit; 4) auditory processing and verbal receptive deficits, including left-sided sensory-neural hearing loss; 5) impairment of abstract thinking and cognition; 6) attention and short-term memory deficits. Testing indicated that he performed best (i.e. at age level) in the area of verbal expression. He performed very poorly (usually at the lowest percentile) on almost all aptitude tests. Despite intensive special education, he was unable to complete a secondary education beyond the eleventh grade.

Numerous neurological examinations and waking EEGs had been normal. A magnetic resonance brain scan at age 18

years revealed only focal atrophy adjacent to the right hippocampal gyrus, of unlikely clinical relevance. Treatment of the ADD with methylphenidate and later dextroamphetamine had been aborted on account of adverse effects. However, for the year prior to referral, he took methamphetamine, 5 mg every morning, to improve daytime alertness, with partial benefit but with no influence on the nocturnal behavior disorder.

He was generally regarded to be a well-behaved, friendly person with several close peer relationships. He regularly participated in athletic activities. His sister reported that he had an excessive attachment to his mother and exhibited intense separation reactions whenever she slept away from home. There was no history of physical or sexual abuse since the time of his adoption at 10 months, with the antecedent history being unknown.

Two consecutive overnight PSG studies were completed. On the first night, two of his characteristic episodes were documented, and each arose from EEG wakefulness. The first occurred 53 minutes after sleep-onset (subsequent to cycling through Stages 1, 2 and 3/4 NREM sleep). There was a 2 minute prodrome of intermittent growling which began several minutes after an epoch of Stage 1 sleep had terminated in sustained EEG wakefulness without behavioral arousal (i.e., the patient lay still and appeared to remain asleep). He then abruptly left the bed and crawled around the room, hissing, growling, loudly grinding his teeth and pulling the mattress with his jaws. He also chewed and swallowed portions of the airflow monitoring device. After 6.5 minutes he abruptly collapsed on the floor while his hands continued to be contorted as if they were paws. After another 2 minutes he was up again, crawling and growling for 4.5 minutes before a final collapse and 30 seconds of clinical unresponsiveness. Normal wakeful EEG activity continued throughout this entire event. He then awakened from this spell and described his typical "dream" in full detail, but was amnesic for all his actions.

The second episode emerged 1 hour 15 minutes after the first episode (having slept during NREM and REM sleep). During EEG wakefulness he growled for 15 seconds before crawling out of bed, knocking a lamp over, biting that lamp, chewing and swallowing portions of another airflow monitoring device and repeatedly banging his head against a wall. The entire episode lasted over 9 minutes and terminated abruptly with collapse; 20 minutes later he became responsive and reported "the same dream again, nothing different." A wakeful EEG was maintained throughout this second episode.

The EEG during both episodes was consistently that of wakefulness or drowsiness without any detectable seizure activity (Figure 2). During the first PSG study, the sleep efficiency (time asleep/time in bed) was markedly reduced at 53.5 percent, and there was only one REM period. The second PSG study was unremarkable, with a sleep efficiency of 99.0 percent. The day after the second (i.e., normal) PSG study, a Multiple Sleep Latency Test (MSLT) (Richardson, et al., 1978) revealed severe hypersomnolence, with a mean sleep latency of 3.0 minutes but without REM sleep on any nap. Thus, positive findings from PSG study and MSLT estab-

lished the diagnoses of nocturnal DD and also Idiopathic Central Nervous System Hypersomnolence (Roffwarg, 1979). The patient was offered treatment with hypnotherapy, which was viewed by the patient and his family as another form of "psychological therapy," and, as such, was received with skepticism and disappointment. After an apparent reduction in the frequency of spells following the initial hypnotherapy session, he did not return, and was then referred back to his psychiatrist.

Nocturnal and Diurnal Dissociative Disorders (Cases 2 - 8)

Case 2. A 34-year-old, 6 foot 1 inch and 288 pound woman was referred independently by two psychiatrists for evaluation of problematic sleep-related behaviors. There was an unremarkable sleep history until 5 months previously when major stressors triggered an abrupt onset of the presenting disorder. As verified by her sister, within a 9 day period the patient's father suddenly died of a myocardial infarction, the patient had her eighth miscarriage (at 16 weeks gestation), her mother committed suicide, and her second husband left her. She then saw the two referring psychiatrists, informing neither of her visits with the other, for the purpose of "coping with the overwhelming stresses." She was diagnosed to have Post-traumatic Stress Disorder (PTSD) and Major Depression, for which treatment with fluoxetine and amitriptyline were ineffective, although full therapeutic trials were not completed. She repeatedly refused psychiatric hospitalization and failed to engage in any ongoing psychotherapy despite seeing various psychologists, which she attributed to the fear that "they would die on me like my father did."

The sleep-related episodes occurred at least twice a week, and daily on a sporadic basis, generally beginning more than 2 hours after perceived sleep onset. The two most elaborate episodes involved driving to an airport, appropriately dressed but without luggage, purchasing a ticket and boarding flights to distant cities. On both occasions, she "awakened" en route and immediately had a panic attack with confusion as to where she was and where her 2 year old daughter was (actually at home). Upon landing, she boarded a return flight home, and resumed her life, with complete amnesia for how she had left home and boarded an airplane. Most episodes involved wandering around her yard or the streets while clad in a nightgown. She frequently crashed through a glass door leading to the back yard, broke windows, ran into furniture and sustained repeated ecchymoses and lacerations in the process. One laceration of her arm required six stitches to repair. Other episodes involved cutting her hair in a bizarre fashion, shredding her daughter's new snowsuit, ripping sweaters, tearing down drapes, putting clothes in boxes, and moving drawers around the house. She had driven her car long distances several times. Once, she "came to" at a gas station 200 miles away. These abnormal events occurred with comparable frequency and form both while working at night and sleeping during the day, and vice versa. She considered them "escape valves for my grief process." She routinely was amnesic for her actions upon terminal awakening from sleep. She was heard to scream only once, and reportedly did not engage in eating or sexual

behaviors during sleep.

Past medical history (verified by her sister and hospital records) was remarkable for meningitis 18 years previously, infectious mononucleosis 16 years previously, thyroidec-tomy 11 years previously for thyroid cancer, hypertension for 4 years and peptic ulcer disease. She also had developed post-traumatic epilepsy 3 years previously after a head injury. Carbamazepine was effective in controlling her seizures (there were no reported spells during sleep), and was discontinued one month before the onset of her sleep-related disturbance, without recurrence of seizures. Two months before referral, she was hospitalized with documented myocardial infarction and had an abnormal coronary angiogram.

There was no premorbid psychiatric history apart from addiction to oxycodone 18 years previously, which originally was prescribed to control pain after a lumbar laminectomy. She then successfully completed inpatient Chemical Dependency treatment.

Other relevant history consisted of her i) first husband (8 years her senior) dying in an accident; ii) her second husband (16 years her senior) separating from her on numerous occasions (she repeatedly referred to him as a "jerk" and claimed not to know why they were married); and iii) another sister dying in an automobile accident two months before referral. The sister reported that the patient was the most intelligent and accomplished person in a family of high achievers. She had a master's degree and was employed as the installer of large business computer systems, which entailed moving from city to city as projects were completed. Psychiatric interview at the time of referral established that she had PTSD, Major Depression, Panic Disorder (with at least 2 major attacks daily), Thanatophobia, and a DD. She was alert, with depressed affect and intermittent tearfulness. There were several panic attacks during the initial interview, each lasting 3-5 minutes. She was not manifestly psychotic. Two consecutive PSG studies performed during her usual daytime sleeping hours did not reveal any electrical or clinical seizure activity, nor any abnormality of REM sleep. Periodic limb movements of NREM sleep (Roffwarg, 1979) were detected, with indexes of 43/hour and 48/hour respectively on the two PSG studies. There was no instance of behaviors arising from NREM sleep (as may be seen in Night Terrors [NTs] and/or SW), but rather there were *two behavioral episodes arising from unequivocal EEG wakefulness* during the first PSG study. The first episode occurred 1 hour 17 minutes after sleep onset, after she had cycled through Stages 1, 2, 3/4 NREM and also REM sleep. She left the bed and wandered into a hallway appearing confused to the sleep center technician while asking what day it was, what time it was, etc. The second episode occurred 4 hours 46 minutes after sleep onset — after she had been awake for several minutes with clear EEG wakefulness — when suddenly she sat up, hyperventilating and was unable to talk for the next 3 minutes, at which time she became coherent and subsequently returned to sleep.

The PSG studies thus confirmed the clinical suspicion of a sleep-related DD. Treatment with hypnosis was attempted, but the patient experienced a panic attack while in a light

trance, abruptly left the psychiatrist's office, and refused further attempts at hypnotherapy. She then was referred to the Chief of Psychology at our hospital for psychotherapy, but saw him only once as she was convinced that "he will die too since he is the same age my father was when he died." The psychologist noted in the chart that "she is unprepared to deal with the traumatic events in her life, and states that she prefers having her current symptoms — as disturbing as they are — to exposing herself to the pain of facing the tragedies which she has recently experienced. The patient is using massive repression, claims to not think at all about the tragedies in her life, her health, or her current situation with her husband." The patient also refused a sodium amylal interview. She repeatedly mentioned that "I want a magic cure without emotional pain."

Carbamazepine was re-started, but a full trial with therapeutic blood levels (9.0 mcg/ml) was not beneficial and so it was discontinued. Prior treatment with clonazepam, 1 mg hs, had been ineffective. Current treatment with diazepam 10 mg q.i.d. had been partially effective in controlling her panic attacks, but had no effect on her sleep-related spells. The patient hired a live-in housekeeper to protect her daughter from any potentially dangerous behaviors on her own part, and also to watch over her daughter during the patient's wanderings. On several occasions, the patient was witnessed to have diurnal episodes of DD during which she wandered to the newborn unit of a hospital and stared at the babies through the viewing window. Although claiming amnesia for these actions, she later recalled searching for her most recently miscarried child whom she was convinced was still alive. Despite the severity of her major psychiatric disorders, the patient continued to work in an accomplished manner.

Her sister then reported to us on several occasions that for the previous month, (i.e., beginning 7 months from the onset of her disorder), the patient referred to herself by various new names and had been assuming at least three "different personalities," consisting of childlike, conservative/highly competent, and rageful alters which exhibited distinctive voices and mannerisms.

Credit cards bearing three different names, but the same billing address, were found in the patient's possession. Furthermore, the sister discovered an "unusual assortment of clothes" in the patient's closet, ranging from conservative garments (appropriate for the predominant alter) to the "completely atypical, glow-in-the-dark, horrible pink dresses." The patient was lost to follow-up 9 months after initial consultation, but shortly after recontact with our sleep disorders center, her three-year-old adopted daughter died of a congenital syndrome which had been detected at birth. The patient's sister confirmed this history and noted that the patient did not weep in grief, although some brief tearfulness was observed. Nocturnal wanderings from sleep then intensified in frequency, as she would go to the yard, without breaking glass, and look around or stare blankly, several times weekly. She resumed working 10 days after her daughter's death.

The sister eventually revealed that the patient had been their father's repeated incest victim, and at the age of 14

became pregnant and subsequently delivered a son-brother, who, twenty years later, still lived in close proximity to her. The sister emphasized that the sexual abuse had occurred "not only with our mother's knowledge, but with her approval." DSM-III-R criteria for Multiple Personality Disorder (MPD) and Psychogenic Fugue (the two airplane trips) were satisfied by this case history.

Case 3. A 22-year-old female presented with a long-standing history of nightly spells occurring at sleep onset, characterized by screaming, agitation, falling out of bed, moving about the room, and sustaining ecchymoses and lacerations. She was invariably amnesic for these actions, but often had associated recall of emotionally and physically traumatic childhood events upon questioning after these episodes.

There was a premonitory psychiatric history including: 1) Recurrent Major Depression with multiple hospitalizations after suicide attempts; 2) Dysthymic Disorder; 3) PTSD; 4) Somatoform disorder, conversion type; 5) Eating disorders (anorexia nervosa/bulimia); 6) Borderline Personality Disorder; 7) and DD, including recurrent amnesic episodes and fugue, during which times she would inflict self-injury by head-banging, cutting herself with knives, etc. Although the most likely diagnosis is MPD, lack of sufficient corroborative history precludes the definitive diagnosis of MPD, thus resulting in the classification of Dissociative Disorder Not Otherwise Specified (DDNOS).

PSG study documented a typical spell lasting 7 minutes which arose from EEG-defined wakefulness as she lay quietly in bed with eyes closed prior to sleep onset. She began by jerking her head from side-to-side, which progressed to squirming in bed followed by violent thrashing behaviors with moaning and groaning. When the episode ended, she was unresponsive for about 1 minute and then reported a "dream" in which "my sister beat me up, hitting me with a stick in my stomach and legs." EEG seizure activity was not detected, and EEG wakefulness continued throughout this spell and the following period of apparent unresponsiveness.

Case 4. A 32-year-old female presented with an 8 year history of injurious nocturnal behaviors characterized by leaving the bed at any time of the night, and wandering around the house. She had often burned herself with lit cigarettes, cut herself with knives, collided with furniture and sustained ecchymoses for which she was always completely amnesic. She often experienced several episodes during a single night. This problem was typically aggravated by a variety of stresses, including psychotherapeutic sessions in which she would discuss her childhood experiences of being physically and sexually abused, and then frequently shift into altered personality states involving a separate child-like self and a separate rageful self, for which she was subsequently amnesic. Treatment with clonazepam and with other benzodiazepines and minor tranquilizers did not control these nocturnal episodes.

Psychiatric history was remarkable for: 1) Recurrent major depressions with numerous hospitalizations after

suicide attempts, 2) Dysthymic Disorder, 3) PTSD, 4) Alcohol and Substance Abuse Disorders, in remission, 5) Borderline Personality Disorder, 6) DDs (MPD and Psychogenic Fugue, with numerous spells occurring in the presence of her psychotherapist).

During PSG study, the patient had an abrupt episode of altered consciousness, accompanied by slurred speech, crying, and peculiar posturing during electrode placement in preparation for PSG study. The EEG immediately afterwards showed normal wakefulness without any suggestion of an ictal or post-ictal state. Her overnight PSG record was unremarkable, apart from complete suppression of REM sleep attributed to phenezine therapy for her affective disorders. EEG abnormalities and NREM sleep disorders were not detected.

Case 5. A 30-year-old woman presented with a three year history of dangerous sleep-related behaviors which usually appeared 3-4 hours after sleep onset. Her husband reported that she would suddenly scream loudly and begin to pound her fists and bang her head. She would leave the bed and once had grabbed a butcher's knife and approached her husband in a threatening manner, but he managed to subdue her. She then typically returned to sleep and was subsequently amnesic for all of these experiences. These episodes initially appeared three nights weekly, and eventually as often as twice nightly. She had once fractured her ulna while falling out of bed, had lacerated her tongue, and had repeatedly sustained ecchymoses and injuries to her hands. At the time of referral, the patient was receiving psychiatric treatment for a Dysthymic Disorder. Her childhood was psychologically traumatic, as she had lived in a succession of foster homes. She identified the circumstance of permanent separation from parents as having had a "devastating effect" on her. The husband observed her to have longstanding, recurrent spells very suggestive of daytime DD, for which she was amnesic.

Two consecutive PSG studies were normal. Unusual behaviors were not observed, nor was there any EEG seizure activity or abnormality of REM/NREM sleep. The final psychiatric diagnoses included Dysthymic Disorder, PTSD, and DD (probable MPD, but DDNOS was assigned, given insufficient clinical data). A nocturnal DD was inferred on the basis of her daytime history of DD coupled with the negative PSG findings.

Case 6-8. Three females, aged 29-36, presented with histories of injurious nocturnal wanderings associated with multiple severe psychiatric disorders. One patient, Case 6, had lifelong recurrent episodes of DD originating in childhood when she was repeatedly abused sexually and physically by her parents. She had mutilated herself numerous times, almost exclusively by cutting her vagina and vulva. Her nocturnal spells of wandering and self-injury usually occurred within 90 minutes of sleep onset and she had at times walked outdoors during winter nights. She was always amnesic for these episodes, which occurred 4-7 nights weekly. When she regained self-awareness, generally she would be in the midst of cutting herself and she would report having the

experience of being in a strange place, surrounded by the frightening presence of her parents whom she could see and hear in a "distorted" manner. The daytime episodes of DD, which had often been observed during psychiatric hospitalizations, typically involved agitation and assaultiveness. There was a history of recurrent major depressions, alcohol/substance abuse disorder in remission, borderline personality disorder, PTSD, and her DD was presumed to be MPD but was classified as DDNOS because of insufficient clinical information.

Another patient, Case 7, was also a victim of childhood incest who had had both diurnal and nocturnal spells of cutting herself, for which she was amnesic. She also had a history of Recurrent Major Depressions with suicide attempts, PTSD, Substance abuse disorder in remission, and Borderline personality disorder. Her DD was also probably MPD but was diagnosed as DDNOS. The final patient, Case 8, had a history of daytime episodes of DD and also of nocturnal episodes during which she would leave the house, with subsequent amnesia. She had a history of recurrent major depression, childhood-onset PTSD, and anorexia nervosa. Her DD was classified as DDNOS, although the history was very suggestive of MPD.

PSG studies in these 3 patients (Cases 6-8) did not document any of their characteristic behavioral spells. EEG seizure activity was never detected, nor were there abnormalities of NREM/REM sleep, such as excessive arousals or complex behaviors which usually are present in NT/SW (Schenck, et al., 1989). Their nighttime experiences were thus considered to represent wakeful nocturnal extensions of their diurnal DDs.

DISCUSSION

Differential Diagnoses

This report establishes that DDs in some patients can present frequently or even exclusively during the sleep period and display a wide variety of injurious, dangerous, bizarre and very elaborate behaviors — when the brain is in a wakeful state by EEG criteria. Therefore, sleep-related DDs can be diagnosed objectively by means of extensive PSG study and audiovisual monitoring. Since treatment should be based on diagnostic precision, PSG study must be considered a cornerstone in the evaluation of nocturnal injury and wandering disorders.

The main differential diagnoses include:

A) Nocturnal seizures (generalized, complex partial), which can emerge from any sleep stage (Popoviciu & Szabo, 1971; Serman, Shouse, & Passouant, 1982; Montplaisir, Laverdiere, & Saint-Hilaire, 1985). Hypnogenic Paroxysmal Dystonia is thought to be an epileptic variant disorder — but with normal scalp EEG recordings — characterized by violent movements during NREM sleep which are controlled with carbamazepine (Lugaresi & Cirignotta, 1981). In addition, there are reports of an episodic nocturnal wandering disorder, with a propensity for injury, which is responsive to anti-convulsant medications, but which usually has a normal EEG (Pedley & Guilleminault, 1977; Maselli, Rosenberg & Spire, 1988).

B) NT and SW occur during abrupt partial arousals from NREM sleep, may involve elaborate, complex behavior (as extensive as leaving the house and driving an automobile long distances), and can be associated with subsequent amnesia, but also with simple or elaborate dream recall (Jacobson, Kales, Lehmanne, & Zweizig, 1965; Kales, Jacobson, Paulson, Kales, & Walter, 1966; Jacobson & Kales, 1967; Broughton, 1968; Fisher, Kahn, Edwards, & Davis, 1974; Schenck et al, 1989). In our PSG study of 100 adults with sleep-related injury, 54 percent were found to have NT/SW (Schenck et al, 1989), as were 56.7 percent (N=85) of our expanded series of 150 patients. Treatment with benzodiazepines (particularly clonazepam) and/or hypnosis is usually effective (Schenck et al., 1989; Hurwitz, Mahowald, Schenck, Schluter, & Bundlie, in press).

C) The REM Sleep Behavior Disorder (RBD) is characterized by excessive loss of the customary paralysis of REM sleep ("REM atonia"), with emergence of complex and frequently violent behaviors which often are attempted dream enactments (Schenck, Bundlie, Ettinger, & Mahowald, 1986; Schenck, Bundlie, Patterson, & Mahowald, 1987; Mahowald & Schenck, 1989). Although predominantly affecting older males, RBD can occur in young adult males and females and also in children, either alone or in combination with NT/SW and/or Narcolepsy (Schenck et al, 1989; Schenck, Bundlie, & Mahowald, 1989). Our PSG study of 100 adults with nocturnal injury documented RBD in 36 percent of cases (Schenck et al; 1989), and in 32.7 percent (N=49) of the expanded series of 150 cases. The problematic behaviors and disturbing, violent dreams are generally well-controlled with bedtime clonazepam therapy (Schenck, Bundlie, Ettinger, & Mahowald, 1986; Schenck, Bundlie, Patterson, & Mahowald, 1987; Mahowald, & Schenck, 1989; Schenck et al., 1989).

D) Dream Interruption Insomnia, as documented by PSG studies, involves repeated awakenings from most REM periods, a duration of at least 2-3 minutes, frequent recall of unpleasant dreams, and unremarkable NREM sleep (Greenberg, 1967; Fredrickson & Krueger, 1989).

All the major conditions listed above in the differential diagnoses (with the exception of the two presumed epileptic-variant disorders already described) have specific, PSG-defined diagnostic criteria which can be utilized in identifying the cause of abnormal, sleep-related behaviors. In addition, the fact that clonazepam was either ineffective or exacerbated the sleep-related spells in Cases 1, 2 and 4 may be a feature discriminating nocturnal DDs from NT/SW and RBD, which generally are very responsive to clonazepam treatment. However, this matter requires further elucidation.

It must be emphasized that the cause of injurious nocturnal behaviors cannot be determined solely from the clinical characteristics of the abnormal behaviors, the time of night in which they appear, nor the degree of subsequent recall for these behaviors and any associated dream-like mentation. These features may be present in any of the conditions described above and hence lack firm discriminatory power. Therefore, PSG study is essential: in 50 percent (4/8) of the cases reported herein, positive PSG criteria

(complex behaviors arising from sustained EEG wakefulness) established the diagnosis, whereas in the other 50 percent (4/8) of the cases the absence of a) NREM/REM sleep abnormalities, b) EEG epileptiform activity, and c) sleep-related breathing disturbance strongly suggested the diagnosis of nocturnal DD when correlated with the clinical history of daytime DDs.

Nocturnal Dissociative Disorders

Case 1, to our knowledge, is the first PSG-documented case of either exclusively nocturnal or of animalistic DD. Although the diagnosis should be classified as DDNOS, we believe that a strong argument can be made to support the diagnosis of animalistic MPD when the DSM-III-R criteria for MPD are carefully reviewed (the italics below are our own emphasis):

A. The existence within the person of two or more distinct personalities or personality states (each with *its own* relatively enduring *pattern of perceiving, relating to, and thinking about the environment and self*).

B. At least two of these personalities or personality states *recurrently take full control of the person's behavior*.

The videotaped behaviors in Case 1 clearly indicate that during these lengthy episodes the behaviors are exclusively animalistic, which would seem to indicate that the mode of perceiving and interacting with the environment is also animalistic. Although there was no direct recall for the behaviors generated by the alter "jungle cat" personality, the almost invariable recall of the stereotypical jungle cat "dream" the morning after a nocturnal dissociative episode — and the lack of such "dream" recall after an unremarkable night — suggest that there existed a recurrent nocturnal personality state, with a high degree of isomorphism between the documented animalistic behaviors and the animalistic "dream." Although the animal mentation may have shared the same onset and offset as the animalistic behaviors during the night (and thereby be considered wakeful mentation), it is also possible that the mentation was dissociated in time either partially or completely from the behaviors, and either be wakeful mentation, but without concurrent behavior, or else be actual dream mentation with REM or within NREM sleep.

The mechanisms underlying an exclusively nocturnal or an exclusively animalistic DD are unknown. However, the "Four Factor Theory" proposed by Kluft for understanding the etiology of MPD (Kluft, 1988) may provide a useful framework for gaining insights into these unique dissociative phenomena: 1) Biological factors: (i) the physical capacity to transform into a jungle cat to a seemingly impossible degree; (ii) particular vulnerability within the nocturnal sleep cycle for sustained EEG wakefulness culminating in elaborate dissociative behaviors. 2) Life events: since a psycho-social history prior to adoption at 10 months was unavailable, early childhood abuse cannot be excluded. 3) Individual factors predisposing to DD: insufficient information currently available. 4) Inability to recover from prior trauma: the severe separation anxiety attacks (undiminished at the age of 19), with regressive behavior observed by his sister whenever the mother slept away from home at

night, can raise the suspicion of prior, severe, emotional trauma with residual symptoms.

To our knowledge, extensive reviews (Putnam, Guroff, Silberman, Parban, & Post, 1986; Putnam, 1989), and also single case reports on MPD have not presented a case of exclusively nocturnal MPD. A possible exception was described in regards to St. Augustine's *Confessions*: "Considering the change that had taken place in him since his conversion, Augustine remarked that his old pagan personality, of which nothing seemed to remain in his waking state, still must exist since it was revived at night and in his dreams" (Ellenberger, 1970, p. 126). In a case of MPD, sleep served as an apparent transition point or switch process from one personality to another personality which then was completely dominant for 5 weeks before reverting to the original personality — after another night of sleep (Greaves, 1980). Another case of MPD involved two personalities which communicated through nocturnal "dreams." However, neither personality was exclusively nocturnal, nor was there any abnormal, sleep-related behavior (Salley, 1988).

Case 2 illustrates the rapid progression of nocturnal and diurnal DDs which emerged in the context of adult PTSD, and which reactivated childhood-onset, incest-induced PTSD. There is insufficient clinical data to determine whether MPD originated in childhood, although this would be expected, given the usual age and mode of onset of MPD, as identified by contemporary research (Kluft, 1988; Putnam, 1989). MPD is currently understood to comprise a chronic, multi-faceted posttraumatic psychopathological disorder originating in childhood from repeated emotional, physical and/or sexual abuse (Kluft, 1988; Putnam, 1989). Recently, 4 cases were reported of MPD emerging in adulthood as a pathological grief reaction: each patient "was an established and successful adult, who had a precipitous decline in his or her level of functioning following the death of a parent" (Putnam, 1989, p. 101). In each case the deceased parent was discovered, in psychotherapy, to have been the primary abuser of the patient during childhood. Therefore, the apparent onset and the determinants of MPD in Case 2 are virtually identical to that of the four cases just described.

The finding that patients 3-8 had suffered from PTSD stemming from severe trauma early in life and that intrusive memories of these experiences triggered recurrences of diurnal/nocturnal psychogenic amnesic spells with self-mutilation is very suggestive of MPD (Kluft, 1988; Putnam, 1989). In fact, any form of recurrent psychogenic amnesic spells is probably a manifestation of MPD (Kluft, 1988). Psychogenic Amnesia as a formal entity generally manifests as a single episode, emerging during a time of major stress (Kluft, 1988; American Psychiatric Association, 1987). Administration of a structured interview for DDs probably would have uncovered MPD in cases 3-8, in addition to Case 4 who had clinically documented MPD. The prominent nocturnal expression of their DDs should be noted, and probably reflects a long history of nocturnal bed-related and sleep-related abuse linked with a biological vulnerability for wakeful dissociation during sleep cycling. The female preponderance reported herein (7/8 cases) is congruent with research findings on MPD, indicating that over 80 percent of

cases are female (Kluft, 1988).

A recent PSG study, reported in an abstract, confirmed that "sleepwalking" episodes in a 40 year old nurse originated during EEG wakefulness (Fleming, 1987). In the sleep lab the subject left her bed and acted in a semi-purposeful manner. When the episode ended, she reported a headache identical to the one she experienced 19 months previously after being sexually assaulted. For 6 months prior to the PSG study, she had similar "sleepwalking" episodes occurring 1-2 hours after sleep onset. She had frequent episodes of DD with amnesia during psychotherapy sessions, which suggests the diagnosis of MPD.

The rapid change of state characteristic of parasomnias (NT, SW, RBD) — in which behaviors emerge abruptly both with partial arousals from NREM sleep and with dreaming behaviors during REM sleep — is also characteristic of MPD and other DDs (Putnam, 1989). Polysomnography is able to document the typically abrupt parasomnia activations involving i) dissociated sleep-wake transitional phenomena (e.g. automatic or semi-automatic behavior occurring with either coexisting or rapidly oscillating EEG sleep and wakefulness), or ii) dissociated sleep phenomena (e.g. muscle tone and complex, vigorous behaviors without tachycardia appearing during REM sleep). A broad spectrum of medical-neurological-psychiatric conditions exists which can result in dissociated sleep-wake phenomena (Mahowald & Schenck, 1989; Mahowald, Bundlie, & Schenck, 1989; Mahowald and Ettinger, 1990; Mahowald, Hurwitz, Bundlie, & Schenck, in press). Similarly, MPD usually manifests rapid switching from one personality to another, and a variety of factors impinging on the switch process have been identified (Putnam, 1989). In sum, the various disorders found to result in sleep-related injury cannot be distinguished on the basis of rapid onset since they all share this feature.

The term "sommambulism" has been used with reference to various altered states. In one schema, a distinction is made between "nondissociative somnambulism" (i.e. traditional SW arising from NREM sleep as a physiologic disorder without meaningful psychologic input) and "dissociative somnambulism" which has traumatic psychodynamic determinants (Nemiah, 1989). The latter condition can now be stated, based on our data, to emerge from nocturnal EEG wakefulness. We urge that "sommambulism" be used in strict adherence to the established nosology (Roffwarg, 1979), referring to SW (i.e. complex behaviors clearly arising out of NREM sleep), so as to avoid confusion with a nocturnal DD emerging from EEG wakefulness. Nevertheless, we concur with the postulation that both SW and DD share some common dissociative features (Nemiah, 1989).

Two recent anecdotal cases without PSG correlates involved patients with MPD who initially sought treatment for nocturnal wanderings which were "extraordinarily complex" and which eventually were found to be "caused by the nocturnal emergence of alters, primarily child alters, who were abreacting childhood trauma or acting out forbidden impulses" (Putnam, 1989, p. 19).

Our PSG data warrant the addition of MPD and DDNOS to the list of differential diagnoses for SW/NT in the official nosology (Roffwarg, 1979), which is currently being up-

dated, and also justify the inclusion of formal SW/NT in the differential diagnosis of DDs. ■

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