A Retrospective Study of Sleepwalking in 22 Patients: Clinical and Polysomnographic Findings

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We evaluated the recordings of 22 patients between 11 and 42 years of age who presented with the complaint of sleepwalking (SW) accompanied by other nocturnal behaviors ranging from mumbling, talking or screaming to more complex automatisms. None of the patients had a history of epileptic seizures. All patients had a history of another parasomnia; seven (33 %) reported family members with a history of parasomnia. More than one third of the patients started sleepwalking after age 10 years and almost two thirds had been sleepwalking for at least 15 years. The Minnesota Multiphasic Personality Inventory (MMPI) indicated psychopathology in nine patients; but there was usually no close association between SW and any psychopathology, and the treatment of one condition did not improve the other condition. In 12 patients, all-night polysomnographic investigations showed epileptiform abnormalities mostly recorded from temporal areas; four of them also had abnormal clinical EEGs. None of the parasomnia episodes in the sleep laboratory was associated with abnormal EEG activity. Anticonvulsant therapy reduced or completely eliminated the episodes in 11 patients with abnormal EEGs. Most of the patients in this study were either adolescents or young adults, but they exhibited the clinical characteristics of both classic (childhood) and adulthood somnambulism. There were also similarities with "episodic nocturnal wanderings" first described by Pedley and Guilleminault in 1977. (Sleep and Hypnosis 2000;5:255-262)

Key words: parasomnia, sleepwalking, episodic nocturnal wanderings, abnormal EEG, epilepsy, anti-convulsant therapy, polysomnography

INTRODUCTION

Sleepwalking (somnambulism) is described as "a series of complex behaviors that are initiated during slow-wave sleep and result in walking during sleep" (1). Sleepwalking (SW) in childhood is benign and usually self-limited, with recurrent episodes spontaneously disappearing by the end of adolescence (2). The persistence of SW into adulthood or its onset after adolescence was believed to be very rare (2-4), but Hublin et al. reported that up to 10% of adults had active SW histories in adulthood, indicating that SW in adults is not rare (5). In another study, one third of 54 adult SW patients had the onset of SW after age 16 years (6). SW is considered as a disorder of arousal from NREM sleep, usually slow-wave sleep, although there is a considerable amount of similarity with the clinical presentations of epilepsy (7,8). On the other hand, in 1977 Pedley and Guilleminault reported six adult patients with episodic nocturnal wanderings (ENW) characterized by violent behavior; screaming or incoherent speech; and ambulation (9). During all-night recordings, two of them exhibited abortive attacks which occurred during stage 2 NREM sleep and were not associated with abnormal EEG findings. Four patients presented epileptiform discharges on EEGs, and all patients responded

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Presented at the "Second Congress of The Asian Sleep Research Society", August 25-28, 1997; Jerusalem, Israel.

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Accepted May 21, 2000

to anticonvulsant therapy. The authors differentiated these patients from classic sleepwalkers and classified them into a subgroup of atypical epilepsy. Similar cases were later reported by another group (10).

In this retrospective study, we summarized the clinical characteristics and EEG findings of 22 patients with SW and compared them both with the subgroup described by Pedley and Guilleminault as well as classic sleepwalkers. Our purpose was to obtain additional clues about the etiology, clinical features and therapeutic strategies of SW.

METHODS

The recordings of 22 consecutive patients (16 males and 6 females), who presented to the sleep laboratory (Sleep Disorders Center, Baylor College of Medicine) between 1992 and 1995 with the complaint of SW, were retrospectively reviewed for this study. The mean age was 27.9 years (range 11 to 42 years). Information about their nocturnal behaviors was obtained from patients and their relatives, spouses or roommates. All patients received general physical and neurological examinations which were within normal limits. They also completed comprehensive sleep questionnaires and except for the 11 year old patient, the Minnesota Multiphasic Personality Inventory (MMPI). The MMPIs were interpreted by one psychologist who was blind to the clinical group of sleep disordered patients. None of the patients had a history of epileptic seizures or trauma prior to the onset of nocturnal episodes, with the exception of one patient who had several febrile convulsions during early childhood. Six patients reported a remote history of alcohol and/or drug abuse; clinical symptoms of parasomnia, however, had begun many years prior to alcohol and/or drug abuse in five of these patients. The remaining patient had first developed his episodes after the age of 30 years, but he denied any temporal relationship between his drug abuse and the appearance of nocturnal episodes. None of the patients was taking psychotropic or hypnotic medication for a minimum six week period prior to their sleep study.

All patients had at least two consecutive all-night polysomnographic (PSG) recordings, with four patients returning later for a third night session; two of these four patients were sleep deprived for 24 hours prior to the third night recording session. Clinical EEG recordings were performed on 21 patients on the second evaluation night just before bedtime, including photic stimulation and 3-minute hyperventilation (HV). Along with EEG activity (C3-A2, O1-A2); eye movements, leg movements, chin EMG, EKG and respiration were recorded on the first evaluation night. The paper speed was 10 mm/sec on the first night and 15 mm/sec on the second- and third night. The scoring was performed according to the criteria of Rechtschaffen and Kales (11). The technician was able to watch, hear and videotape any movements or sounds the patient made throughout the night with the assistance of an intercom and continuous video monitoring system. The number of EEG channels were increased to eight on the second night (A1-T3, T3-C3, C3-Cz, Cz-C4, C4-T4, T4-A2, Fz-Cz, Cz-Pz).

RESULTS

All patients had a history of another parasomnia including sleeptalking, enuresis nocturna, night terrors, nightmares, confusional arousal and bruxism. Fifteen patients (68%) reported a history of multiple symptoms of parasomnia. Of 21 patients, seven (33%) reported at least one family member with a history of parasomnia. One patient was unable to provide sufficient information about his birth and family history, since he was adopted. Two patients had a history of premature birth; another one reported a history of speech therapy during childhood. Two patients had relatives with neurological diseases (Friedreich's ataxia and multiple sclerosis). More than one third of the patients (N=8; 36%) had an age of onset after the age of 10, while almost two third of the patients (N=14; 63.6%) had been sleepwalking for a minimum of 15 years. Insufficient sleep (n=5), increased stress (n=5), fever (n=1) and sleeping in strange places (n=1) were emphasized by patients as aggravating factors for nocturnal episodes (Table 1).

Characteristics of the Attacks

Although all patients reported that they were still sleepwalking at variable intervals, they also described a large spectrum of behaviors ranging from mumbling, talking or screaming to more complex automatisms such as crawling, chewing-like chin movements, scratching, tossing around or sitting in the bed with a blank stare, etc.; according to the observations of their family members or bedpartners. One patient (# 16) for example, had his episodes since childhood. As a child, he would often wake up in another room or find himself sitting up in bed or wake up unable to scream and out of breath; but his complaints had only recently begun to cause problems in his relationship with his girlfriend. He often kicked her out of bed and had aggressive behavior during these episodes. Although his eyes were open when he stood up or walked around, he was unresponsive to stimulation and unable to recall these episodes the following morning.

Another patient (# 14) described his symptoms as sitting up in bed or screaming. These episodes lasted about 30 seconds. SW episodes were relatively rare. If he walked or got out of his house, he would remember

Table 1: Demographic data and clinical characteristics

Pts	Sex	Age	Family history of parasomnia	Patient s history	History of other parasomnia	Aggravating factors	Age of onset	Duration (years)	Frequency	Abnormal Sleep EEG	Abnormal clinical EEG	SW in lab
1	M	19	+	-	+	-	10	9	3-4/week	-	-	++
2	M	24	-	-	+	-	childhood	20	2-3/week	-	-	- -
3	M	24	-	-	+	-	childhood	>15	1-2/week	-	-	- -+
4	F	27	+	-	+	-	6	21	2-7/night	-	-	++
5	M	31	+	-	+	-	childhood	>25	every night	-	-	- -
6	F	35	-	speech therapy	+	stress	childhood	>20	2-3/month	-	-	- -
7	M	37	-	-	+	stress	12	25	1/month	-	-	- -
8	F	37	-	febrile convulsion	+	-	childhood	>30	3-4/week	-	-	- -+
9	M	38	-	-	+	-	childhood	>30	variable	-	-	++
10	F	42	-	-	+	-	41	1	every night	-	-	- - -*
11	M	11	+	-	+	fever	10	1	2-3/month	+	+	- -
12	M	16	+	-	+	insufficient sleep	11-12	3-4	1-2/year	+	-	- -
13	M	17	adopted	?	+	insufficient sleep	3	14	1/month	+	-	- -
14	M	20	-	-	+	insufficient sleep, strange places	18	2	2/month	+	-	- +
15	M	21	+	premature birth	+	stress	5	16	every night	+	+	++
16	M	26	-	-	+	-	childhood	>20	variable	+	+	- -
17	M	28	-	-	+	-	childhood	>20	1-2/week	+	-	- - -
18	F	28	-	insomnia	+	insufficient sleep,stress	childhood	>20	4-6/night	+	-	- -
19	M	29	-	-	+	stress	16-17	12-13	2-3/night	+	-	++
20	F	31	+	premature birth	+	-	childhood	>25	1-2/month	+	-	- -
21	M	32	-	-	+	-	childhood	>25	1/2-3years	+	+	- -
22	M	42	-	-	+	insufficient sleep	after 35	<5	2-3/month	+	no clinical EEG	- -

Patients 11-22: Abnormal sleep EEG group

* No sleepwalking but other behaviors on the 3rd night

it like a dream the following morning. He claimed that he was usually awake during the episode and knew what he was doing, but he felt threatened and thought the episode was real. There were also occasions when he did not get up, but sat in bed and opened his eyes with a blank stare; he was unable to recall these episodes.

Patient 3 presented for evaluation in the laboratory because he had recently broken a window during one of his episodes and sustained a severe injury to his arm. He might also cry, scream, fight with imaginary objects or beat his wife. He was unable to wake up on his own. If awakened, he usually could not recall what happened.

In spite of the rich symptomatology and even violent behaviors, injuries were reported in only six patients. Three patients had minor injuries such as lacerations on the hands or knees. In the remaining three patients, however, nocturnal episodes caused severe injuries. Patient 1 fell on his head during a SW episode at the age of 10, was knocked unconscious for 2-3 minutes, and had to get stitches on his eye. Patient 3 sustained a severe injury to his arm as mentioned before. Patient 17 sustained a broken shoulder during one of his episodes.

Patients generally estimated that their episodes ranged from 30 seconds up to 20-30 minutes. During the SW episodes in the sleep laboratory, patients were awakened by the technician when they tried to break or peel off the electrodes, or they woke up on their own since the connections did not allow them to walk away. Because of this interruption, we were unable to collect sufficient data concerning the duration of the episodes. One patient, however, was able to return to sleep after a 30-second episode in which he just sat up in the bed and acted out.

The behavioral patterns the patients exhibited during their episodes in the sleep laboratory were similar to those described above. Eight patients sleepwalked or attempted to stand up during their PSG evaluations. Five of them had SW episodes on both evaluation nights. This finding was consistent with the information given by patients concerning the frequency of SW episodes. On the other hand, patient 18 did not exhibit any SW attempt on either night of her PSG evaluation, even though she had previously reported 4-6 episodes per night. Patient 14 exhibited SW only on the second night of the evaluation. Two patients sleepwalked during the third night of their evaluation; one of these subjects was sleep deprived. Another patient, however, still did not have any episode even after 24-hr sleep deprivation on night three.

Thirteen patients exhibited other behaviors (with- or without SW) in the sleep laboratory such as talking, screaming, mumbling, chewing, scratching, turning over, sitting in bed and looking around in a confused manner, etc. Bruxism was also noted in two patients. A striking finding was that all patients who sleepwalked or exhibited other behaviors during their sleep study, had two or more episodes per night. All episodes occurred during stage 4 NREM sleep except for one patient who screamed once during stage 2 sleep. Another patient had periods of bruxism during all stages throughout the night. The episodes generally occurred in the first 2.5 hr after sleep onset; but we also observed some episodes in the latter parts of the night. Patient 1, for example, had four episodes during his first study night. His third and fourth episodes occurred at 277th and 397th minute following sleep onset, respectively; but they again were initiated from stage 4 sleep.

EEG Findings

All-night PSG recordings revealed abnormal EEGs in 12 patients. Spikes and sharp waves were recorded from temporal regions in 11 patients. Generalized high

Table 2: Abnormal EEG findings in 12 patients.

Patients	Age	All-night EEG	Clinical EEG	MRI
1	11	left temporal spikes	left temporal spikes (photic stimulation)	Normal
2	16	bilateral temporal spikes and sharp waves	normal	-
3	17	bilateral temporal spikes	normal	slight lateral ventricular prominence (min. atrophy)
4	20	right temporal spikes and sharp waves	normal	-
5	21	right temporal spikes	right temporal spikes	-
6	26	left temporal sharp waves	left temporal sharp waves	Normal
7	28	bilateral temporal sharp waves along with occasional paroxysmal discharges	normal	-
8	28	bilateral temporal spikes	normal	-
9	29	bilateral temporal spikes and sharp waves	normal	Normal
10	31	right temporal spikes and sharp waves	normal	Normal
11	32	bilateral centro-temporal spikes	bilateral centro-temporal spikes (after HV)	-
12	42	generalized high-amplitude sharp and slow waves	no clinical EEG	-

Table 3: Clinical characteristics of the patients with and without abnormal sleep EEG

	Abnormal Sleep EEG		Normal Sleep Sleep EEG			
	n = 12	%	n = 10	%	Total (22)	%
Mean of age	25.08		31.4		27.95	
Abnormal clinical EEG*	4	36.3	-	0	4	19
Sleepwalking in lab	3	25	5	50	8	36
Other behaviors in lab	7	58.3	6	60	13	59
History of other parasomnia	12	100	10	100	22	100
History of multiple parasomnia	8	66.7	7	70	15	68
Family history**	4	36	3	30	7	33
Age of onset>10	5	41.6	3	30	8	36
Duration>15yrs.	6	50	8	80	14	63.6
Aggravating factors	8	66.7	2	20	10	45
Psychopathology (MMPI)	5	41.6	4	40	9	41
Injury	3	25	3	30	6	27
Anticonvulsant therapy	11	91.6	1	10	12	54.5

* Clinical EEG was performed in 11 patients
** Adopted patient excluded

amplitude sharp and sharp-slow activity was noted in one patient. These EEG abnormalities were observed in all NREM sleep stages, but were more prominent during stages 1 and 2. They also persisted throughout REM sleep in two patients. Four out of 12 patients also had abnormal clinical EEGs (Table 2,3).

None of the episodes was associated with abnormal EEG activity. Cranial MRI investigations in five patients did not reveal any pathological finding which could explain the abnormal EEG patterns.

Psychopathological Findings

Three patients had been previously diagnosed with depression or panic disorder. Psychopathological findings indicated through the MMPI were as follows:

Normal EEG group: Patient 2 had previously been diagnosed as having "depression with suicidal ideation". The MMPI confirmed depression, panic state and paranoid ideation. Patient 6 had been previously diagnosed as panic disorder. His MMPI indicated personality disorder (probably borderline or hysteroid) and emotional instability with depressed and anxious mood. Patient 8 was previously diagnosed with depression. Her MMPI showed depressed and irritable mood. Patient 9 had an MMPI which indicated mild depression with insecurity, possible interpersonal problems, sexual concerns and elevated anxiety.

Abnormal EEG group: Patient 16 had social withdrawal and isolation, elevated anxiety level, depressed mood and irritability with the possibility of dissociative episodes. Patient 17 had somatic concerns and sexual complaints suggesting dissatisfaction with his marital partner. Patient 18 showed conversion and somatic complements. Patient 19 had somatic concerns with inclination toward the development of conversion. Patient 22 had severe depression and indication of paranoid ideation.

Nevertheless, there was usually no close association between SW and any psychopathology, and the treatment of one condition did not improve the other condition. It should also be emphasized that no clinical psychiatric interviews were conducted, and therefore no clinical correlates of the MMPI findings could take place.

Treatment

Out of 12 patients with abnormal EEGs, 11 were administered antiepileptic drug (AED) therapy. Following a neurological consultation for each patient, carbamazepine was prescribed for 10 patients, while the remaining patient was given valproate. Dosage was adjusted according to clinical responses and serum concentrations. Seven patients, including the one taking valproate, reported either complete elimination or significant reduction of their nocturnal episodes during follow-up periods ranging from 12 to 36 months. We do not have current information on the remaining four patients since they were out of town. One patient in the abnormal EEG group, who also had periodic limb movements during sleep, was given clonazepam which significantly reduced the symptoms.

It was interesting that in one patient with normal EEG, who was given carbamazepine instead of benzodiazepine due to a drug abuse history in the past, the AED therapy controlled the symptoms during the observation period.

DISCUSSION

The pathophysiology underlying SW is not completely known yet. SW was believed to be related to epilepsy in the past but since Broughton s work in 1968,

SW has been considered as a disorder of arousal (7,8,12). It appears to be due to genetic, developmental, and scheduling problems in childhood (13,14), whereas psychopathology (particularly personality disorders and difficulties in handling aggression) has been found frequently in adult sleepwalkers (3,4,6). Sudden, paroxysmal high voltage delta bursts during slow wave sleep (so called "immaturity factor") which are commonly seen in sleepwalkers until the age of 16 years (15) suggest that developmental delay is an etiologic factor in SW. The high percentage of coexisting parasomnias (particularly enuresis) also supports this theory (4). It has been reported that adult sleepwalkers have more frequent events, more intense (violent) clinical behavior during the episodes, later age of onset, increased familial occurrence of SW, and more frequent self-injury (2,4). Although most of the events initiate from stage 3-4 NREM sleep, all NREM sleep stages may be involved in adults (2,6).

In our study group, we did not document the presence of any high amplitude delta bursts (immaturity factor) during all-night recordings. Similar to our observation, it was reported in a recent study (16) that "delta wave buildup" was detected in less than 2% of 252 nonbehavioral or behavioral arousals from slow-wave sleep in 38 adults with injurious SW and sleep terrors.

Although we do not have a control group, and half of the patients were unable to recall the exact age of onset, more than one third of our patients reported an age of onset after age 10, supporting Kales et al. study (4) which showed a later age of onset in adult sleepwalkers.

The patients or their families estimated that SW episodes could last 20-30 minutes. This is an important information because of its forensic utility. Dreaming during SW was another interesting point described by a patient.

Psychopathology

Nine patients (41%) in our study group had psychopathological MMPI profiles supporting the importance and high prevalence of psychopathology in adult sleepwalkers. Additionally, a female patient who had a normal MMPI profile, started SW after she was raped. Three patients with pathological MMPI findings had already been given a psychiatric diagnosis by other psychiatrists. Depressive mood, irritability, anxiety, conversion, somatic problems and paranoid ideation were remarkable psychological disturbances indicated by the MMPI. Since control MMPIs were not available, nor were normative data on the baseline population rate of psychopathology as detected by the MMPI, there is no reference context to determine whether this SW group had an elevated rate of abnormal MMPIs.

Psychopathology seemed to be an aggravating factor rather than an etiological factor in our patient group, as seven out of nine patients with pathological MMPI findings developed SW during childhood, and five patients also had EEG abnormalities. Similar to our findings, Schenck et al. (6) reported that 64% of the 36 adults with SW who completed an MMPI had an abnormal profile (usually depressive, impulsive and passive-aggressive traits). However, there was no association between any identified axis I psychiatric disorder with either the onset or progression of SW; treatment of any psychiatric disorder did not control the SW; bedtime clonazepam therapy promptly controlled the SW, in both patients with and without psychopathology; and nearly all SW patients maintained a high level of psychosocial functioning. Also, Crisp et al (17) did not detect psychopathology in a group of adult SW patients.

EEG Findings and Treatment

Considering the EEG findings (abnormal EEG activities mostly originated from the temporal regions in approximately 2/3 of patients), the clinical symptoms, and the response to AED therapy, our patient group is also very similar to Pedley and Guilleminault s group (9,10). The tendency to demonstrate two or more episodes per night observed in our patients during their all-night evaluations is another clinical characteristic in common. On the other hand, "the negative family history", "the occurrence of the episodes out of stage 2 NREM sleep and usually 4 hr or more after sleep onset", and "an age of onset between 15 and 20 years of age" (9) in the patients with ENW are the clinical features which are not consistent with our study results.

Comparing the clinical characteristics of the patients with abnormal EEG to those with normal EEG in our study, it appears that there is no clear difference between the groups (Table 3). They only seem to differ from each other in terms of "duration", "age of onset", and "aggravating factors". In the abnormal EEG group, 41.6% of the patients first developed their symptoms at the age of 10 years or later (30% in normal EEG group), whereas 80% of the patients in the normal EEG group had experienced continuous SW for at least 15 years (50% in abnormal EEG group). These findings suggest that the patients in the abnormal EEG group may seek medical help earlier because of the more dramatic symptomatology. Indeed, the lower mean age in the abnormal EEG group (25.1 vs 31.4) supports this possibility. In the normal EEG group, however, increased frequency of the episodes or changes in symptoms (e.g.,

violent behavior) after a long period of time from the onset may have forced these patients to consult a physician, since more frequent episodes (nightly or weekly) were reported by patients in the normal EEG group (Table 1).

More than 2/3 of the patients in the abnormal EEG group reported several aggravating factors (Table 3) which also have been known as provocative factors giving rise to epileptic seizures. In contrast, only 1/5 of the patients in the normal EEG group noticed a connection between "stress" and the occurrence of their episodes. These results suggest that the abnormal EEG group is more sensitive to external influences than the normal EEG group.

On the basis of the results and considerations mentioned above, it does not seem to be easy to make a clear distinction between the clinical presentations of classic somnambulism (during childhood), somnambulism in adults, and ENW. It appears that they may be representing variable clinical presentations of the same syndrome (2), depending on the "severity" of the underlying etiology. The etiology, however, still remains unclear.

Although a direct correlation between epileptiform discharges and nocturnal wanderings has been established and the coexistence of epileptic seizures and NREM parasomnia has been reported in some cases, these conditions are very rare (8). Nevertheless, our study provided several findings which indicate a relationship to epilepsy.

The episodes exhibited by our patients in the laboratory were not accompanied by epileptic EEG activity, even in patients who exhibited abnormal EEGs. It is well known that many epileptic patients have a normal interictal and occasionally normal ictal EEG using standard techniques (10,18,19). On the other hand, it has been showed by several studies that epileptic seizures, particularly those of frontal lobe origin, can mimic the clinical manifestations of parasomnias, and depth electrodes are usually needed to establish the correct diagnosis (20,21). Indeed, ENW and some parasomnias like nocturnal motor attacks or paroxysmal awakenings are now considered as different clinical forms of frontal lobe epilepsy (19,21). Since this is a retrospective study and the montage used during sleep was inadequate to screen the frontal lobe, we may have missed some epileptic discharges. Comprehensive and repetitive all-night evaluations along with depth electrodes, telemetric systems, activation methods (particularly sleep deprivation) and clinical EEG recordings have to be performed in the future in order to obtain sufficient data regarding EEG features of sleepwalkers.

Soldatos et al. (22) suggested that the daytime clinical EEG was of limited value in evaluating adult sleepwalkers. In our study, only four patients (all in abnormal EEG group) had abnormal clinical EEGs. Interestingly, two of them showed abnormalities in the form of spikes and sharp waves during photic stimulation or just after HV. We believe that the clinical EEG recordings accompanying all-night studies may provide further information, especially using activation methods and depth electrodes.

Two patients in the abnormal EEG group reported a history of premature birth (one of them also had severe icterus at birth, while the mother of the second patient had pregnancy complications) which may suggest the existence of a brain damage, possibly also responsible of the abnormal EEG activity. Another patient in the normal EEG group underwent speech therapy during childhood, possibly as a result of a "more benign lesion" which might have been overwhelmed by the maturation process and does not generate recordable abnormal EEG activity. Additionally, two patients in the abnormal EEG group reported relatives with neurological diseases. One of them had a cousin suffering from Friedreich's ataxia (FA), while the other patient had a sister who was diagnosed with multiple sclerosis (MS). Since FA and sometimes MS show familial patterns, it is possible that these patients may have some minor pathological changes which have not surfaced up to now.

Whatever the underlying pathology, it may cause bioelectrical and/or biochemical changes depending on its severity and localization in the brain which result in SW and/or various other nocturnal behaviors. For this reason, MRI, SPECT- and PET-scan investigations should also be performed in these patients which would be helpful in demonstrating the anatomical and/or physiopathological changes.

Although a positive response to anti-convulsant therapy does not necessarily indicate an underlying epileptic disorder, the efficacy of AED therapy in our abnormal EEG group provides additional support to the theory of epileptic origin. Furthermore, we do not know exactly what happens to classic sleepwalkers; for example, how many child sleepwalkers continue to sleepwalk as adults (8); how many of them have EEG abnormalities, or whether they will develop abnormal EEGs in the future; in other words, do they really constitute a different group?

It is well known that benzodiazepine (particularly clonazepam) therapy has been widely used for SW. Schenck et al. reported that immediate and sustained efficacy with nightly clonazepam therapy was observed in more than 80% of adult SW patients (6). They also reported another large series of adult SW patients who

responded to nightly clonazepam (and other benzodiazepine) therapy for years, with sustained efficacy (23). Nevertheless, given the fact that benzodiazepines used by sleepwalkers also have more or less anticonvulsant effect; and our observation about the efficacy of carbamazepine therapy in a patient with normal EEG, further placebo-controlled, double-blind long term follow-up studies are needed to clarify the therapeutic role of anticonvulsants in adult sleepwalkers with both typical SW and atypical/epileptic variant SW.

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