



Review

The Parasomnias and Sleep Related Movement Disorders—A Look Back at Six Decades of Scientific Studies

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Abstract: The objective of this article is to provide a comprehensive personal survey of all the major parasomnias with coverage of their clinical presentation, investigation, physiopathogenesis and treatment. These include the four major members of the slow-wave sleep arousal parasomnias which are enuresis nocturna (bedwetting), somnambulism (sleepwalking), sleep terrors (pavor nocturnus in children, incubus attacks in adults) and confusional arousals (sleep drunkenness). Other parasomnias covered are sleep-related aggression, hypnagogic and hypnopompic terrifying hallucinations, REM sleep terrifying dreams, nocturnal anxiety attacks, sleep paralysis, sleep talking (somniloquy), sexsomnia, REM sleep behavior disorder (RBD), nocturnal paroxysmal dystonia, sleep starts (hypnic jerks), jactatio capitis nocturna (head and total body rocking), periodic limb movement disorder (PLMs), hypnagogic foot tremor, restless leg syndrome (Ekbom syndrome), exploding head syndrome, excessive fragmentary myoclonus, nocturnal cramps, and sleep-related epileptic seizures. There is interest in the possibility of relationships between sleep/wake states and creativity.

Keywords: parasomnias; bedwetting; sleepwalking; sleep terrors; confusional arousals; nocturnal anxiety attacks; aggression; REM nightmares; REM sleep behavior disorder; restless leg syndrome; nocturnal paroxysmal dystonia; sleep starts; jactatio capitis nocturna; periodic limb movement disorder; nocturnal cramps; exploding head syndrome; excessive fragmentary myoclonus



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1. The Personnel and Technical Advances

In late 1960 or early 1961, Cesira Batini arrived in Marseille from Pisa where she had worked with Giuseppe Moruzzi. Batini was interested in sleep ever since her research in Pisa, where using the pretrigeminal preparation, she had discovered that sleep controlling centers existed in the brainstem caudal to the trigeminal nerve nucleus. Dr. Batini was able to interest Professor Henri Gastaut (Figure 1) in sleep and sleep disorders. Until then Gastaut's research had focused on epilepsy and clinical neurophysiology.

When I arrived at the end of June in 1962, Jack Rhodes from New Mexico, USA, was helping Batini in her research and several articles on normal sleep had already been published in English or French journals. These were the first published sleep studies by the Marseille group and were authored by Batini, Fressy and Gastaut [1,2]. Jacques Fressy was a psychiatrist in the French army who wrote an excellent doctoral “thèse du 3ème cycle” on polysomnographic techniques [3]. Almost all of the research in this thesis was performed at the Centre Saint Paul and the main features were either initiated or were improved in Marseille. They included:

1. A large number of channels ($N = 15$) on the two available Alvar EEG machines. Most other centers at the time were using only 8 or 10 channels.
2. Multi-channel EEG, EOG (horizontal and vertical), submental EMG, peripheral limb EMGs, ECG (rate and variability), respiration (thoracic and abdominal to detect obstructive and central apneas and hypopneas), electrodermogram (skin resistance or skin potentials), body movements by actigraphy, and the pipigram for the study of enuresis.

3. The use of very long wire electrodes which permitted sleepwalkers to get out of bed, walk a short distance, and return to bed.
4. Telemetry—A 4 channel Alvar system (montage: Cz-Oz and CZ-earlobe) and EOG (horizontal only). This was sufficient to differentiate and analyze NREM sleep, REM sleep and wakefulness. If the number of channels was reduced to 3, we could hear FM music on the unused channel!
5. Cinematography—there were large wall fixtures for cameras to film parasomnia events. The system required very bright floodlights in order to have high quality images.
6. A new system to classify different stages of somnolence, especially at sleep onset that was created by Professor Gastaut. It had substages 1A1, 1A2 and 1A3.

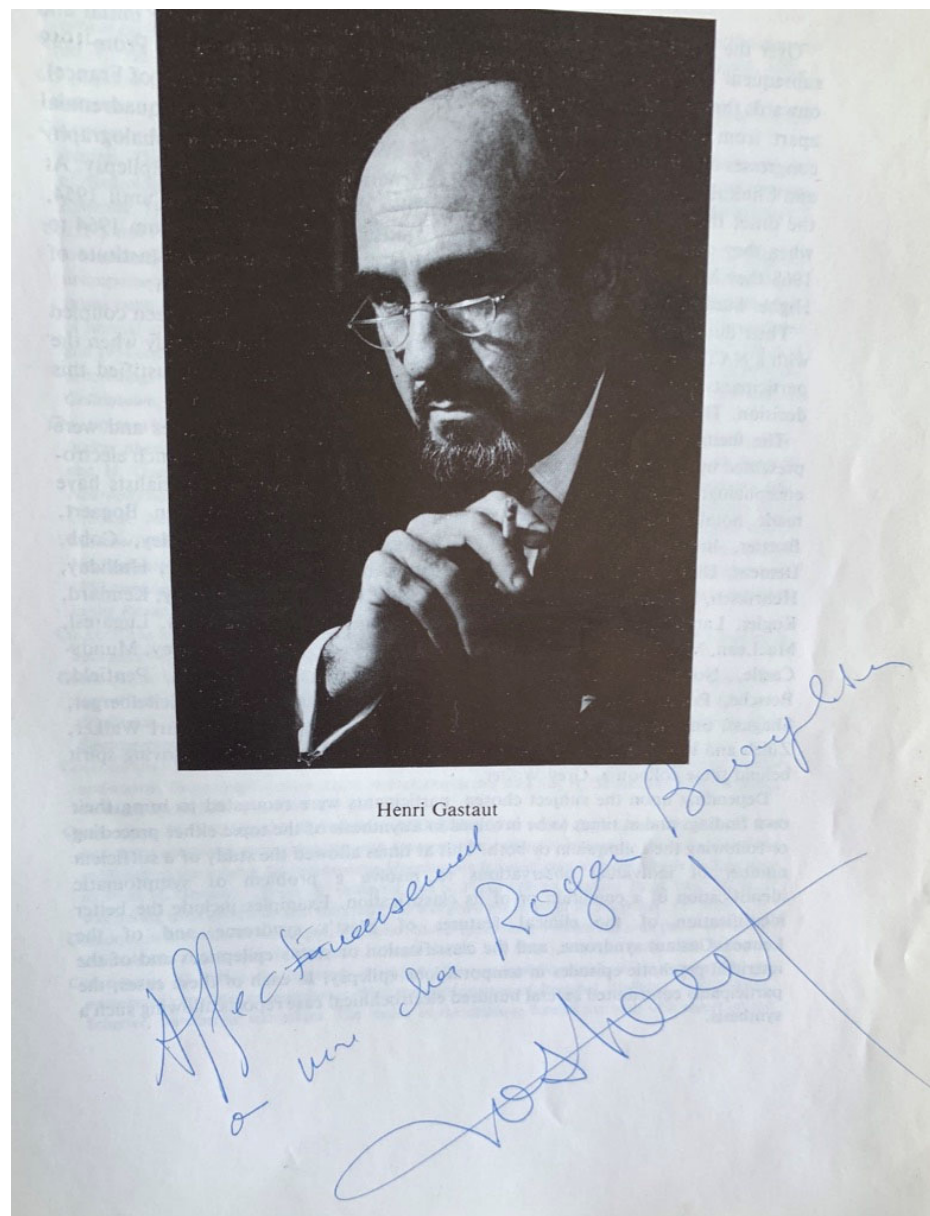


Figure 1. Professor Henri Gastaut (1915–1996) the director of the research program in the 1960s on sleep and sleep disorders which took place at the Centre Saint Paul in Marseille. The photograph signed by Gastaut states “to my dear Roger Broughton with affection, Henri Gastaut”.

2. Enuresis in Children

The worldwide prevalence of bedwetting in children up to 6 years of age has been estimated as 20–25%. It is a significant and often embarrassing medical and social problem. George Orwell in his book *Such, Such Were the Joys* [4] wrote: “I knew that bedwetting was (a) wicked and (b) outside of my control. The second fact I was personally aware of, and the first I did not question. It was possible, therefore, to commit a sin without knowing you committed it, without wanting to commit it, and without being able to avoid it”.

The earliest sleep studies of enuresis were published by Ditman and Blinn in 1955 and focused on the sleep levels associated with the onset of enuresis [5]. Jacques Saint-Laurent had arrived in Marseille from Quebec in the fall of 1961 and began the first study of enuresis in epileptic children which was co-authored with Batini, Broughton and Gastaut [6]. After Saint Laurent moved in the fall of 1962 to Paris in order to work with Pierre Buser, I became the leader of the research group and continued the research program on enuresis nocturna.

The issue of whether the enuretic episodes in epileptic children were related to seizures or not was important, as this would define future treatment and prognosis. We found that enuresis regularly occurred in slow-wave sleep (SWS), most frequently at the end of the first or second NREM/REM sleep cycle in the first third of the night [7]. This was somewhat of a surprise because at the time many believed that enuresis (and other NREM sleep parasomnias, such as sleepwalking and sleep terrors) were an expression of dreaming which was known to occur mainly in REM sleep. We also found that enuresis could be induced by forced arousal or other strong stimuli, such as loud noises in SWS [8]. It seemed evident that we could not be triggering bedwetting at the exact moment that dreaming or other mental activity, of which we were unaware, was about to cause an enuretic event. In late June 1962 Gastaut gave me instructions to record as many enuretic epileptic children as possible during his upcoming absence during the July and August summer holidays. I recorded 18 enuretic epileptic children and found that no bedwetting events were related to seizures or subclinical epileptic discharges.

To detect the precise moment of the onset of micturition a special bikini was invented by myself and manufactured by my wife Joan. It had two parallel bare electrode wires sown about 1 cm apart throughout the crotch area of the bikini (Figure 2). In tests it was found that only 2 or 3 drops of urine on the bikini were enough to cause electrical conduction between the wires and change the 50 Hz artifact in the recording channel into a fine line (Figure 3). This was progress, as all previously reported recordings had used sensors for bedwetting that were placed under the mattress and, consequently, the signal was very delayed. We called the recording channel the “pipigram” to align its terminology with that of the electroencephalogram, oculogram, and the other variables. At congresses, Professor Gastaut would on occasion twirl the bikini around a finger to get the attention of, and to amuse, the audience.

Using the bikini detector it was found that the enuretic episodes always began in stage 3 or 4 of slow wave sleep usually at the termination of either the first or second NREM/REM cycle [9]. This was so reliable that, if bedwetting did not occur with the partial arousal ending the first cycle, one could predict almost exactly when it would take place later. This allowed for a rest period to drink a coffee or smoke a cigarette.

We noted that there was a regular series of events that occurred with bedwetting. It consisted of a sequence of initial high voltage delta waves, subsequent lowering of the amplitude of the EEG, some body movements, and eventually an arousal (Figure 3). We termed the phenomenon the “enuretic process”. The latter was sometimes incomplete with the patients mumbling incomprehensively or even denying that urinary incontinence had occurred despite being in a very wet bed. The subjects typically had no recall later in the day of having wet their bed.

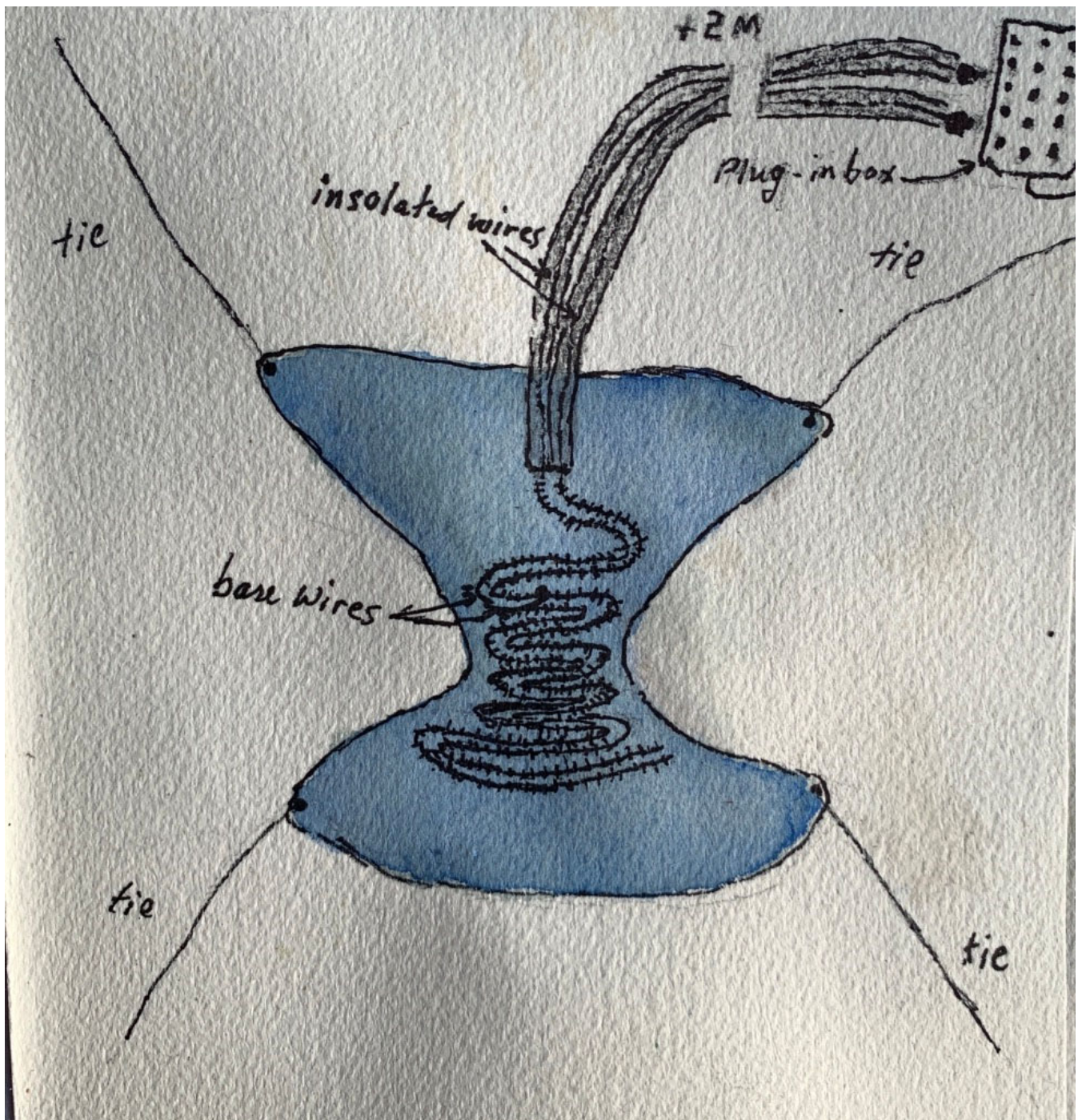


Figure 2. The bikini for the detection of the precise moment of the onset of enuresis was designed by Roger Broughton and manufactured by his wife Joan. Two electrode wires with insulation removed and with the bare wires were sewn close together in serpentine fashion in the crotch area of the bikini. A few drops of urine on the bikini caused a short-circuit between the wires and the 50 Hz mainline artifact become a fine line. The ties were tied just above the hips (unpublished personal photo of the author).

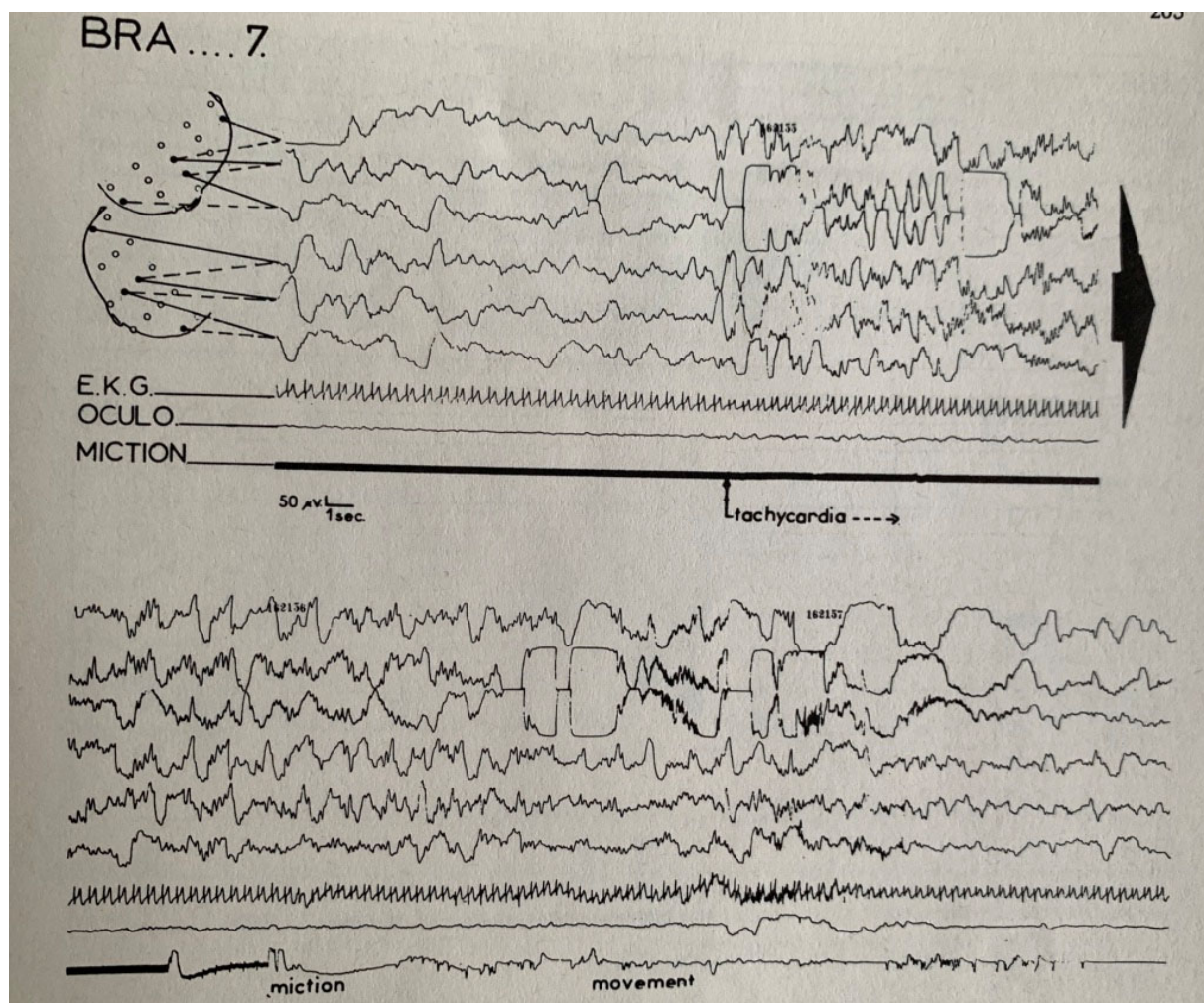


Figure 3. Polysomnographic recording of an enuretic episode in a 7-year-old girl. It begins in slow-wave sleep with a movement associated with an increased amplitude of the EEG slow waves and the onset of tachycardia. The channel “mition” shows the exact moment of onset of enuresis where the dark 50 Hz artifact becomes a thin line. There is further movement and the patient continues to be in NREM sleep (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

Others, such as Hawkins et al. [10], Ritvo et al. [11] and Gambi et al. [12] later confirmed the regular occurrence of bedwetting in slow wave sleep. However, Kales et al. [13] and Mikkelsen et al. [14] reported that enuresis took place in all sleep stages in proportion to the amount of sleep for each sleep stage. No later researchers would report this pattern.

When Professor Gastaut returned from his holidays, he went immediately to the lab to look over the polysomnographic recordings of the enuretic epileptic children. He confirmed that no bedwetting had been associated with epileptic phenomena. It was concluded that bedwetting in epileptics is almost always a case of co-existent idiopathic enuresis and is not related to a clinical seizure or a seizure discharge. Additionally noted was the finding that the frequency of bedwetting in the laboratory studies was less than in reports for the home environment.

An important issue concerning enuresis became: why do children and adults with bedwetting not sense the urge for micturition early enough, or strongly enough, to awaken and go to the bathroom in time? Gastaut decided that I should catheterize some children to obtain intra-vesicular recordings of bladder pressure. Control studies were performed

in age and sex matched normal subjects. It was found that enuretic subjects had many increases in bladder pressure from the baseline level. These were typically increases of 10–30 cm water pressure. Moreover, I found that bladder pressure in enuretic children was exquisitely sensitive to auditory stimuli. Just a brief snap of the fingers near the subject's head created an increase in pressure associated with a K-complex. This phenomenon did not occur in the control subjects [9]. During an enuretic episode, the intra-vesicular pressure would increase to over 80 cm H₂O and thereby exceeded the pressure at which an awake child was able to inhibit bedwetting (Figure 4). Other physiological parameters were found to be different in enuretic compared to normal control subjects. The enuretic children had higher heart rates in wakefulness and throughout sleep with some further increase just before the enuretic episode. This tachycardia then decreased abruptly immediately after bedwetting occurred. There was also an increase in the respiratory rate in enuretic subjects during slow wave sleep that also dropped after bedwetting (Figure 5).

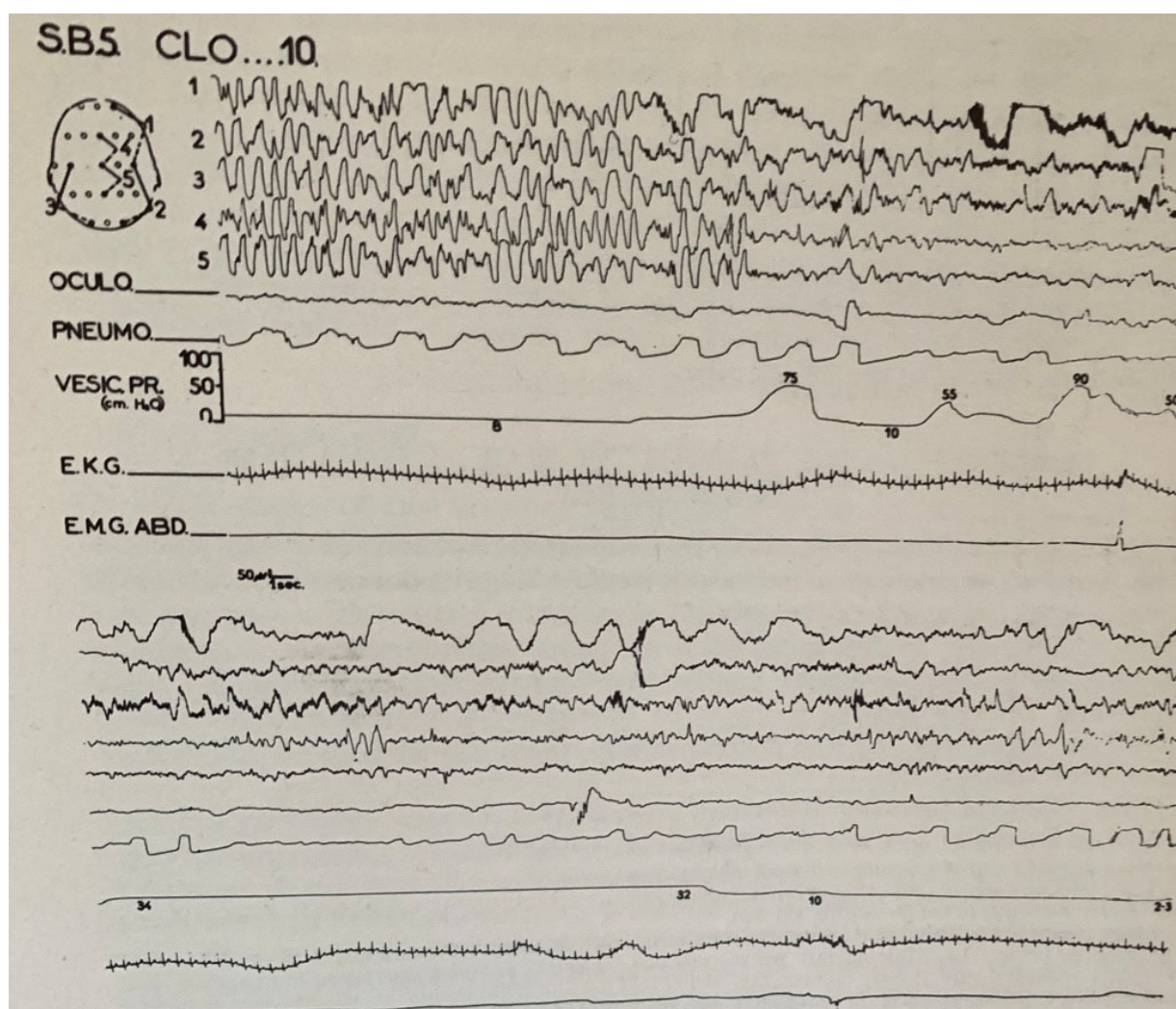


Figure 4. Enuretic episode with intra-vesicular pressure recording. The episode begins in slow-wave sleep and shows large waves of increased bladder pressure reaching a maximum of 90 cm water pressure. As the episode continues the urine flows out around the catheter and the intra-vesicular pressure plateaus around 30 cm pressure and then decreases to 2–3 cm pressure, at which time the bladder has fully emptied. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

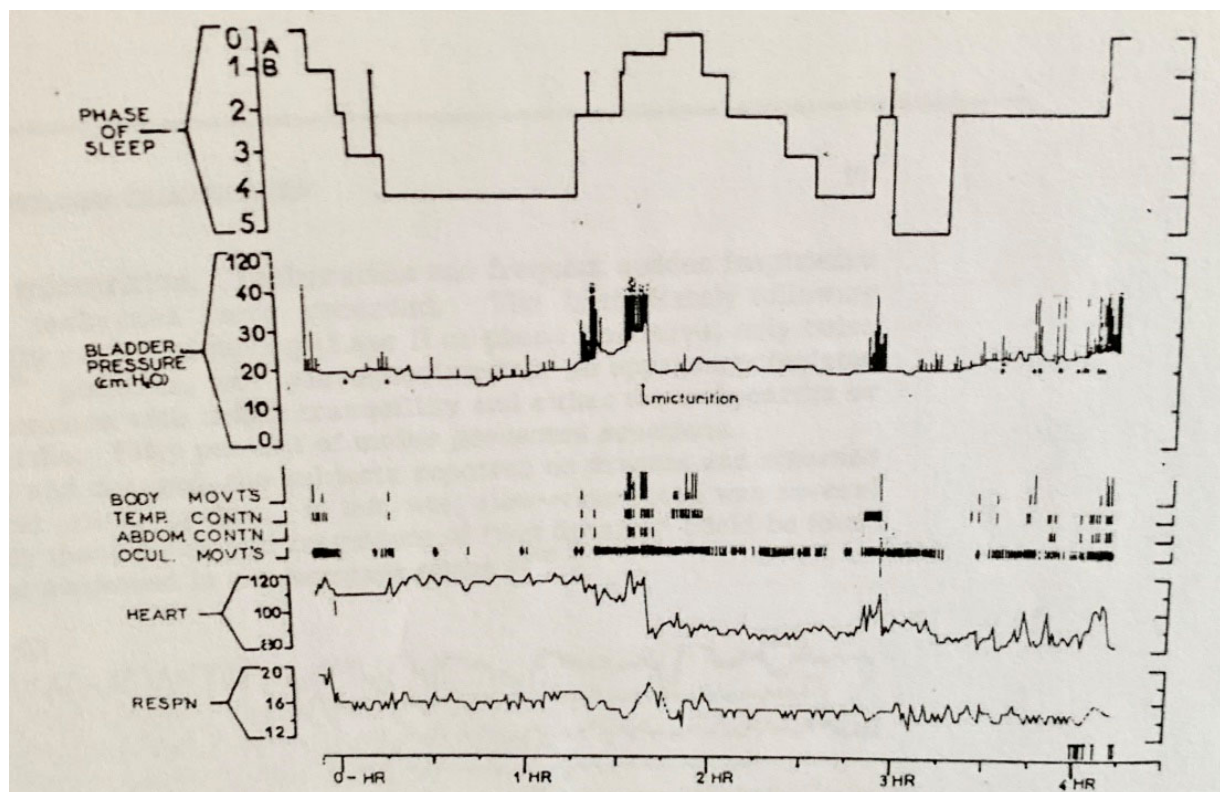


Figure 5. Histogram of an enuretic episode with intra-vesicular recording of bladder pressure. Sleep stage 5 is REM sleep. Before the micturition around the end of stage 4 there is an increase in the baseline bladder pressure with superimposed increases reaching 80 cm water pressure. When enuresis takes place there is a precipitous drop in bladder pressure followed by an absence of further contractions for about an hour. The high heart rate before enuresis drops abruptly when enuresis occurs. There is a similar but less dramatic decrease in respiratory rate when enuresis takes place (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

The causes of the increase in bladder activity in enuretic subjects without awakening in time to go to the bathroom have been explored. Early studies by Muellner [15] documented that enuretic children have smaller bladders than normal subjects. Therefore, their bladders both fill up more rapidly and are fuller than the bladders of normal subjects. Research shows that enuretics are in general very deep sleepers which, combined with their smaller bladders, increases the incidence of uncontrollable bedwetting.

This raised the possibility that enuretic subjects have a sensory defect causing them to not know when their bladders are full and ready to void. There is now strong evidence that this is true. Rapid injection of water through a catheter and into the bladder in enuretic subjects has been shown by Di Perri and Meduri [16,17], and also by Bradley [18], to induce arousals in normal subjects but not in enuretics. Such injections in enuretic children can trigger micturition around the catheter during continuous EEG patterns of sleep (Di Perri and Meduri [17]).

Studies have shown that enuresis nocturna tends to occur in families suggesting a genetic predisposition. Bakwin published a case of enuresis in monozygotic and same-sex dizygotic twins [19]. A large epidemiological study with a cohort of over 8000 cases by von Gontard et al. documented a strong family history of enuresis [20]. It is, therefore, evident that there is an inherited genetic predisposition that expresses itself in the physiopathogenesis of bedwetting.

The treatment of bedwetting has been improved by the use of conditioning approaches, such as associating bedwetting episodes with a bell or other loud sound, by the use of inducing very slow arousals, by psychotherapy to minimize shame for bedwetting, and by improved drug treatment using anti-cholinergic drugs and tricyclic medication, such as imipramine.

3. Sleepwalking

Sleepwalking (somnambulism) occurs in 29% of young children and affects some 4% of adults [21]. It typically consists of getting out of bed in the first third of the night, walking for a variable distance, sometimes with mumbling or frank sleep talking, and then returning to bed. The behavior is rather stereotyped. There is incomplete reactivity to external stimuli and the behavior seems to just run itself out. Tassinari et al. have proposed that sleepwalking (and frontal lobe seizures) characterize “innate” motor behaviors which express subcortical central pattern generators [22]. Later there is always more or less complete amnesia for the events. Sleepwalking may be associated with injury due to walking into walls, doors, or other objects. Subjects who are sleepwalking should never be awakened suddenly as they will be confused and may lash out at the person who has awakened them. Rather they should be gently walked back to bed.

The first polysomnographic recordings of sleepwalking episodes were reported by Gastaut in 1964 at a congress in Paris and were published by Gastaut et al. in 1965 [23]. Further details were provided in the Sakel Academic Lecture of the Society for Biological Psychiatry in May 1964 by Gastaut and Broughton [9]. Independently in 1965, the UCLA researchers presented confirmatory evidence that sleepwalking episodes took place during incomplete awakenings from deep slow wave sleep [24]. When he returned to Marseille, Gastaut reported that, while presenting our findings at the Sakel lecture in Los Angeles, Jacobson interrupted his talk by jumping up and saying “Sir, you cannot say that sleepwalking occurs in slow wave sleep. That is what we are finding!” The occurrence of bedwetting, sleepwalking, sleep terrors and confusional arousals in deep slow wave sleep and the ability to trigger them by forced arousals were detailed in a lead article published by Broughton in the journal *Science* in 1968 [25].

Although an early episode of sleepwalking was recorded during a transition from stage 2 into REM sleep by Gastaut and Broughton [26], this was never encountered subsequently when all episodes initiated in slow wave sleep. This occurred almost always at the end of the first or second NREM/REM sleep cycle confirming that almost all sleepwalking occurs in the first third of the night. Using both long electrode wires and telemetry it was found that during sleepwalking the EEG mainly showed stage 1 EEG patterns of predominantly theta sometimes intermixed with alpha activity (Figure 6). Occasionally there was a period of diffuse alpha activity that the Marseille group termed stage 1A3. Filming was performed during recordings of sleepwalking using both very long electrodes and telemetry. This required the use of strong floodlights for a good film image and, unexpectedly, it was found that the brilliant illumination did not block the alpha rhythm during sleepwalking as normally occurs during wakefulness. This was strong evidence of a deafferentation of the visual system [9]. The sleepwalking episode was often initiated by a brief period of relatively high amplitude delta activity before the arousal inherent in sleepwalking occurred. Montplaisir et al. were the first authors to document increased daytime sleepiness in sleepwalkers [27].

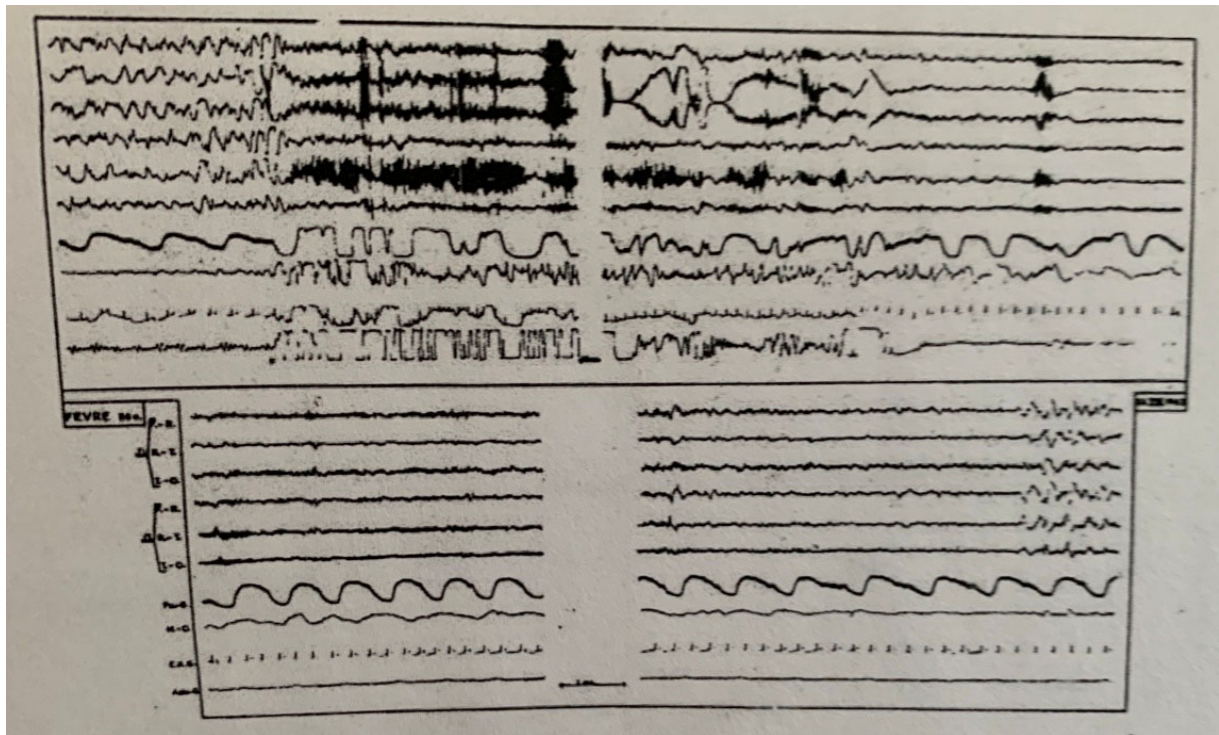


Figure 6. Sleepwalking in a 20-year old male recorded by very long electrode wires. The episode begins abruptly in stage 4 sleep (upper left segment) with movement artifacts on the actogram and intense muscle artifacts as he tries to get up. In the upper right segment, the patient gets out of bed, muscle artifacts are reduced, and the EEG has desynchronized. There is mild tachycardia. In the lower left the EEG shows stage 1 sleep patterns. In the lower right after several minutes of recording have been removed there is a transition into the onset of stage 2 sleep. Although sleepwalking historically was considered to reflect the acting out of a dream, the attacks never occur in REM sleep and are rarely associated with recall of dream-like mentation. They always begin in deep slow-wave sleep. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

Sleepwalkers also showed complex automatisms in bed during continuous sleep which mainly consisted in sitting up with eyes partially or fully open (Figure 7). Like for bedwetting, and also sleep terrors, events of sleepwalking occurred less frequently in the laboratory than at home. The Marseille studies confirmed that triggering episodes of somnambulism in chronic sleepwalkers by forced arousals in slow wave sleep occurred. Forced arousal during slow wave sleep in normal individuals will generate a confusional arousal but not induce a sleepwalking episode. As one cannot accept the hypothesis that the experimenter was always forcing the arousal at the precise time when the sleeper's ongoing mental activity (dreaming, thought-like, or other) was about to trigger a sleepwalking attack, this proved that it is the arousal process itself that is abnormal [25]. Almost any behavioral event during sleep is associated with some degree of arousal, but this does not confirm that it is a disorder of arousal. Sleepwalking has also been recorded by telemetry (Figure 8).

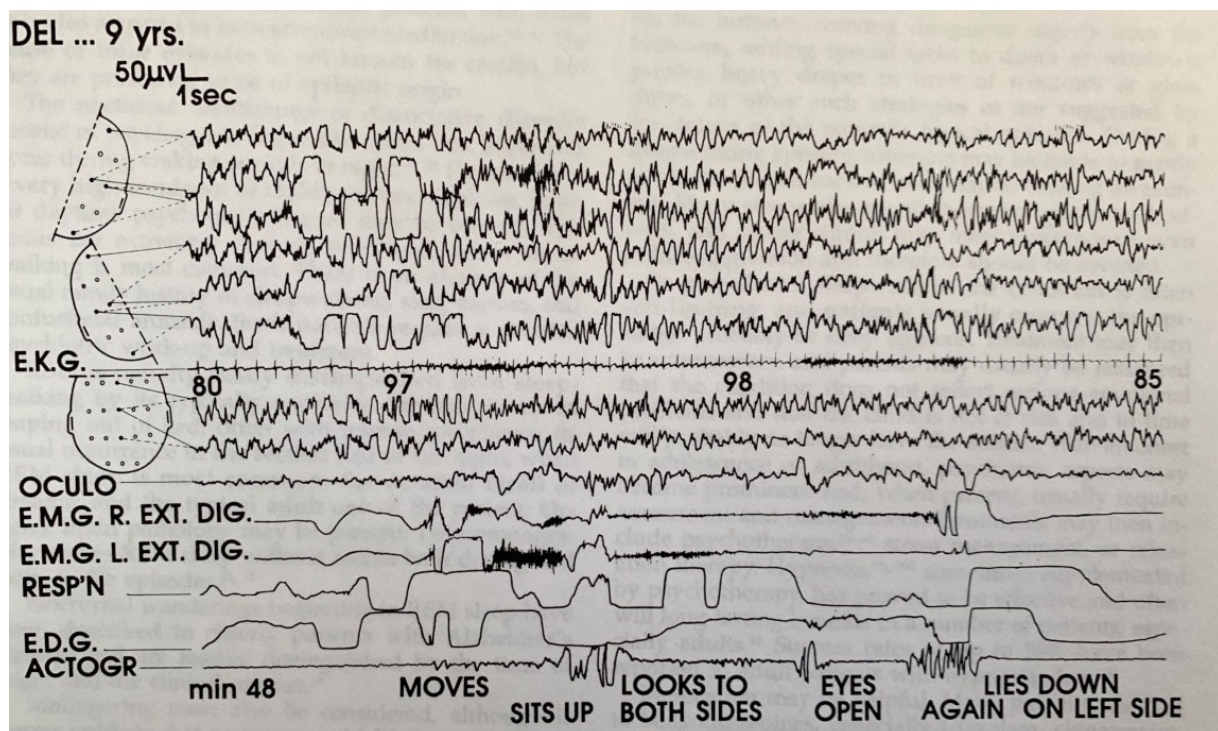


Figure 7. An episode of sitting up and looking around in a 9-year old chronic sleepwalking girl who moves, then sits up, looks around the room with eyes open and lies down again. The EEG shows continual sleep throughout the behavioral event. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

Jacobson and Kales found that sleepwalkers are more difficult to awaken than normal control subjects during both NREM and REM sleep. They also found no difference between sleepwalkers and controls in the amounts of NREM or REM sleep. Moreover, they noted the presence of a short period of high amplitude EEG delta waves before the onset of attacks, a pattern that Gibbs and Gibbs had described as “steady slow wave activity on arousal”. Later the UCLA group documented that fever was associated with an increase in the number of sleepwalking episodes [28].

Originally, sleepwalking was often considered to reflect the existence of dreaming. However, the likelihood that preceding or ongoing dreamlike mental activity can cause an attack is very low, as there is virtually always no recall of it. One would have to postulate that the experimenters were always creating the arousal just when ongoing mental activity, unknown by them, was about to cause an attack. Arousal by whatever cause, such as a loud noise, calling the sleeper’s name, internal body stimuli, or the arousal ending a sleep cycle, can provoke an episode of sleepwalking in sleepwalkers.

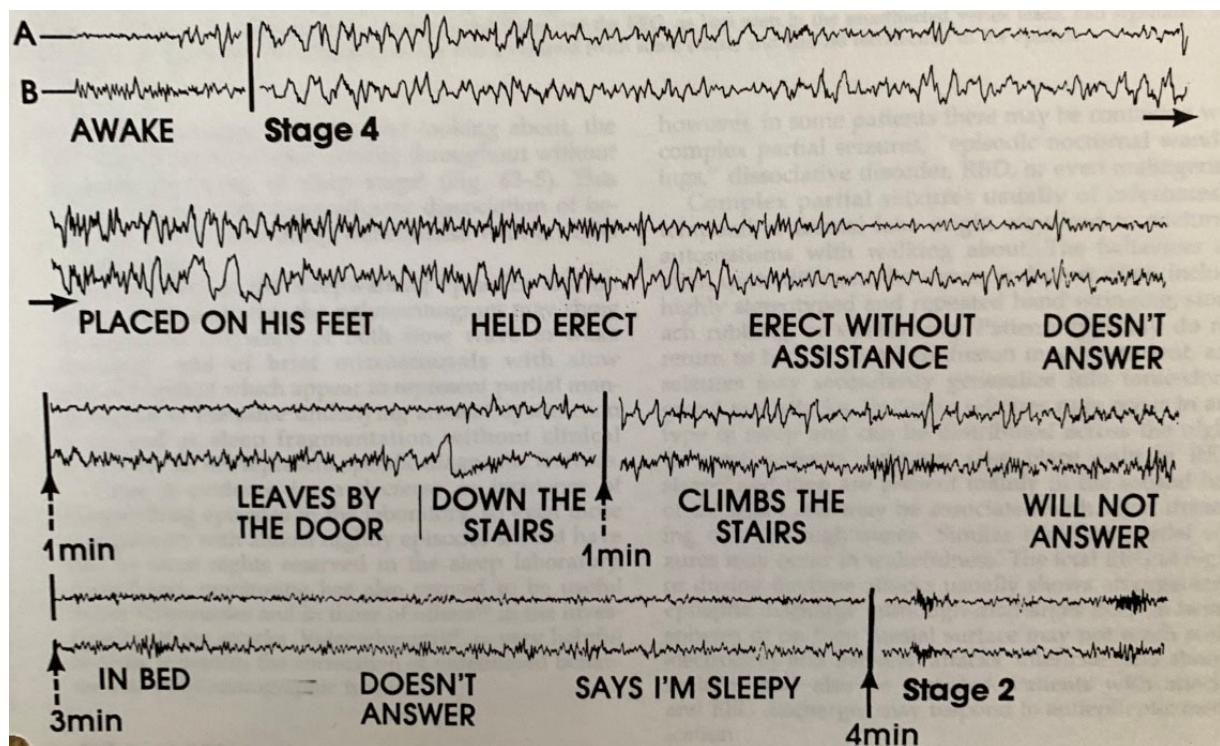


Figure 8. A sleepwalking episode induced by simply lifting a child sleepwalker out of bed and putting him on his feet. The recording was made using a four-channel Alvar telemetry system. The child is erect without assistance and does not initially respond to questioning. He then exits the bedroom, and descends two stairs. During a minute of recording that is removed, he walked some 10 m, turned around, then climbed back up the stairs. He still did not respond to questioning, climbed into bed again, did not answer, and at last says “I am sleepy” and 4 min later is in stage 2 sleep with spindles. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

The main issue is: why do such subjects have difficulty in generating a full awakening over a substantial period of time that allows the automatic behaviors? Such “twilight states” can occur in a number of other medical conditions, such as idiopathic hypersomnia, intense daytime sleepiness, petit mal status, temporal and frontal lobe partial seizures, and after head injury with concussion. Publication on the performance deficits evoked potential changes and other aspects of this frequent and interesting phenomenon have been published elsewhere [29]. Performance after a sleepwalking event has not yet been assessed in sleepwalkers versus matched control subjects. To my knowledge, no quantified comparison of thresholds for awakening have been made between sleepwalkers and controls. Bassetti and Weder recorded single photon emission computed tomography (SPECT) in an adult male sleepwalker [30]. The results suggested a dissociation arising from activation of thalamo-cingulate pathways with persisting deactivation of other thalamocortical arousal systems. Sleepwalking is rarely associated with aggressive behavior. This occurs most commonly when one tries to awaken a sleepwalker. A few studies have reported this pattern [31,32]. Broughton et al. reported the case of Ken Parks who during a sleepwalking episode attempted to strangle his father-in-law and then stabbed to death his mother-in-law. The Supreme Court of Canada found him non-guilty as he was unaware of his behavior during the attack [33].

A genetic component for this difficulty in fully awakening seems highly likely given the strong familiar tendency for somnambulism. There may well be a neurochemical deficit of dopaminergic, noradrenergic, histaminergic, or orexinergic nature, an increase of inhibitory substances, such as GABA, or an insufficiency of cortical or subcortical acetylcholine release during and after the arousal.

Despite their episodic nature, no reported studies showing concomitant epileptic discharges or frank epileptic seizures have been reported for sleepwalking even in patients with chronic epilepsy. However, sleepwalking must be carefully differentiated from the sleep-related epileptic seizures with automatisms described by Pedley and Guillemainault [34]. The criteria for distinguishing an epileptic from a non-epileptic event include the existence of frank epileptic seizures in awakening, consistency of the seizure symptoms and response to anti-epileptic medication.

Zadra et al. [35] have experimentally confirmed the clinical experience of an increased occurrence of sleepwalking after sleep deprivation when a homeostatic rebound of deep slow wave sleep takes place. A later SPECT study published by the Montreal group showed that during sleepwalking and in slow wave sleep there was a reduced perfusion in the frontal and parietal lobes compared to normal and also a reduced perfusion in the dorsolateral frontal lobe and the insula during recovery from sleepwalking. These findings help explain the confusional aspects, the automatic behaviors and the reduced sensation of pain inherent in somnambulism.

In a detailed study of QEEG during sleepwalking episodes, the Montreal group found that there was a mixture of sleep and wakefulness patterns which was demonstrated by the persistence of sleep in the frontal areas and waking patterns in motor areas. The episodes were preceded by EEG changes that were quite gradual in nature and showed that there was a change in functional connectivity before the onset of the clinical events [36]. A study by Gaudreau et al. showed differences in the dynamics of slow-wave activity between sleepwalkers and control subjects [37].

Sleepwalking fairly often affects many members of a family tree and so indicates a genetically transmitted propensity [38]. The treatment of sleepwalking has improved by behavioral approaches, psychotherapy, use of very slow arousals, and medication mainly tricyclic drugs, such as imipramine.

4. Sleep Terrors (Night Terrors, Pavor Nocturnus, Incubus Attacks)

The earliest known publication on sleep terrors (NREM nightmares) was a book published in 1949 by Earnest Jones with the title *On the Nightmare* [39]. Sleep terrors (pavor nocturnus in children and incubus in adults) were studied at the Centre Saint Paul in otherwise normal and in epileptic children, and also in normal adults [40]. The consist of a sleeper abruptly awakening from a very deep sleep, almost always sitting up, emitting a loud often bloodcurdling cry, and having subjective dyspnea and palpitations. At the end of an attack, patients are confused and have poor recall of the event [41]. They are of non-epileptic etiology as sleep studies never show a clinical epileptic seizure or an ictal discharge associated with the attack.

During attacks, the patient has a facial expression of extreme terror, marked sweating, tachycardia with palpitations and a remarkable increase in heart rate. The tachycardia can reach double the frequency of heart rate in the immediately preceding sleep (Figures 9 and 10). There is difficulty in full awakening and subjects are un-consolable. The events typically take considerable time to subside. As was reported by the Marseille group, there is also little or no recollection of events despite the abruptness and intensity of attacks [40]. Five years later Fisher et al. [42]. confirmed that the tachycardia was very abrupt and could double during the attack. They also found that there was a correlation between the degree of tachycardia and the intensity of the scream. Rarely there is a “hybrid attack” in which a sleep terror is immediately followed by sleepwalking. Fisher et al. later reported that diazepam was effective in suppressing sleep terror attacks [43]. Tassinari et al. have reported that sleep terrors are non-epileptic in enuretic children [44].

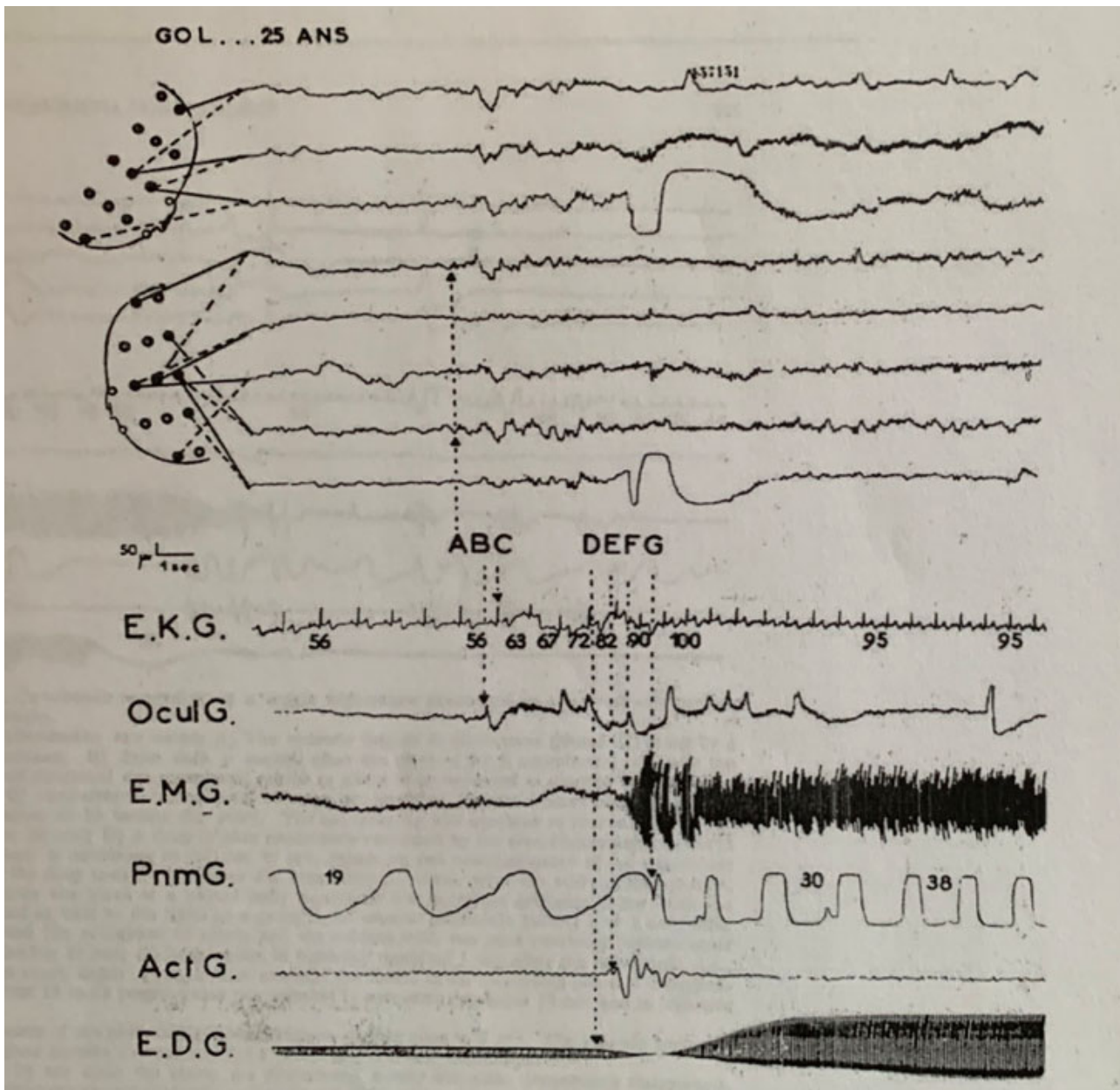


Figure 9. The onset of a sleep terror. It begins in slow-wave sleep and exhibits features of a very intense abrupt awakening. In rapid succession one documents (A) a brief increase in EEG slow waves; (B) an eye movement; (C) the onset of tachycardia; (D) the onset of a rapid decrease in skin resistance due to sweating; (E) movement on the actogram; (F) the beginning of tachypnea which rapidly doubles; (G) a notch on the respiratory channel due to the sudden increase in muscle tone. The recording continues in Figure 10. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

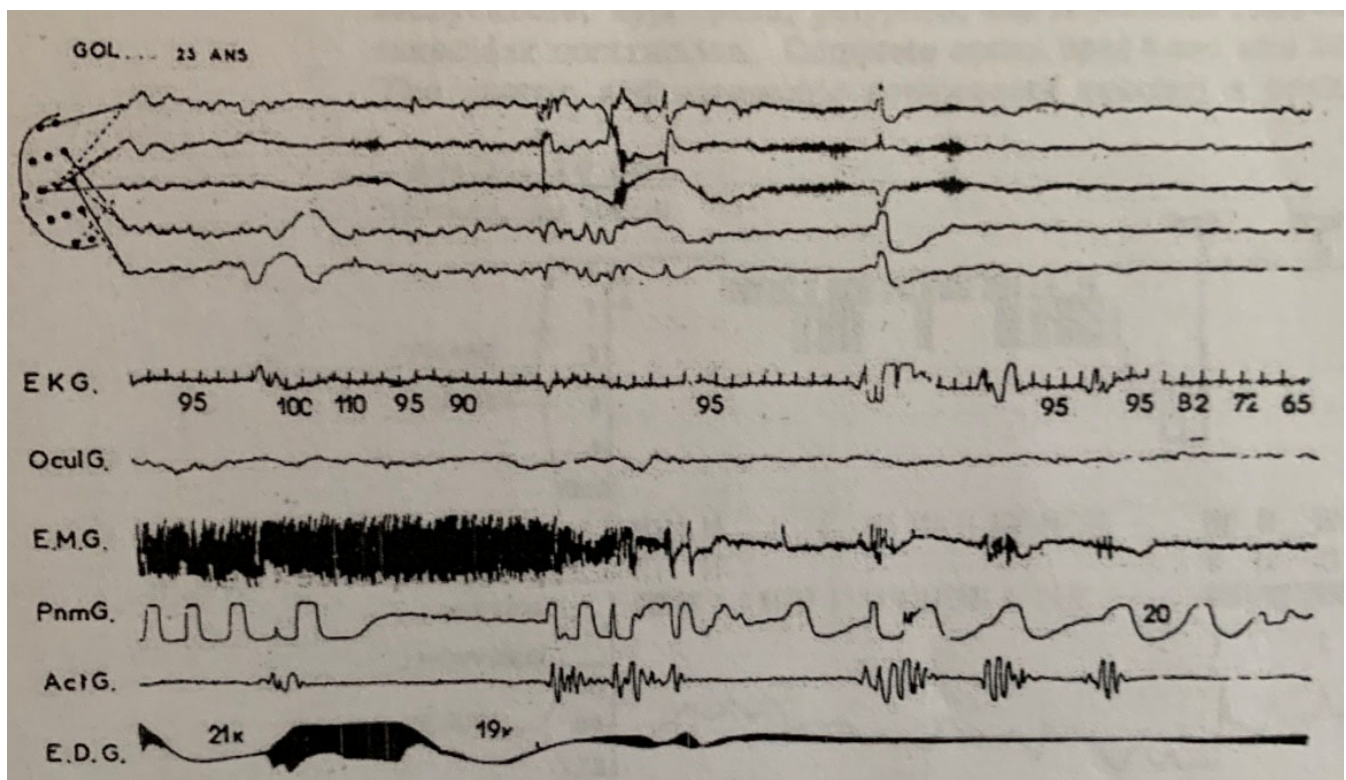


Figure 10. Continuation of the recording of a sleep terror. There is continuation of high muscle tension in the recording and the polypnea is stopped by an explosive bloodcurdling scream the heart rate has doubled from the level before the event and shows a maximum of 110 beats per minute. The actigraph documents significant body movements. This is followed by a progressive slowing of heart rate. The patient is not yet fully awakened and has had no eye movements typical of wakefulness (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

The recording of the onset of a sleep terror is shown in Figure 9 and its continuation in Figure 10.

A painting of a sleep terror is shown in Figure 11.

There is evidence that sleep terrors have a familial tendency, and therefore, there is a genetic component in their physiopathogenesis [45]. Hartmann has provided two publications on nightmares [46,47] one of which is on schizophrenia and creativity. A very interesting recent evolutionary explanation of sleep terrors has been published by Boydon et al. [48]. The treatment of sleep terrors has been pharmaceutical using diazepam and, more recently, clonazepam. Psychotherapy with counseling for patients to understand the nature and origin of the attacks is helpful.



Figure 11. Johann Heinrich Füssli (1741–1825) painted this picture in 1781. It expresses the belief at the epoque that the nightmare word is based on the German word Nacht. The root word of mare is the Teutonic word mar to designate a devil and becomes mare. Mare is also the English word for a female horse which is why a female horse is depicted leering in the back left of the painting. (Permission to reproduce granted by the Detroit Institute of Art).

5. Confusional Arousals

Early authors described confusional attacks as *l'ivresse du sommeil* by Marc [49] and as *Schlafrunkenheit* by von Gudden [50]. They occur with arousals from deep SWS and last typically from 5 to 15 min. Some 15% of young children and around 4% of adults [51] are affected. The behavior is that of intense confusion and disorientation in time and space, slow mentation, poor and slow speech, and difficulty in understanding what has happened. Confusional arousals can be associated with aggression, but this is rare. The changes in the visual evoked potential during a confusional arousal are shown in Figures 12 and 13.

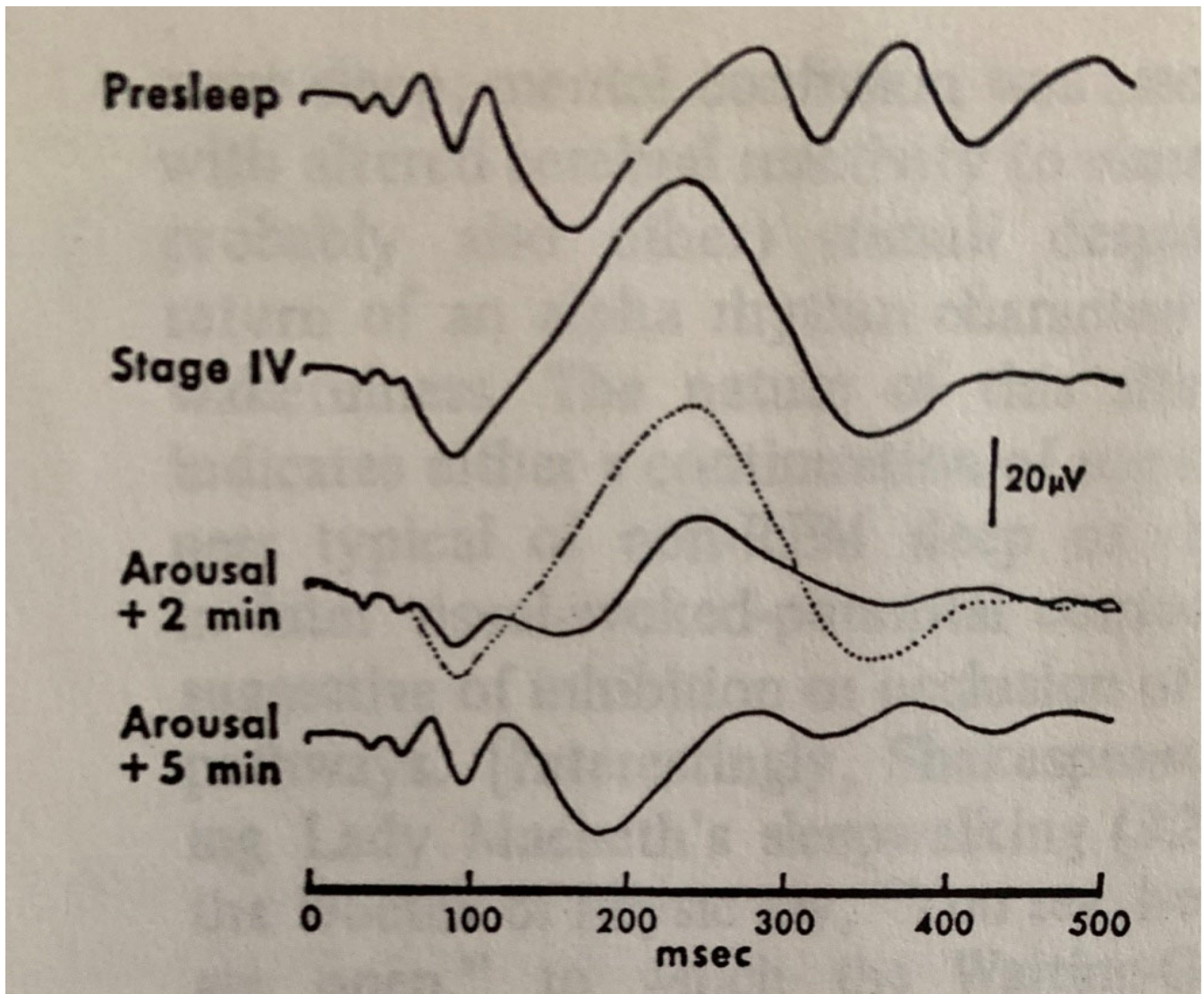


Figure 12. Visual evoked potentials (VEPs) in a healthy young adult in pre-sleep wakefulness, in deep stage 4 slow-wave sleep and 2 min and 5 min after a forced awakening associated with confusional arousals. Positivity at the active electrode is down. In the pre-sleep potential note the large positivity at about 190 msec latency followed by the summation of rhythmic alpha rhythm. In stage 4 there is a large three component response with a positivity at around 100 msec, a huge negativity at about 250 msec and a positivity at about 360 msec. This is essentially an averaged K-complex. Two minutes after the forced confusional arousal the VEP shows features of both slow-wave sleep and wakefulness. Five minutes after arousal the VEP is very similar to that of presleep wakefulness but of somewhat lower amplitude and with slightly longer latencies. (Reproduced with permission from Broughton R. Sleep disorders: disorders of arousal. Science 1968, 159:1070–1078, Published by: American Association for the Advancement of Science).

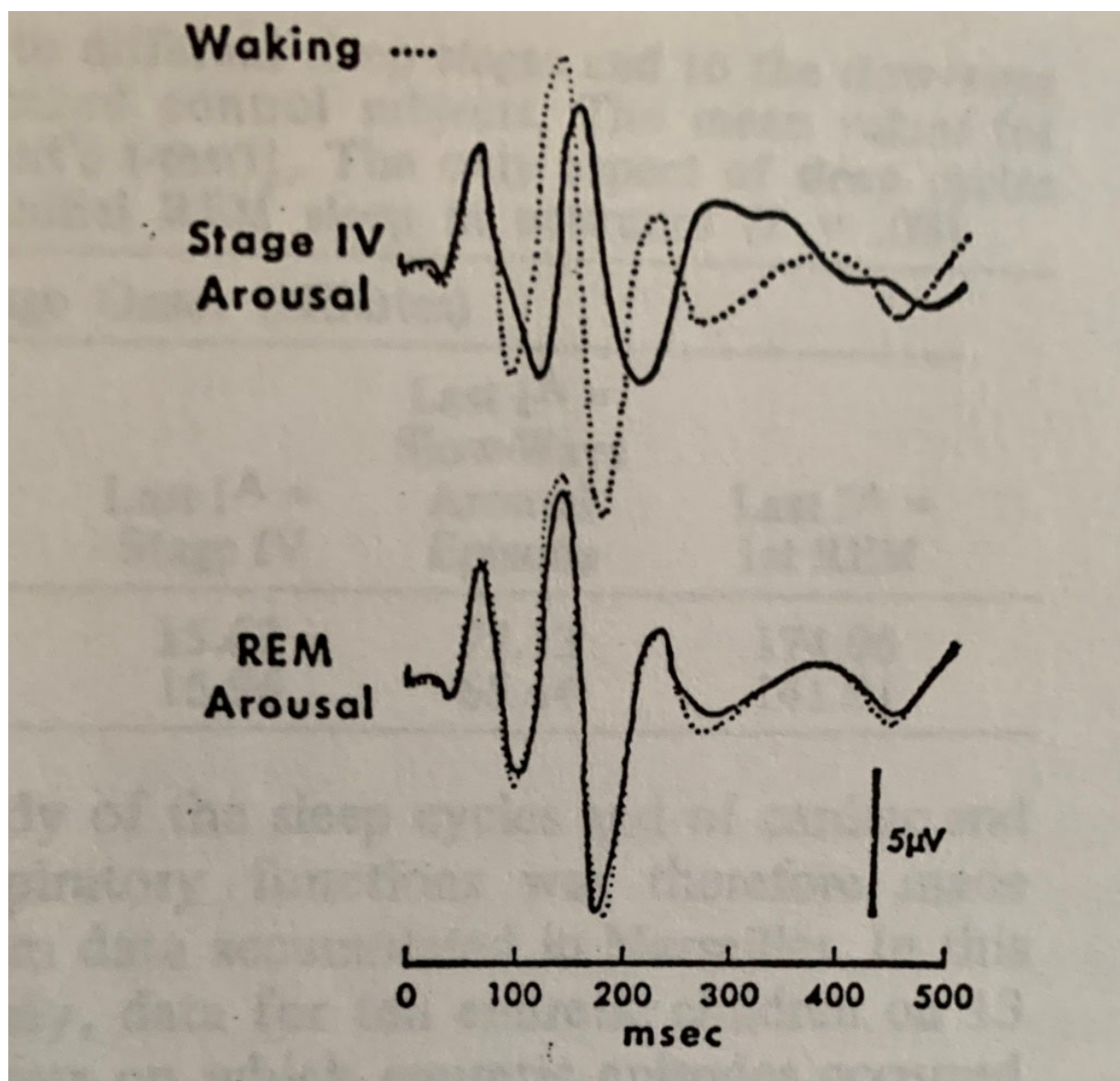


Figure 13. Visual evoked potentials in presleep wakefulness (dotted line) and after arousal in slow-wave sleep and REM sleep. After slow wave sleep arousals the VEP components are somewhat delayed and the amplitude is decreased. After REM sleep arousals the VEP is immediately essentially identical to that of pre-sleep wakefulness. (Reproduced with permission from Broughton R. Sleep disorders: disorders of arousal. *Science* 1968, 159:1070–1078, Published by: American Association for the Advancement of Science).

Similar symptoms upon awakening called sleep drunkenness are seen in Idiopathic Hypersomnia as was first described by Roth et al. [52]. The assessment of visual evoked potentials and visual vigilance using forced SWS arousals in normal subjects by Feltin and Broughton shows a mixture of evoked potential components of sleep and wakefulness and poor recognition of the color of visual stimuli in a vigilance test [53]. There is a well-documented and very interesting instance of a confusional arousal in a rat that attacked a cat [29].

6. Development of the Concept of “Disorders of Arousal”

After leaving Marseille and moving to the Montreal Neurological Institute in the fall of 1964, and reflecting upon our results on the NREM parasomnias of bedwetting, sleep

walking, sleep terrors and confusional arousals, I realized that the common pathophysiological aspects were their occurrence in very deep slow wave sleep, the genetic tendency for the attack types, the memory deficits and, above, all the fact that all could be triggered by forced arousal in slow wave sleep. It was, therefore, evident that it was the arousal process itself that was abnormal. Forced arousals in SWS in normal subjects did not induce sleepwalking or sleep terrors but did lead to a confusional arousal documented by slow reaction times and evoked potential changes. I, therefore, proposed in a lead article in the journal *Science* [25] that these disorders were disorders of arousal and not simply disorders with arousal. This new category of sleep disorders was incorporated into the first International Classification of Sleep Disorders chaired by Howie Roffwarg and has continued to be included in all subsequent classifications, and is now universally accepted.

The main features of these parasomnias can be summarized as in Table 1.

Table 1. A comparison of the features of four parasomnias associated with arousals.

	From SWS	Can Induce	Automatism	Amnesia	Autonomic	Genetic
Bedwetting	++	+	-	+	+	+
Sleepwalking	++	+	+	+	—	+
Sleep terrors	++	+	+	+	+++	+
Confusional arousals	++	+	+	+	—	+

7. Aggression and Sleep

Attacks or aggression are most common during episodes of confusional arousal and also when persons try and awaken sleepwalkers who then strike out against the awakener as reported by Broughton and Shimizu [54]. These may be very intense and cause injury. Homicide may occur during sleepwalking as is confirmed in the 1878 case of Yellowless [31] and the 1994 case documented in great detail by Broughton et al. [55] which involved Ken Parks who strangled and stabbed his father-in-law, who did not die, and then killed his mother-in-law, whom he loved, by repeated stabbing. Sleep studies and study of slow-wave arousal parasomnias in his family tree helped in confirming that it was indeed during an episode of somnambulism. A histogram of Ken Park's sleep the night after the homicide of his mother-in-law is presented in Figure 14. Eighteen months after the homicide during which he was treated with nocturnal clonazepam it shows an absence of slow wave sleep (Figure 15). A study of his family tree shows frequent bedwetting, sleep talking and sleep terrors (Figure 16). Ken Parks was found innocent of murder by the Supreme Court of Canada based on the evidence that there was no motivation, that during sleepwalking the sleepwalker is unable to plan his or her behavior, and that during sleepwalking the individual is unaware of his or her behaviors and cannot control them.

A further case of homicidal sleepwalking involved the killing of his wife Yarmilla due to 44 stab wounds by Scott Falater, a resident of Phoenix, Arizona. He was accused of murdering his wife on the night of 16 January 1997. According to an eyewitness, Falater was also seen holding his wife's head underwater. When he was tried, the prosecution claimed that after the murder had been committed, Falater changed his clothes, put the murder weapon in a Tupperware container, put the container in a trash bag with his boots and socks, and then stashed the bag in the spare tire well in the trunk of his car. Prosecutors testified that Falater's actions were "too complex" to have been carried out while sleepwalking. In June 1999, Scott Falater was convicted of first-degree murder and sentenced to life in prison without the possibility of parole.

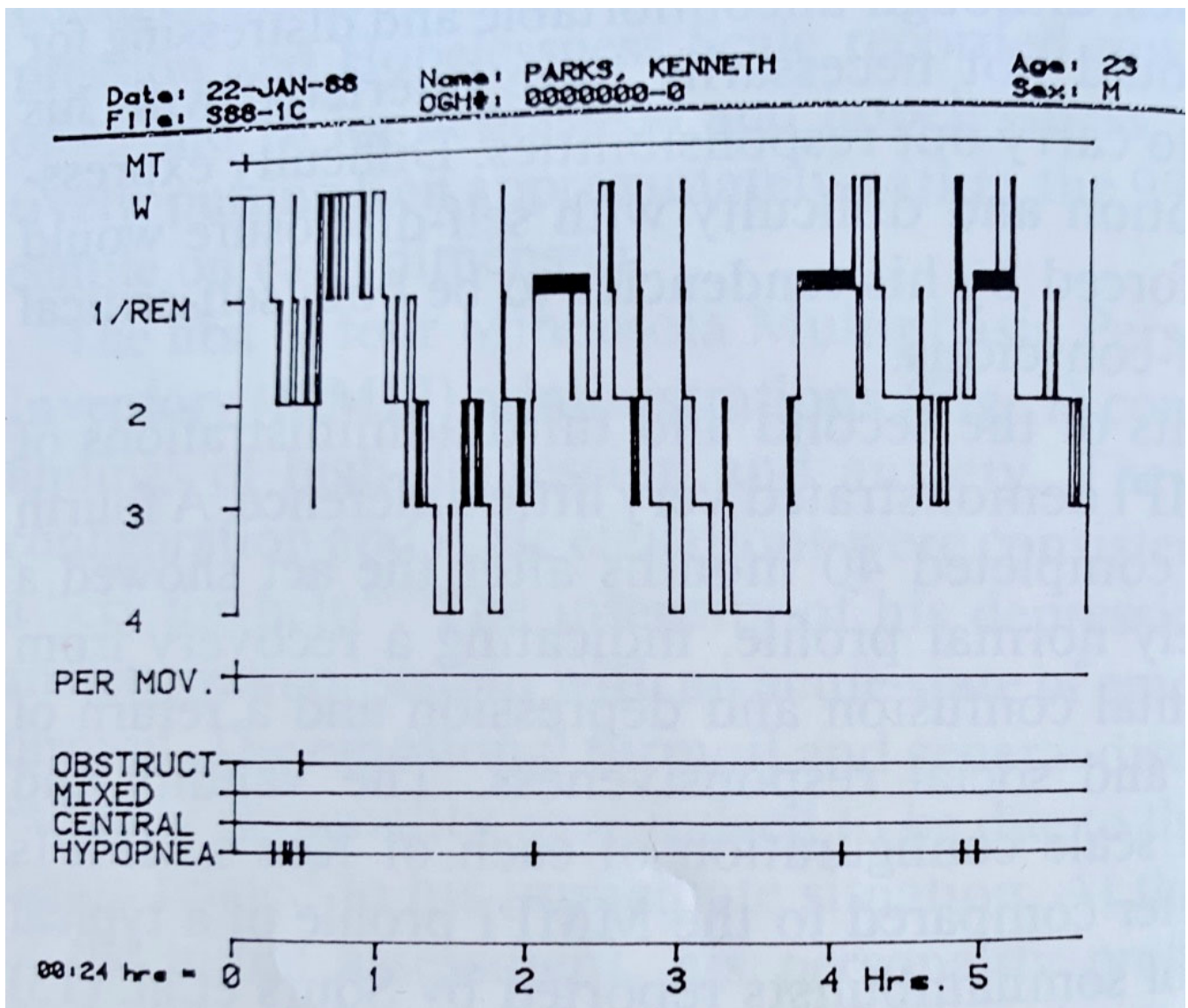


Figure 14. Histogram of the polysomnogram the night after the homicide of his mother-in-law by Ken Parks. Note the fragmented sleep and, especially, the presence of five awakenings from deep slow-wave sleep (SWS). These occur at around 30 min, 2 h, 3 h, 4.5 h and 6 h after sleep onset. Normal subjects very rarely have direct arousals from sleep-wave sleep to waking, and this pattern of multiple SWS arousals is characteristic in sleepwalkers. (Reproduced with permission from Broughton R., Billings R, Cartwright R et al. Homicidal Somnambulism: A Case Report. *Sleep*, Volume 17, Issue 3, May 1994, Pages 253–264, <https://doi.org/10.1093/sleep/17.3.253>, published by Oxford University Press).

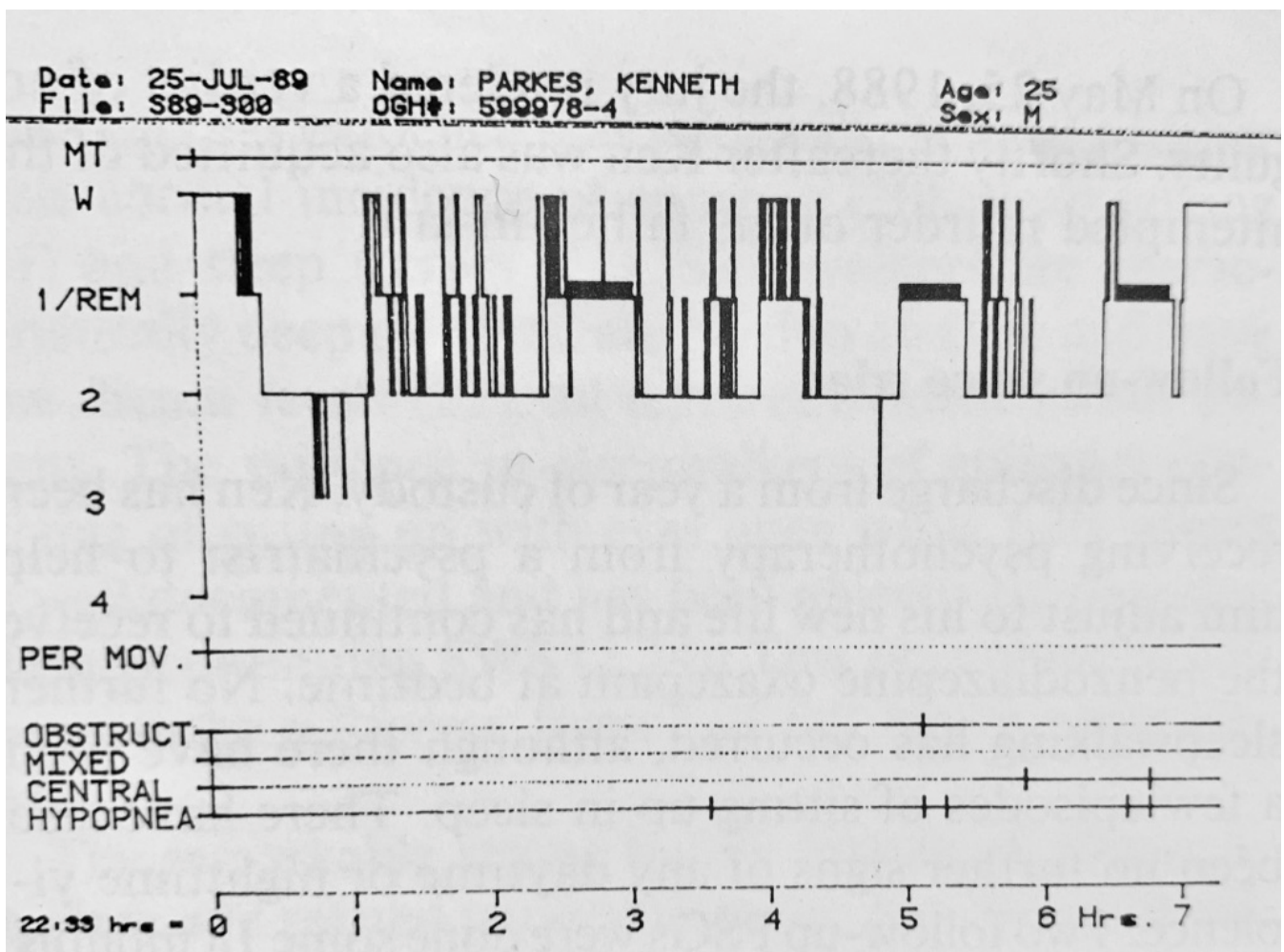


Figure 15. Histogram of the sleep of Ken Parks some 18 months after the homicide. He had been treated by the benzodiazepine clonazepam during this period. Like with all benzodiazepines there is more or less total suppression of deep slow-wave sleep. There was only a single direct transition from SWS to wake and this occurred about one hour after sleep onset. (Reproduced with permission from Broughton R, Billings R, Cartwright R et al. Homicidal Somnambulism: A Case Report. *Sleep*, Volume 17, Issue 3, May 1994, Pages 253–264, <https://doi.org/10.1093/sleep/17.3.253>, published by Oxford University Press).

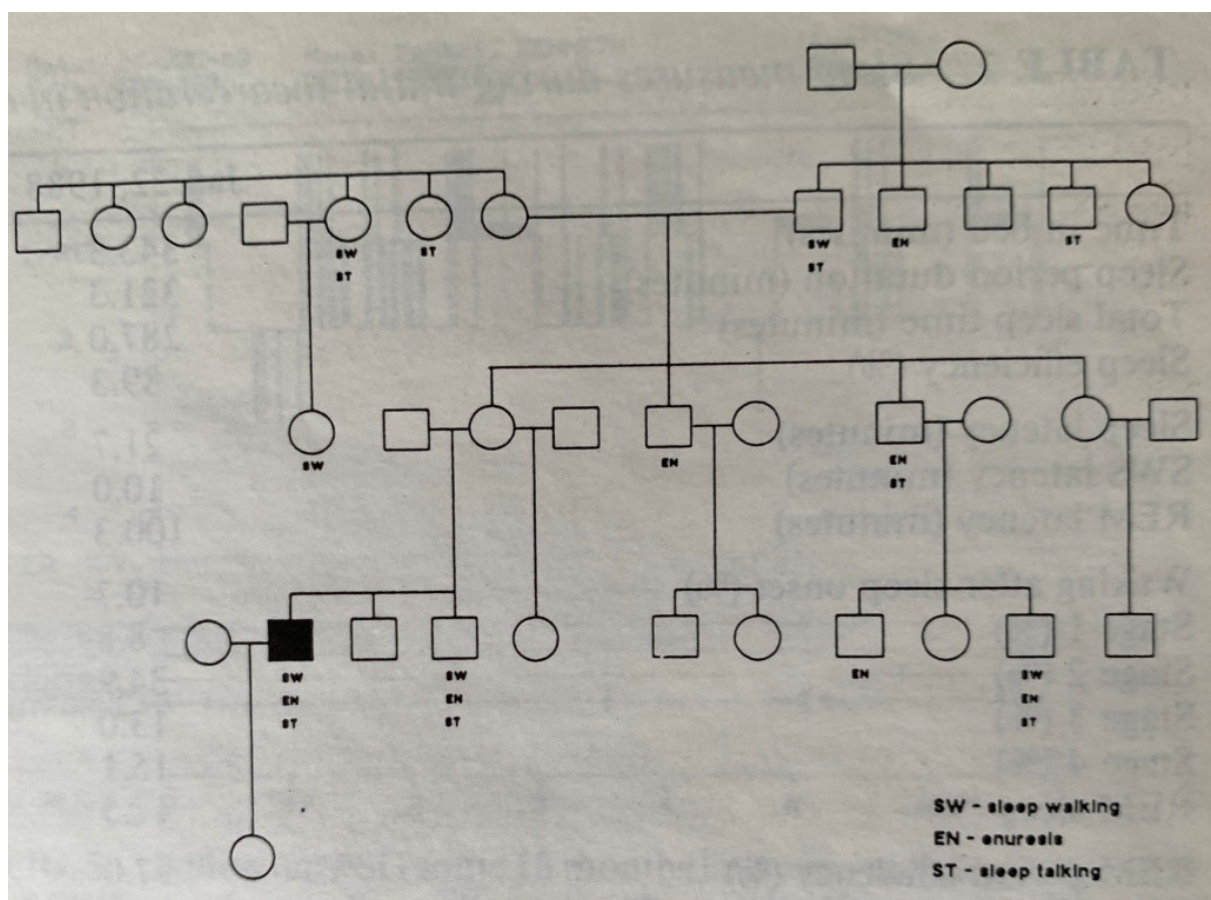


Figure 16. This diagram shows the family tree of Ken Parks. Males are in squares and female are in circles. The black square is Ken Parks. It is notable that in his extended family there are many cases of enuresis nocturn, sleepwalking and sleep talking. Such a family pattern is present in many patients with enuresis, sleepwalking, sleep terrors or confusional arousals. (Reproduced with permission from Broughton R, Billings R, Cartwright Ret al. Homicidal Somnambulism: A Case Report. Sleep, Volume 17, Issue 3, May 1994, Pages 253–264, <https://doi.org/10.1093/sleep/17.3.253>, published by Oxford University Press).

8. Nocturnal Anxiety Attacks

Gastaut et al. were the first to publish on the occurrence of nocturnal anxiety attacks [45]. They studied four patients with the condition and recorded five nocturnal attacks and two diurnal ones. The nocturnal attacks usually occurred during sub-phase 1A showing slowing and diffusion of the alpha rhythm. Clinically the patient would wake up with subjective feelings of impending catastrophe and intense fear. Exceptionally the episodes occurred during REM sleep and were considered as REM anxiety attacks. They are to be differentiated from sleep terrors by the much lower levels of tachycardia and their lack of occurrence in deep slow wave sleep. Patients with the condition often have daytime anxiety attacks in wakefulness and have chronic trait anxiety. There have been few subsequent studies reporting nocturnal anxiety attacks.

9. Vivid Hypnagogic and Hypnopompic Hallucinations

Hypnagogic/hypnopompic hallucinations are experienced quite frequently in children and in adolescents. They may also affect adults, although this is quite rare. These hallucinations can be extremely frightening. Younger children may become terrified enough to scream and cry and they may be confused by their hallucinations. The episodes most usually occur at sleep onset (hypnagogic form) and, less frequently, upon awakening

(hypnopompic form.) Children may become too embarrassed or stressed to discuss them, and may not be able to explain their feelings of fear. Such hallucinations have been reported in 39% to 50% of pediatric patients with narcolepsy. They represent one of the five main features of narcolepsy type 1. The other symptoms of narcolepsy include more or less irresistible brief sleep attacks in wakefulness, cataplexy, sleep paralysis, and fragmented sleep including REM sleep.

Hypnagogic or hypnopompic hallucinations can be mistaken for, or diagnosed as, panic attacks, nightmares, or night terrors in children. Very severe episodes can even be confused with psychotic disorders. There is no widely accepted treatment. Subjects should be persuaded to discuss the content of their hallucinations with health care workers. If hypnagogic hallucinations become very frequent and intense, the symptoms may create a form of sleep onset insomnia. Keeping regular hours for sleep can be beneficial, as may REM suppressant medication or the use of a short acting benzodiazepine at sleep onset. Psychotherapy may also be helpful.

10. REM Nightmares (REM Sleep Terrifying Dreams)

The term nightmare is often used to designate both NREM sleep terrors and REM sleep related terrifying dreams. It is however best used for dreamlike mental activity during sleep that exhibits content that progressively becomes frightening to the sleeper and leads to awakening. As these nightmares are generated during REM sleep, they typically occur in the second half of nocturnal sleep, whereas sleep terrors occur during deep slow wave sleep typically in the first third of night sleep. The term night terror is often used for the sleep terror but should be abandoned, as the episodes may also occur in daytime naps with slow-wave sleep. The earliest polysomnographic study of REM nightmares was published by Fisher et al. in 1970 [41]. In a subsequent article, Fisher and colleagues published a detailed distinction between REM nightmares and sleep terrors using polysomnography [42]. They noted differences in the state in which they occurred, the great tachycardia associated with sleep terrors, whereas heart rate shows only a minimal increase in REM nightmares and the fact that the latter are vividly recalled whereas sleep terrors are generally associated with little or no recall. Major contributions to REM nightmares were also made by Ernest Hartmann in a book [46] and in an article that noted the possible association with schizophrenia and with creativity [47]. Some reduction in REM nightmare frequency and intensity can be obtained using REM sleep suppressant medication, such as tricyclics (imipramine) and MAO inhibitors. See Table 2 for a comparison of terrifying dreams and sleep terrors.

Table 2. A comparison of the differences between the terrifying dreams of REM sleep and sleep terrors.

	Terrifying Dreams	Sleep Terrors
State of occurrence	REM sleep	SWS arousal
Stability of state	Stable (REM)	Changing
Circadian aspect	Late night	Early night
Preceding autonomic activation	Present	Absent
Intense behavioral arousal	Rare	Always
Heart rate increase	Mild	Marked
Respiratory rate increase	Mild	Marked
Muscle tone increase	Mild to moderate	Marked
Mental confusion	Minimal	Marked
Memory problems	Rare	Marked
Paralysis	Atonic	Hypertonic
Recalled mental activity	Dream-like	Single scene
Anxiety level	Mild to moderate	Intense
Genetic factors	Minimal	Important

11. REM Sleep Behavior Disorder (RBD)

Carlos Schenck and colleagues first reported chronic behaviors in REM sleep in 1986 [56]. The condition consists of an attack in REM sleep without atonia and paralysis. The sleeper lives out a dream for which there is more or less full recall and the dreams often have violent content. There are often marked myoclonus and vigorous movements (Figure 17). Almost always the episodes occur in the second half of the night when REM sleep predominates. RBD can be idiopathic but in most instances appears in the context of a neurological disorder, as the episodes often precede by many years the appearance of Parkinson's disease, Lewy body disease, stroke, or other neurological conditions affecting the brainstem. There is an animal model of RBD which was discovered in 1965 by Jouvet and Delorme [57]. It was induced by lesions of the peri-locus coeruleus. During paradoxical sleep (active sleep) the cats showed both oniric and predatory behaviors. RBD usually is present in adults but may also occur in children. At times there is an overlap syndrome having the combined features of narcolepsy-cataplexy, sleepwalking, sleep terrors and RBD, with or without marked period movements and twitching in NREM sleep. Occasionally multiple sclerosis, brainstem cancer, brain trauma, Mobius syndrome or psychiatric disorders are related. SPECT and PET scans have shown reduced dopamine innervation in the striatal areas similar to what is found in Parkinson's disease. Treatment with clonazepam has been almost always successful in both adults and children.

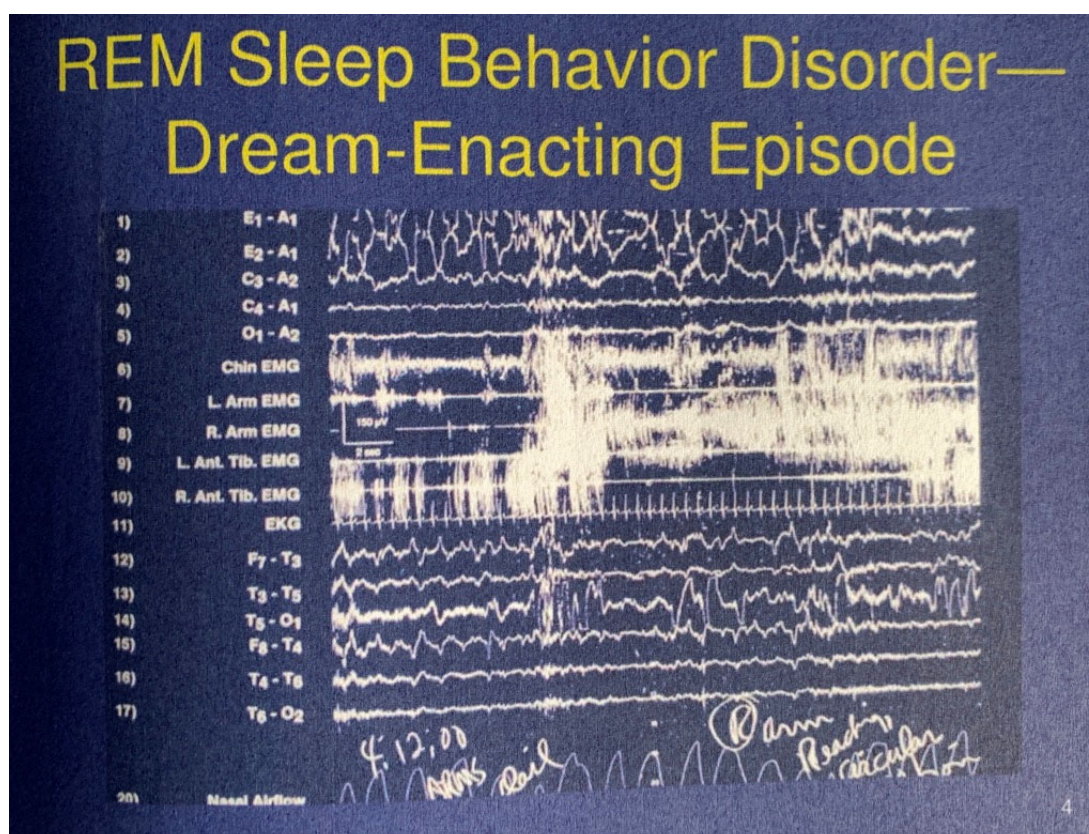


Figure 17. An episode of REM sleep behavior disorder (RBD). Note the intense rapid eye movements in the top 2 channels. The EMG channels show a loss of muscle tone at the start associated with some myoclonic potentials and then high amplitude EMG artifacts associated with the writhing movements of the patient. Some notes are written at the bottom of the tracing by the technician. The patient recalled a detailed dream in which he was in a boat on Lake Superior. In his recalled dream the boat was rocking dangerously and he fell into the hold of the boat. (Reproduced with permission from Schenck CH, Bundlie SR, Ettinger MC et al. Chronic behavior disorders of human REM sleep: a new category of parasomnia. *Sleep* 1986; 9(2): 293–308, published by Oxford University Press, 2021).

12. Nocturnal Sleep Paralysis

Night sleep paralysis is quite common. It may be induced in normal subjects by irregular sleep schedules, such as is often encountered after jet lag [58] and in medical students [59,60]. As sleep paralysis occurs in REM sleep without atonia there is often recall of an associated dream. Sometimes the inability to move causes acute anxiety and the dream is a REM nightmare. In some instances, just touching the person who is experiencing sleep paralysis is sufficient to break the spell and movement is possible. There is a rare form of familial sleep paralysis which was first carefully documented by Bedrich Roth and colleagues [61]. Sleep paralysis is also one symptom of the tetrad of narcolepsy with cataplexy [62,63]. Treatment in normal subjects is normally effective by avoiding irregular hours of sleep and jet lag.

13. Nocturnal Paroxysmal Dystonia

Paroxysmal dystonia in NREM sleep was first reported by Lugaresi and colleagues [64]. The episodes consist of ballistic and choreo-athetoid movements that are short lasting (<1 min) and are often preceded by an EEG pattern of arousal [65] (Figure 18). There is no sex or age predilection and no familial transmission. Some patients also have similar movements in wakefulness and rare patients have associated epileptic seizures. They respond well to carbamazepine, even in small doses. Nocturnal paroxysmal dystonia must be distinguished from similar patients documented by Lee et al. that, however, show a familial occurrence as an autosomal dominant trait and which have a poor or no response to carbamazepine therapy.

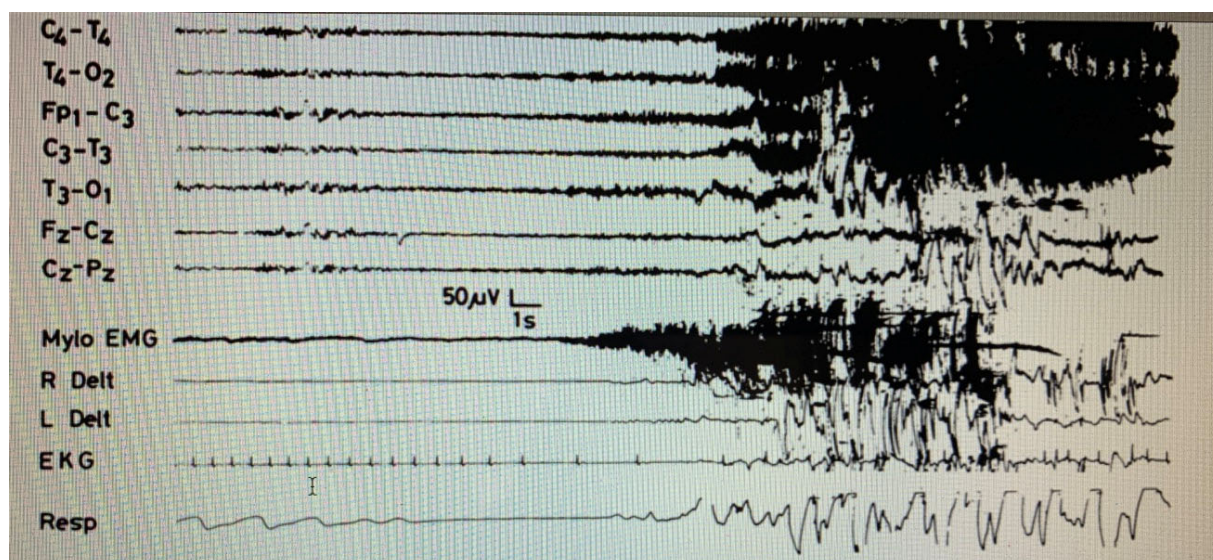


Figure 18. An episode of nocturnal paroxysmal dystonia. The episodes occur in NREM sleep here in stage 2 sleep. The chin muscle (mylo EMG) channel shows an increase in muscle tone and there are also movement artifacts in the deltoid EMGs and elsewhere. The episode lasted about 15 sec and the patient remained asleep. (Reproduced with permission from Lugaresi E, Cirignotta F and Montagna P. Nocturnal paroxysmal dystonia. *J Neurol Neurosurg Psychiatry*. 1986; 49: 375–380 Published by BMJ Journals).

14. Sleep Talking (Somniloquy)

The earliest sleep studies of sleep talking were by Gastaut and Broughton [9] and showed that sleep talking could occur in any stage of sleep but was most common in stage 2 of NREM sleep. As would be expected speech was associated with a variable lightening of the sleep patterns which occasionally were those of stage 1 and at times had some diffuse alpha activity although the subject remained asleep. In some instances, it

was possible to create a brief conversation with the subject as is documented in Figure 19. In this case, the experimenter asks if the subject is asleep and receives the answer “Yes!” and then asks, “do you hear me?” and the answer is “Yes!”! Throughout this exchange, the EEG shows continuous patterns of sleep. This fascinating brief conversation indicates that the subject clearly hears the experimenter, understands the questions, realizes he is asleep, and answers appropriately. After awakening, he had no memory of the event. A particularly interesting situation, which unfortunately could not be recorded, involved two sleep talkers who had a dialogue in which they exchanged questions and information while both remained asleep and had no recall of doing so after their awakening. Such findings raise questions concerning consciousness and dissociated forms of consciousness.

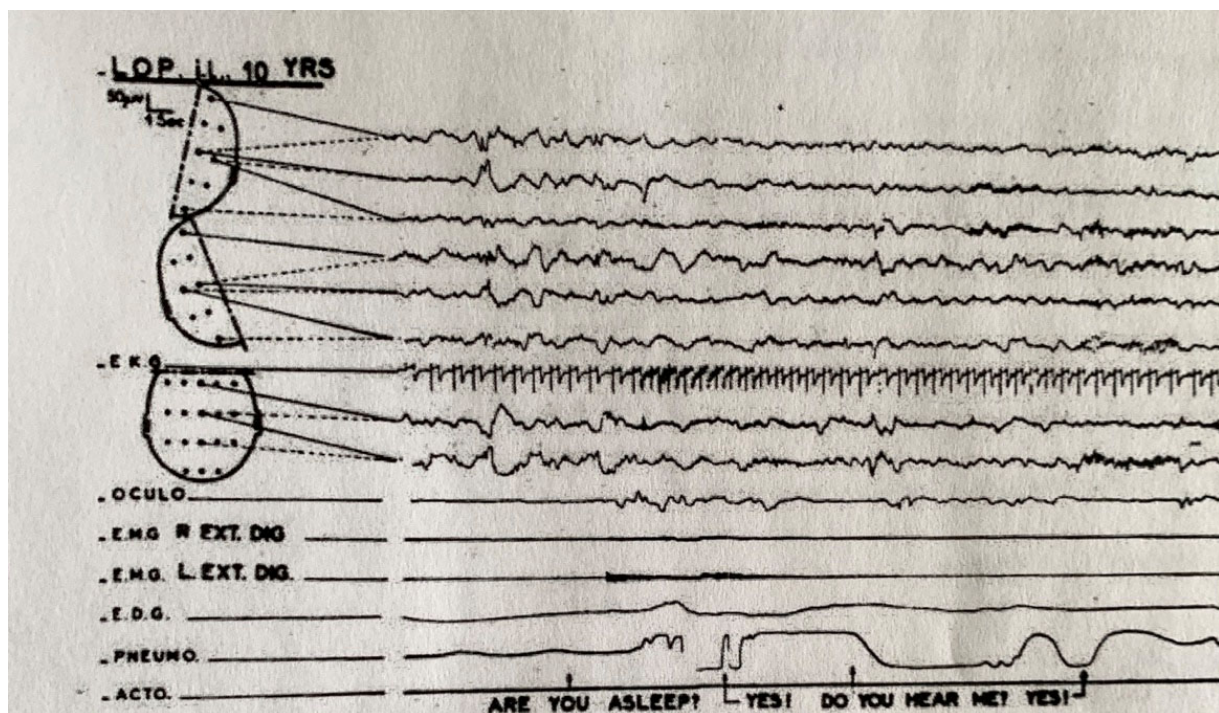


Figure 19. An episode of sleep talking in a child in NREM sleep. The investigator the investigator asks, “Are you asleep?” and the child answers “yes”. Later the investigator asks, “do you hear me?” and the child responds “yes”. Each time that a question is asked there is an evoked k-complex and each time the child answers there is a brief arousal response. Therefore, the child has heard the questions, understood them, and answered appropriately. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

15. Sleep Starts (Hypnic Jerks)

Although early books on sleep and its disorders mention the occurrence of episodes of intense body jerks often mainly of the legs during the period of somnolence at sleep onset, the first polygraphic recording of the phenomenon was published by Oswald in 1959 [66]. However, Oswald did not record peripheral EMGs and the sleep starts were only evident in his recordings as movement artifacts. Figure 20 (taken from reference [67]) shows a personal recording that includes EEG, eye movements, submental EMG, EKG, respiratory and abdominal measures, and leg EMGs. Oswald called the phenomena sleep starts. Hypnic jerks appear to be an accentuation in somnolence of the waking startle response and are quite frequent. Sometimes they are very intense, even painful, and can involve the legs, arms, head and thorax in massive flexion. They can lead to abrupt and brief awakenings with the person then falling back asleep. In rare cases, there is a series

of intense generalized myoclonic jerks that leads to a form of sleep onset insomnia with a consequent increase in daytime sleepiness.

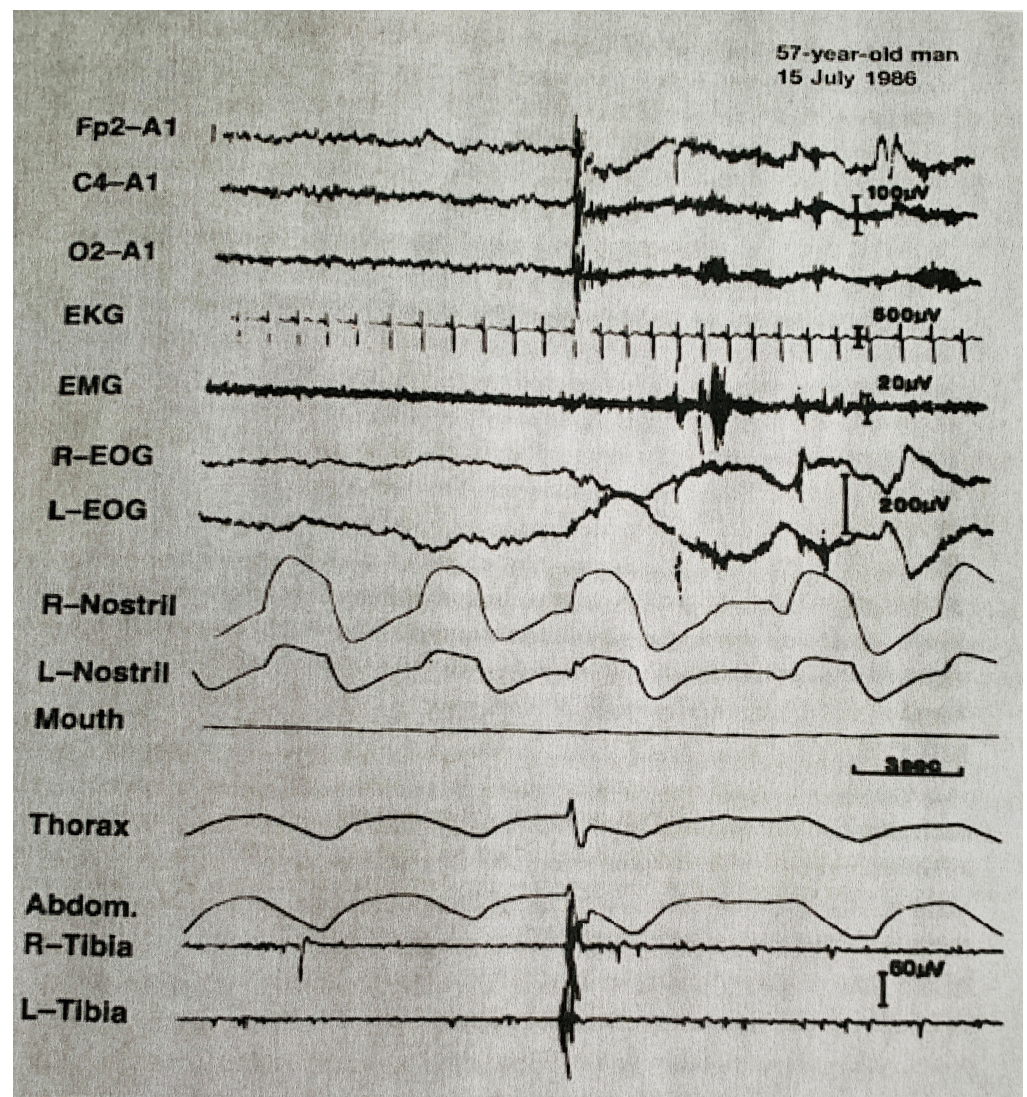


Figure 20. A sleep start, also called a hypnic jerk, in a 57-year old drowsy man recorded in July 1986. The jerk consists of total body myoclonus mainly affecting the legs but also often the arms, head and chest in an abrupt flexion. The phenomenon occurs apparently spontaneously during stage 1 somnolence at sleep onset. In this personally recorded example, the myoclonus is recorded as EMG potentials in the scalp electrodes, abrupt expiration in the chest and nasal leads, and as myoclonic EMG potentials recorded over the right and left anterior tibialis muscles. The subject is in stage 1 drowsiness prior to the massive sleep start and immediately afterward there is diffuse alpha activity and some eye movements which together indicate an arousal due to the jerk. As the name suggests, sleep starts are akin to the startle responses in wakefulness that are induced by unexpected intense stimuli. The cause of their occurrence in somnolence is uncertain but may well be due to endogenous stimuli from internal organs. Repeated sleep starts during somnolence may lead to a form of sleep onset insomnia with a consequent increase in daytime sleepiness. (Reproduced with permission from Broughton R. Some important underemphasized aspects of sleep onset. In: R Ogilvie and J Harsh (eds). *Sleep Onset Mechanisms*. American Psychological Association, Arlington VA 1994: 19–36, published by American Psychological Association).

16. Jactatio Capitis Nocturna

Jactatio capitis nocturna is a form of tic occurring at sleep onset that consists of rhythmic lateral movements of the head and at times of the entire body. It generally occurs in young children but may persist into adolescence or even adulthood. It was first recorded polygraphically by Gastaut and Broughton [9]. A burst of the movements is generally preceded by several nystagmoid eye movements and takes place in stage 1 drowsiness. (Figure 21). The rhythm is typically slow at 1 to 2 per second and the pattern recurs in bursts lasting in the order of 5 to 10 s. There is no accompanying significant change in heart rate or respiratory rate. Although it mainly occurs at evening sleep onset, in severe cases body rocking movements may recur throughout the night in all stages of sleep without any modification of the EEG sleep patterns (Figure 22). In some instances, the condition is associated with mental retardation or personality problems. The body rocking may be very intense and can displace the bed. Unfortunately, if the child is causing self-injury there is no treatment other than using restraints. Silvestri and Walters [68] have reported the association of such rhythmic movements with many sleep disorders, with sleep-related epileptic seizures, and with Tassinari's description of innate motor patterns related to central pattern generators [69].

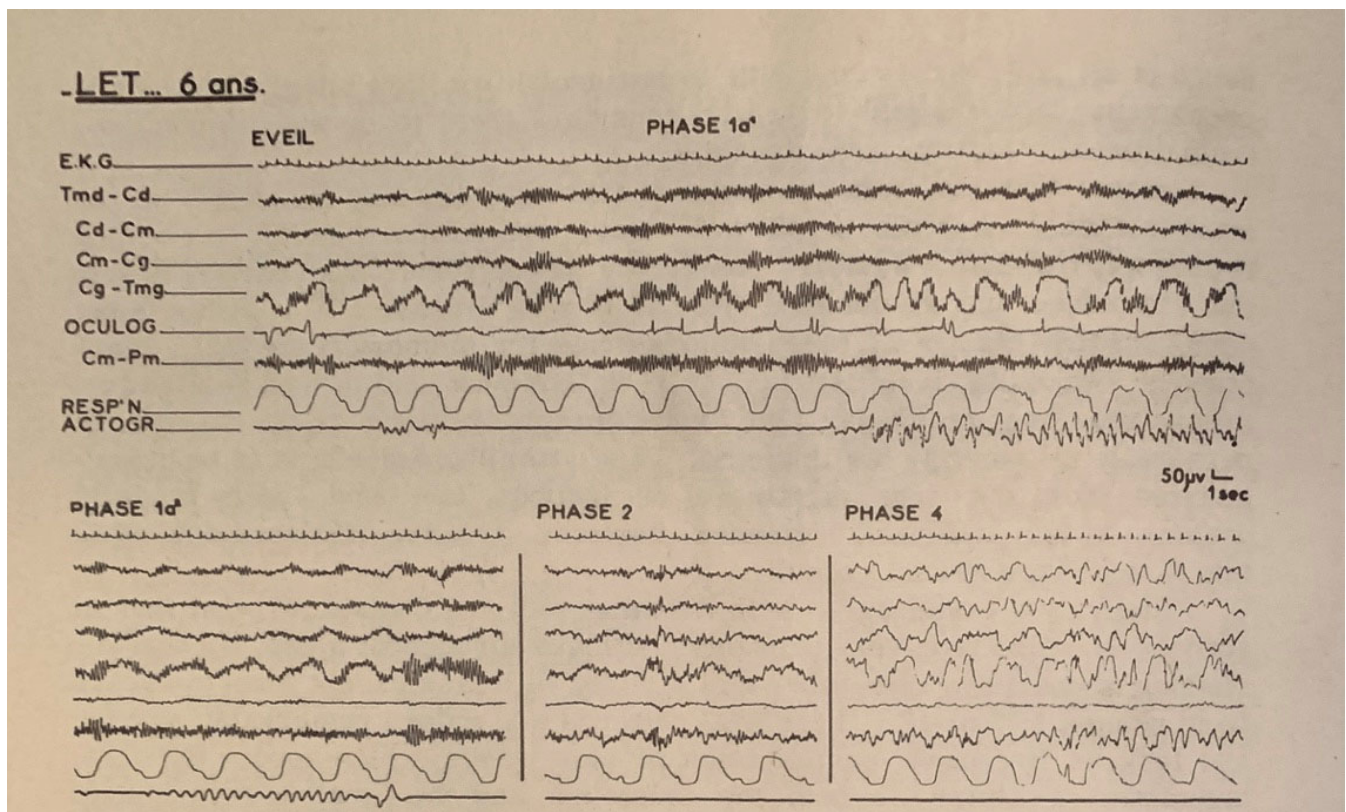


Figure 21. An episode of jactatio capitis nocturna in a 6-year-old boy. The movements consist mainly of repeated head movements which are picked up by the actogram. Some respiratory movement artifacts are also recorded by the mid-temporal electrodes. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).).

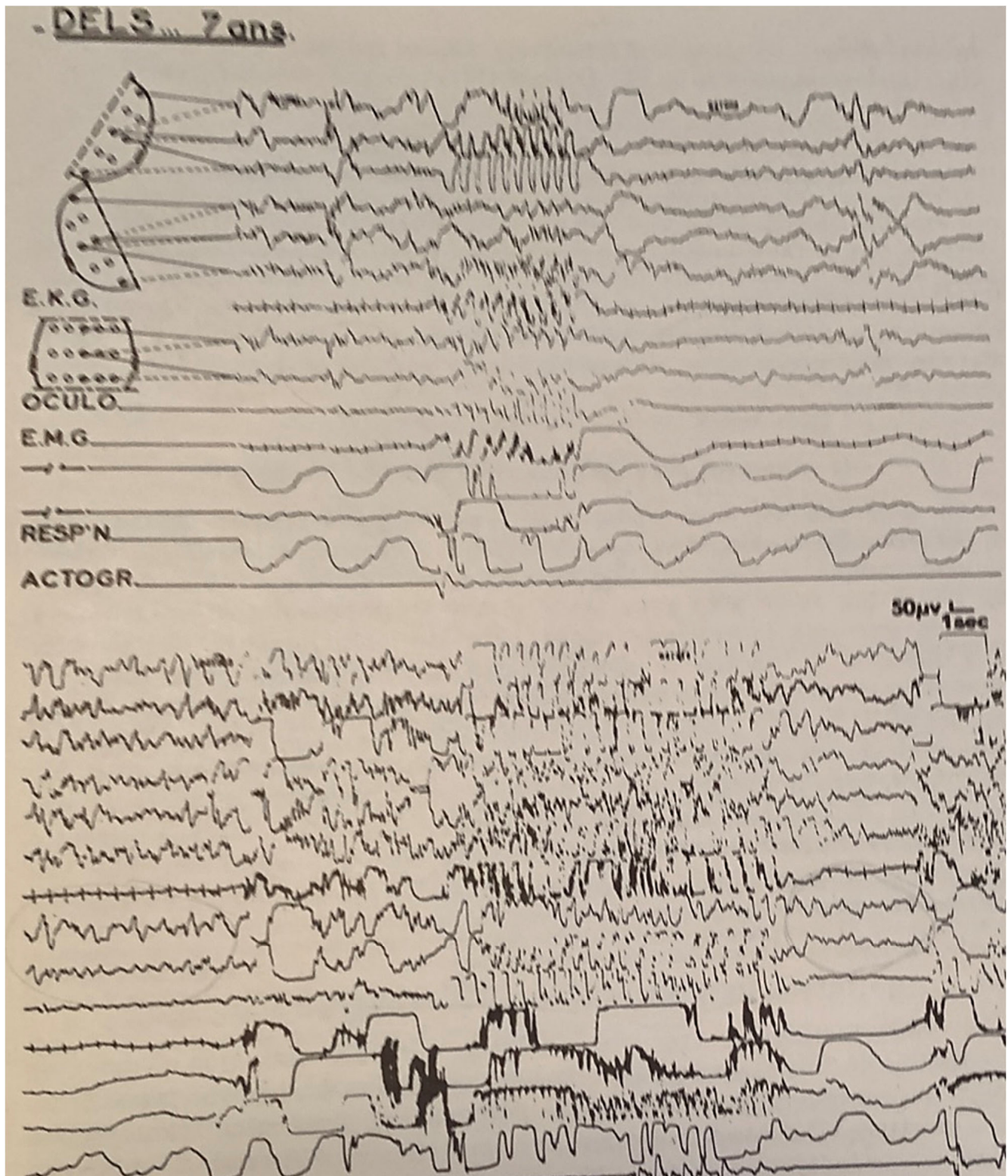


Figure 22. Extreme jactatio capitis nocturna in a 7-year old child with total body movements. Such body rocking recurs at about 2–3/second and is recorded in all EEG channels and as artifacts on the EKG channel. Sometimes self-injury may occur with violent body rocking. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

17. Bruxism

Bruxism is tooth grinding which may recur during sleep and is usually seen in persons with dental problems. It may produce audible noise that is disagreeable to a sleeping partner. When tooth grinding episodes occur there may be some lightening of sleep patterns. Reding et al. in 1968 contributed a psychophysiological study of nocturnal tooth-grindings [70]. This was followed by a 1977 review of the literature by Glaros and Rao [71]. Richmond et al. in 1984 described bruxism in institutionalized mentally retarded patients [72]. Ware and Rugh in 1988 studied destructive bruxism and its relationships to sleep stages [73]. The induction of rhythmic tooth grinding by temporal lobe seizures was published by Meletti et al. [74]. More recently, significant contributions have been made by the Université de Montreal group led by Gilles Lavigne who has studied the physiology of bruxism [75] and its relationships to respiratory sleep medicine [76].

18. Sexsomnia

Sexsomnia is the most recently described parasomnia. It was first reported in 2003 and was based upon a case series of 11 patients by Colin Shapiro and colleagues who also gave the condition its name [77]. The authors noted that the context of these NREM sleep events was different from that of sleepwalking in that the arousal was more prominent, the motor activities were more restricted, and that recall of dream mentation is usually present. These attacks may take a large variety of expressions during sleep including masturbation, sexual fondling, and attempting or having intercourse with a sleeping partner. During sexsomnia, the person has no awareness of the events. Andersen et al. in 2007 published an important series of patients with sexsomnia [78].

Rodriguez et al. [79] in 2014 reported four cases with events occurring 2–3 times per week and were the first to note an association with obstructive sleep apnea. In 2014 Ingravallo et al. [80] reported 18 cases of sexual behavior in sleep and 18 cases with sleep-related violence. The sexual behaviors in sleep ranged from sexual touching to rape and the sleep-related violent behaviors included homicide or attempted homicide. Their article recommended the creation of an international multidisciplinary consensus for the forensic evaluation of sleep-related violence and sexual behaviors during sleep. Unfortunately to date, this consensus has not been created. Schenck in 2015 provided an excellent review of sexsomnia [81]. He noted that they almost always were associated with confusional arousals from deep slow wave sleep. Schenck added the symptom of sexual vocalizations to those of masturbation, fondling, intercourse, or attempted intercourse which had already been reported.

A very interesting single case report was published in 2016 by Soca et al. in which sexsomnia was classified as a subtype of the NREM sleep parasomnias [82]. This patient had a form of overlap parasomnia disorder in which there were superimposed episodes of sleepwalking, sleep-related eating disorder, sleep talking, REM sleep behavior disorder, and sexsomnia. Sayin and Schenck in 2019 published a fascinating review of the biological bases of pleasure that appear to be involved in sexsomnia. [83] These included the involvement of dopaminergic neurons in the ventral tegmental area, the release during sexual activity of dopamine, oxytocin and norepinephrine, and the activation of the anterior cingulate gyrus, the insula, the hippocampi and the hypothalamus.

19. Epileptic Seizures

Two excellent edited books cover the topic of epileptic seizures and sleep, as well as sleep deprivation [84,85] The earliest polysomnographic recordings of epileptic seizures and sleep were reported by Janz in 1962 [86] who focused on the circadian aspects. There are several inter-relationships between epileptic events and sleep/wake rhythms. Seizures show different patterns in sleep according to whether they are generalized or focal in origin. Seizures that are generalized from the start almost always occur in NREM sleep. Partial and focal seizures occur mainly in REM sleep. Epileptic seizures cause awakening during sleep, and therefore, induce sleep deprivation. So, a vicious cycle may occur with seizures causing

sleep deprivation and sleep deprivation facilitating seizures. Three years later Gastaut et al. reported the occurrence of tonic generalized seizures in children with Lennox-Gastaut syndrome [87]. These all occurred in NREM sleep and sometimes presented as a form of sleep-related status epilepticus. Clinically the seizures consist of increased axial muscle tone during the episode with, very rarely, brief generalized myoclonus at the end of the attack (Figure 23).

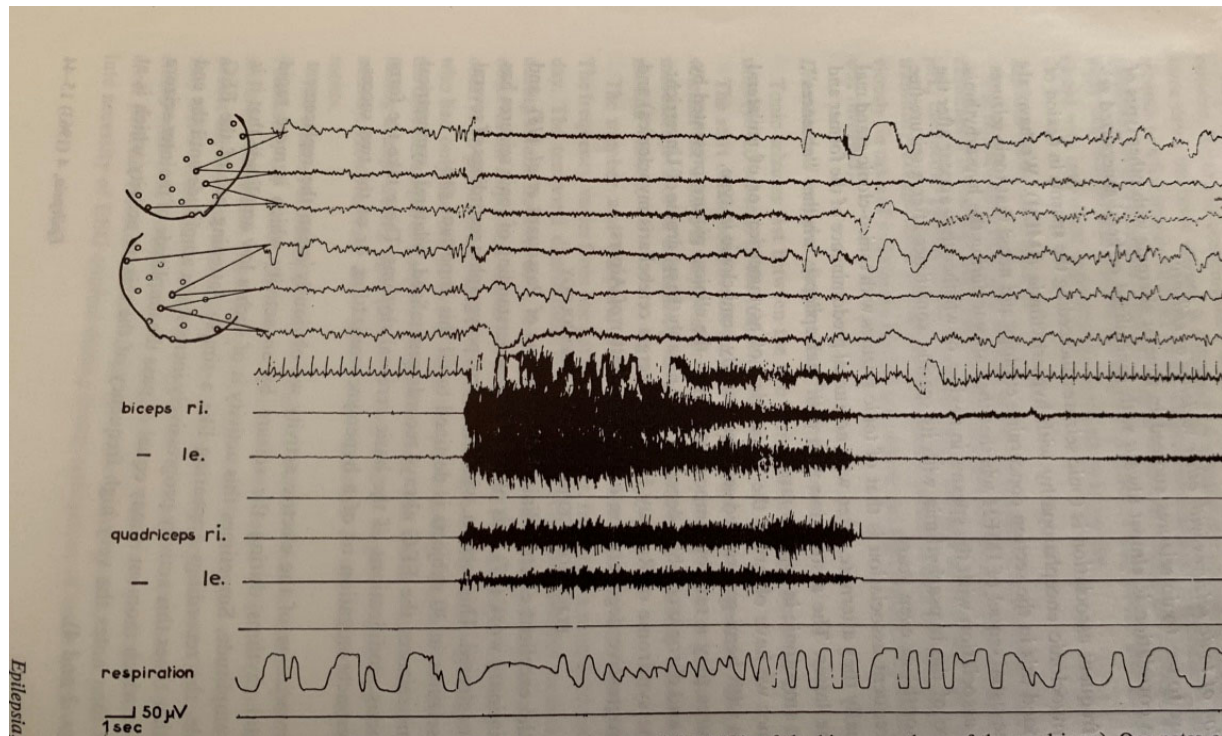


Figure 23. A generalized tonic epileptic seizure. In the EEG there is a brief polyspike and wave complex followed by a flattening during which there is increased muscle tone in the arms and legs without any myoclonus. There is a minor amount of post-ictal EEG slowing. (Reproduced with a permission from Gastaut H, Roger J, Ouachi S, et al. An electro-clinical study of seizures of tonic expression. *Epilepsia* 1963; 4: 15–44; published by Wiley-Blackwell Publishing).

Seizures may also be induced by awakening. This is the case for certain generalized tonic-clonic seizures (Figure 24) and in Ramsay Hunt syndrome (cerebellar symptoms associated with photosensitive epilepsy) by generalized myoclonic seizures with intact consciousness (Figure 25).

A specific form of epilepsy is autosomal dominant frontal lobe epilepsy [88], which is characterized by tonic postures in NREM sleep (Figure 26). The findings that there is often a version of the head and eyes, and that only rarely is an epileptic discharge recorded on the scalp are both highly suggestive of the epileptic focus being in the supplementary motor area on the mesial surface of the frontal lobe. Occasionally, as Scheffer et al. have reported [89], frontal lobe epilepsy is misdiagnosed as a sleep disorder. Phillips et al. have clarified the molecular cause of the seizures which involves genetic heterogeneity and the evidence for a second locus at 15q24 [90]. There is quite a broad spectrum of clinical expression for autosomal frontal lobe epilepsy which has been documented by Provini et al. [91].

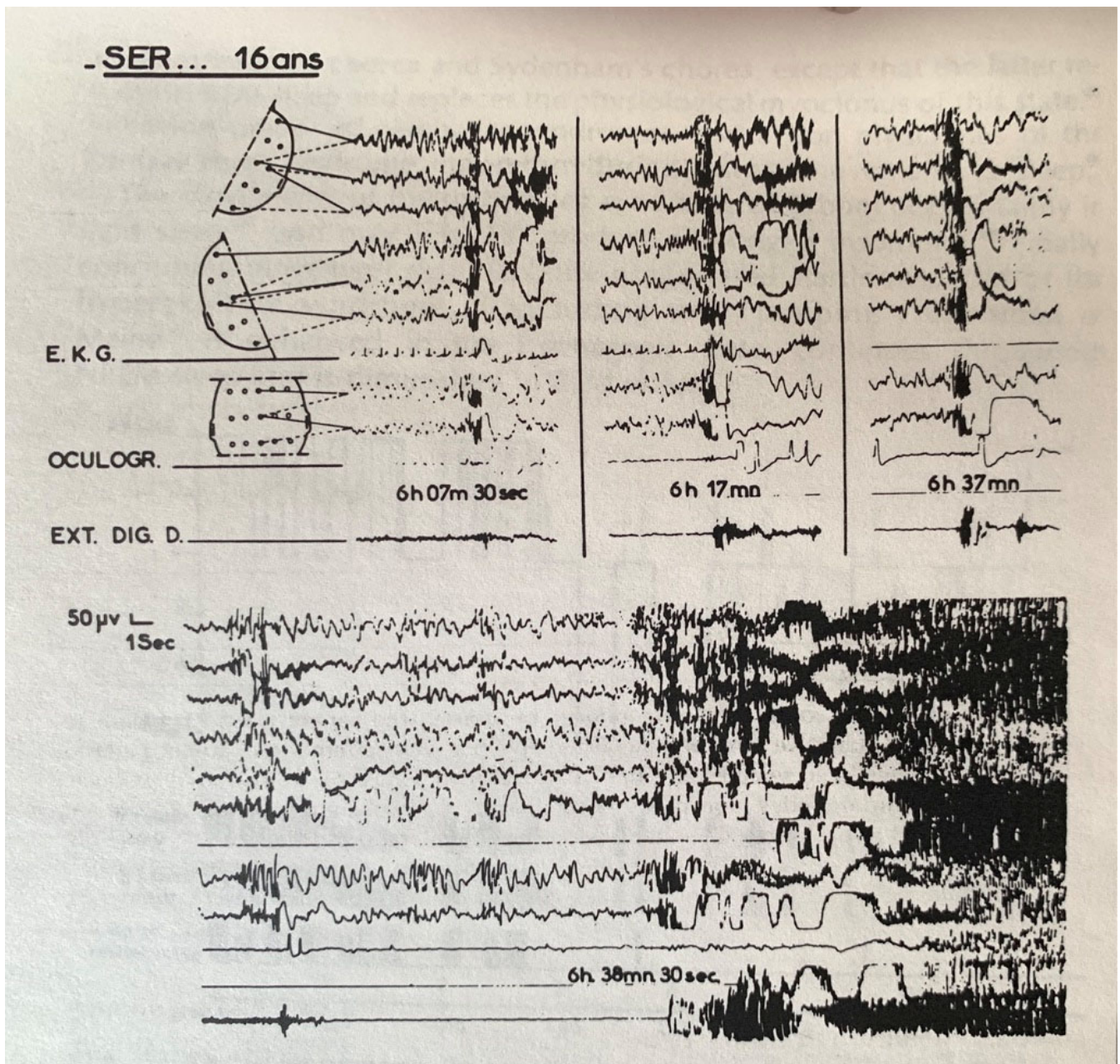


Figure 24. A generalized tonic-clonic seizure induced by awakening. In the first segments selected there are polyspike and wave discharges, these increase in frequency and culminate in a generalized tonic clonic seizure at 6 h 38 min in the morning. (Adapted from Gastaut, H. and Broughton, R.: A clinical and polygraphic study of episodic phenomena during sleep. Academic Address, in: Wortis, J. (Ed.), Recent Advances in Biological Psychiatry, New York, Plenum Press, 1965, pp. 197–221).

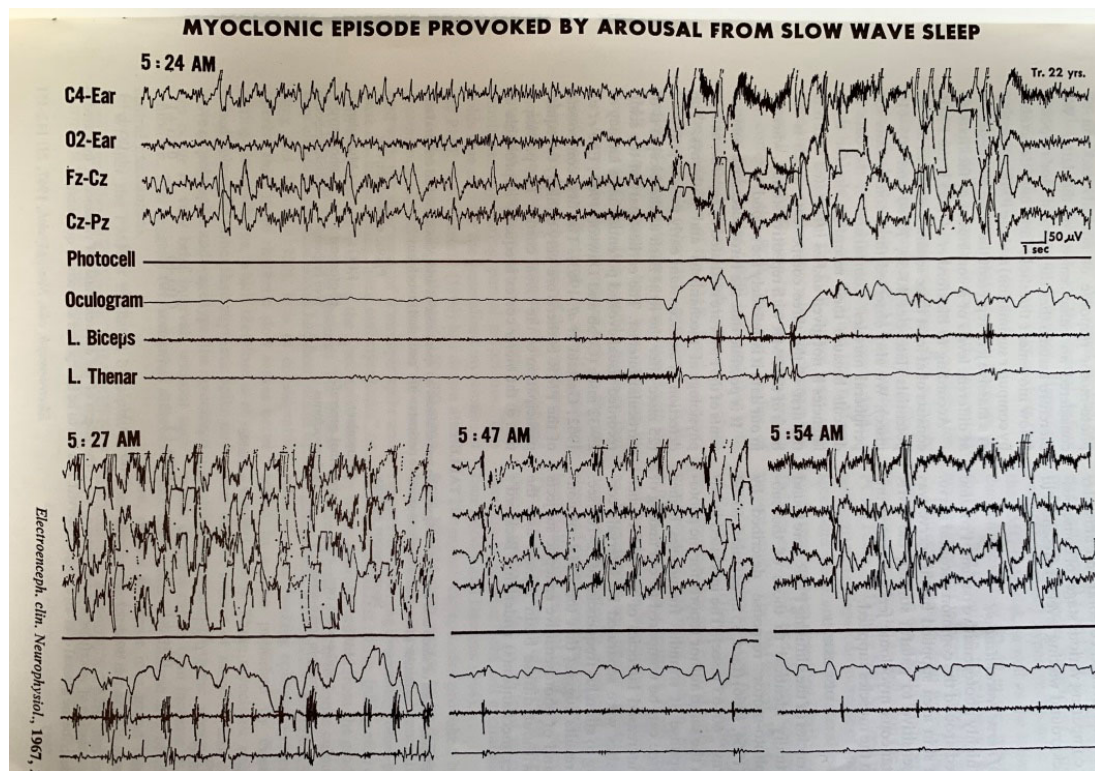


Figure 25. A generalized myoclonic seizure in a 22-year-old patient with Ramsay Hunt syndrome (photosensitive generalized epilepsy associated with cerebellar symptoms). He awakens at 5 h 24 min and the figure shows the continuation of the myoclonic epilepsy at different times after the awakening. The patient was awake during the seizure and shows eye movements due to looking around the room. (Reproduced with permission from Meier-Ewert, K. and Broughton, R: Photomyoclonic response of epileptic and nonepileptic subjects during wakefulness, sleep and arousal. *Electroenceph. Clin. Neurophysiol.* 23: 142–151, 1967, published by Elsevier Publishing).

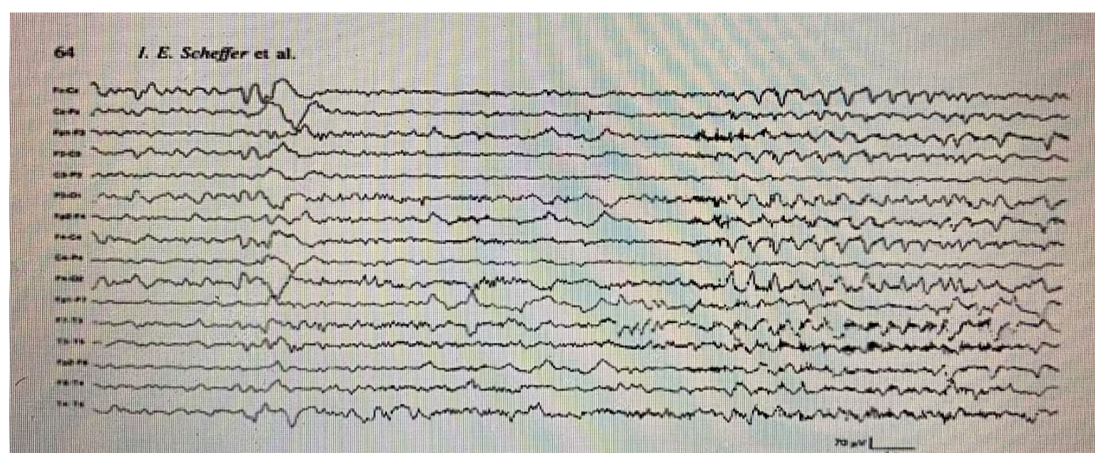


Figure 26. Hereditary frontal lobe epilepsy. The figure shows a seizure in NREM sleep which is of frontal lobe origin. The patient deviates the head and eyes and lifts the arm. The majority of patients with hereditary frontal lobe epilepsy do not show EEG discharges. This is consistent with the belief that the epileptogenic focus is in the supplementary motor area on the mesial face of the frontal lobe. (Adapted from Scheffer et al. in the article “Autosomal dominant nocturnal frontal lobe epilepsy: a distinctive clinical disorder. *Brain* 1995, 118: 61–73. Permission to reproduce granted by Oxford University Press).

20. Periodic Limb Movement Disorder (PLMs)

These movements represent the most common movement disorder in sleep. Early studies were published by Lugaresi and colleagues. They are often an incidental finding on limb EMGs. PLMs last some 0.5–10 s and occur in bursts separated by 5–90 s. Four or more such movements must occur in a row to be classified as PLMs. The movements may recur throughout the night in both light NREM and REM sleep. The individual movements consist mainly of dorsiflexion of the foot with flexion at the level of the knee and thigh. They are stereotypic and at various times may involve the left or right leg or both legs together. The kicking movements may disturb the sleep of the sleeping partner. Occasionally PLMs spill over into wakefulness. If so, the patient often seems to be emotionally distressed and has a shortened attention span. The long duration of the movements and the long inter-movement intervals suggest that any comparison with sleep-related epilepsy is remote.

21. Hypnagogic Foot Tremor and Alternating Leg Activation

Hypnagogic foot tremor consists polygraphically of bursts of 0.3–4 Hz activity in leg EMGs. The patient normally does not complain of the involuntary movements that can occur in presleep wakefulness or light stages of NREM sleep. In alternating leg movements, there is an alternation of leg movements with a similar frequency of 0.5–3 Hz and a similar duration of 100–500 msec. The movements can occur in all stages of sleep. There are few publications on the disorder.

22. Propriospinal Myoclonus

In propriospinal myoclonus jerks occur at sleep onset. Patients do not have PLM or restless leg syndrome (RLS). It has been found that the origin of propriospinal myoclonus resides in the propriospinal tracts of the spinal cord. There are no published specific scoring rules for propriospinal myoclonus at sleep onset. They can also occur during wakefulness prior to sleep onset and in general, do not include rhythmic myoclonic features.

23. Restless Leg Syndrome (RLS)

In restless leg syndrome (also called Ekbom syndrome) the patient has an uncontrollable urge to move the legs which reduce the dysesthesia often described as a “crawling” sensation in the legs. Moving the legs temporarily reduces the symptoms which then recur. RLS typically appears in patients about 40 years of age. Treatment is typically very difficult and unsuccessful. The cause of RLS in most cases is not known. The condition is sometimes present in a family tree so that, in such cases, there is a definite genetic component to the physiopathogenesis. When very severe the condition causes disability with considerable daytime sleepiness due to the severe sleep onset insomnia. Severe depression may develop in this chronic sleep disorder.

24. Exploding Head Syndrome

Exploding head syndrome is a rare abnormal sensory perception during sleep in which a person experiences unreal noises that are loud and of short duration during falling asleep or waking up. The noises may be frightening, typically occur only occasionally, and are not a serious health concern. Patients may be helped by reassurance and reducing daytime anxiety [92–94].

25. Excessive Fragmentary Myoclonus (EFM)

Broughton et al. [95] were the first authors to publish on the condition which they named excessive fragmentary myoclonus. EFM presents movements resembling muscle fasciculations and are arrhythmic muscle twitches that do not cause any large movements but may give rise to twitches of the thumbs or finger. The fasciculations are widely disseminated and may be recorded on the limbs and also on the face. They resemble the twitch potentials that characterize REM sleep. They in fact can occur with intensity in all stages of sleep (Figure 27). Most of the recorded movement potentials last around 150 msec. At least 5 twitches per

minute must be recorded in NREM sleep to meet EFM criteria [96]. EFM is often associated with an increased level of daytime sleepiness. There have been associations with obstructive and central sleep apnea, narcolepsy, insomnia, periodic limb movements, neurodegenerative diseases including Parkinson's disease and peripheral nerve dysfunction [97] (Figure 28). The wide number of associate diseases supports the belief that EFM is not a specific sleep disorder nor a specific polysomnographic finding. The possibility that EMF is a normal variant has not been ruled out. However, it can be associated with marked daytime sleepiness [98].

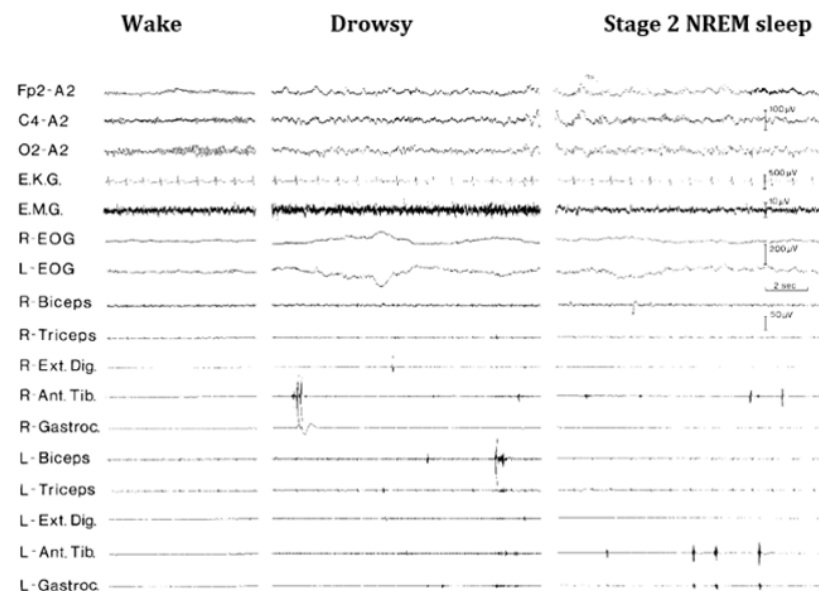


Figure 27. Excessive fragmentary myoclonus in a 42-year-old complaining of daytime fatigue and drowsiness. The myoclonus which appears clinically as fasciculations occurs in drowsiness (middle PSG sample) and NREM sleep (right PSG sample) occurs bilaterally and independently in different areas of the body. (Permission to reproduce granted by Cambridge University Press).

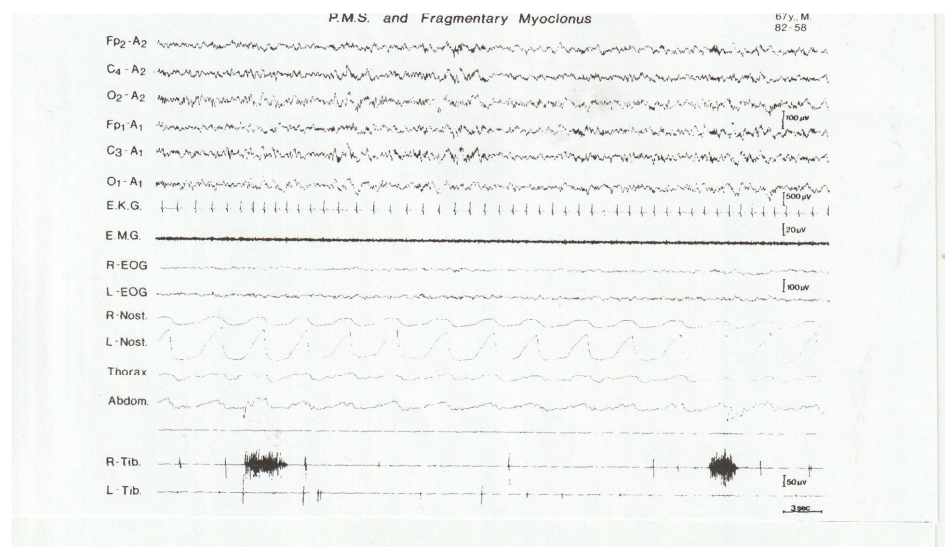


Figure 28. Excessive fragmentary myoclonus associated with periodic leg movements in his right leg in a 56-year-old patient complaining of excessive daytime sleepiness. (Reproduced with permission from Broughton R, Tolentino MA and Krelina M. Excessive fragmentary myoclonus in NREM sleep: a report of 38 cases. *Electroenceph clin Neurophysiol* 1985; 61:123–133; published by Cambridge University Press).

26. Sleep-Related Leg Cramps

Leg cramps may occur in sleep and be extremely painful with intense contraction of the gastrocnemius muscles. The cause is often unknown but at times is related to excessive daytime vigorous exercise or a degree of dehydration. The strong contraction of the gastrocnemius muscles causes plantar flexion of the feet. Although it is almost always the calf muscles that are involved, rarely muscles in the feet or the thighs may also cramp. Massaging the cramps is often counterproductive as it increases severe pain. Slow progressive dorsiflexion of the feet stretches the muscles and usually causes relief. Strategies to avoid sleep-related cramps include: drinking lots of water to avoid dehydration, stretching the legs regularly, changing the sleeping position and avoiding heavy or tucked-in bedding.

27. The Future

In closing, it is interesting to consider what will be the future of our diagnoses and understanding of the parasomnias. There will, no doubt, be much progress. This will include more sophisticated resolution and analysis of the EEG data associated with the parasomnias which will develop greater localization using dipole and other analyses. Brain imaging will no doubt be greatly improved in resolution and in neurochemical specificity. We will also have a better understanding of the roles of biological rhythms including circadian, ultradian and infradian ones. As is true for medicine in general there will no doubt be broad and intensive developments in molecular genetics. There will also be progress in the analysis of the neural mechanisms of innate behavior patterns following the lead of Tassinari, Silvestri and others in this domain. There has been little research on parasomnias occurring in so-called primitive cultures and this should be corrected. One also expects that the relationships of sleep and biological rhythms synchronized with solar, lunar and other cosmological events will be further clarified. We, earthlings, are, after all, only a very small part of the cosmos that patterns our biological rhythms. Perhaps above all one hopes that there will be much greater research on the socio-economic and quality of life impacts of the parasomnias. In any event in future years, it will be very interesting to learn what new parasomnias have been discovered.

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References

1. Batini, C.; Fressy, J.; Gastaut, H. A propos du sommeil chez le sujet normal. *Rev. Neurol.* **1962**, *106*, 216–218.
2. Batini, C.; Fressy, J.; Gastaut, H. Étude du reflexe cutané-plantaire au cours des différentes phases du sommeil. *Rev. Neurol.* **1963**, *108*, 172.
3. Fressy, J. *Apport de la Polygraphie Dans L'étude Polygraphique de la Phase Dite Des Mouvements Oculaires au Cours du Sommeil Chez L'homme. Thèse de Troisième Cycle*; Université de Marseille: Marseille, France, 1963; 102p.
4. Orwell, G. *Such Were the Joys*; Harcourt: New York, NY, USA, 1945.
5. Ditman, K.S.; Blinn, K.A. Sleep levels in enuresis. *Amer. J. Psychiatr.* **1955**, *111*, 913–920. [[CrossRef](#)] [[PubMed](#)]
6. Saint-Laurent, J.; Batini, C.; Broughton, R. A polygraphic study of nocturnal enuresis in the epileptic child. *Electroenceph. Clin. Neurophysiol.* **1963**, *15*, 904.
7. Broughton, R.; Gastaut, H. Polygraphic studies of enuresis nocturna. *Electroenceph. Clin. Neurophysiol.* **1964**, *16*, 625.
8. Broughton, R.; Gastaut, H. Further polygraphic sleep studies of enuresis nocturna (intravesicular pressure). *Electroenceph. Clin. Neurophysiol.* **1964**, *16*, 626.
9. Gastaut, H.; Broughton, R. A clinical and polygraphic study of episodic phenomena during sleep. The Sakel Academic Lecture. *Recent Adv. Biol. Psychiatr.* **1965**, *7*, 197–223.

10. Hawkins, D.R.; Scott, J.; Thresher, G. *Sleep Patterns in Enuretic Children*; Association for the Psychophysiological Study of Sleep: Washington, DC, USA, 1965.
11. Ritvo, E.R.; Ornitz, W.M.; Gottlieb, F. Arousal and non-arousal enuretic events. *Am. J. Psychiatr.* **1969**, *126*, 77–84. [[CrossRef](#)]
12. Gambi, D.; Pinto, F.; Torriolio, M.G. Night Sleep in Enuretic Children. In *Sleep 1974*; Levin, P., Koella, W.P., Eds.; Karger: Basel, Switzerland, 1975; pp. 92–97.
13. Kales, A.; Kales, J.D.; Jacobson, A.; Humphrey, F.J.; Soldatos, C.R. Effects of Imipramine on Enuretic Frequency and Sleep Stages. *Pediatrics* **1977**, *60*, 431–436. [[CrossRef](#)]
14. Mikkelsen, E.J.; Rapoport, J.L.; Gruenau, S. Sleep patterns in childhood enuresis. In *Sleep Research*; Chase, M.H., Mitler, M.M., Walter, P.I., Eds.; Brain Information Service/Brain Research Institute: Los Angeles, CA, USA, 1978; p. 242.
15. Muellner, S.R. Development of urinary control in children: A new concept in cause, prevention and treatment of primary enuresis. *J. Am. Med. Assoc.* **1960**, *172*, 1256–1261. [[CrossRef](#)]
16. Di Perri, R.; Meduri, M. L'enuresi notturna ulteriori elementi in tema di diagnostic strumentale. *Riv. Neurol.* **1972**, *27*, 22–27.
17. Di Perri, R.; Meduri, M. A polygraphic approach to the study of enuresis nocturna. In *Sleep 1974*; Levin, P., Koella, W.P., Eds.; Karger: Basel, Switzerland, 1975; pp. 413–416.
18. Bradley, W.E. Electroencephalography and Bladder Innervation. *J. Urol.* **1977**, *118*, 412–414. [[CrossRef](#)]
19. Bakwin, H. Enuresis in twins. *Am. J. Dis. Child.* **1971**, *121*, 222–225. [[CrossRef](#)] [[PubMed](#)]
20. von Gontard, A.; Heron, J.; Joinson, C. Family history of nocturnal enuresis and urinary incontinence: Results from a large epidemiological study. *J. Urol.* **2011**, *185*, 2303–2307. [[CrossRef](#)] [[PubMed](#)]
21. Petit, D.; Pennestri, M.-H.; Paquet, J. Childhood sleepwalking and sleep terrors: A longitudinal study of prevalence and familial aggregation. *JAMA Pediatr.* **2015**, *169*, 653–658. [[CrossRef](#)]
22. Tassinari, C.A.; Rubboli, G.; Gardella, E. Central pattern generators for a common semiology in frontal-limbic seizures and parasomnias: A neuroethologic approach. *Neurol. Sci.* **2006**, *3*, 225–232.
23. Gastaut, H.; Batini, C.; Broughton, R. Études électroencéphalographiques des phénomènes épisodiques non-épileptiques au cours du sommeil. In *Le Sommeil Normal et Pathologique*; Fischgold, H., Ed.; Masson: Paris, France, 1965; pp. 215–236.
24. Jacobson, A.; Lehmann, D.; Kales, A. EEG recordings during somnambulistic sitting and walking. *Science* **1965**, *148*, 975–977. [[CrossRef](#)]
25. Broughton, R. Sleep disorders: Disorders of arousal? Enuresis, sleepwalking, sleep terrors and confusional states of arousal are not in “dreaming sleep”. *Science* **1968**, *159*, 1070–1078. [[CrossRef](#)]
26. Gastaut, H.; Broughton, R. Paroxysmal events and certain phases of sleep. *Percept. Mot. Ski.* **1963**, *109*, 333–334.
27. Montplaisir, J.; Petit, D.; Pilon, M. Does sleepwalking impair daytime vigilance? *J. Clin. Sleep Med.* **2011**, *7*, 219. [[CrossRef](#)]
28. Kales, J.D.; Kales, A.; Soldatos, C.R.; Chamberlin, K.; Martin, E.D. Sleepwalking and night terrors related to febrile illness. *Am. J. Psychiatry* **1979**, *136*, 1214–1215. [[CrossRef](#)] [[PubMed](#)]
29. Broughton, R. Confusional sleep disorders: Interrelationship with memory consolidation and retrieval in sleep. In *Hinks Memorial Lectures: A Triune Concept of the Brain and Behaviour*; Maclean, P., Boag, P.J., Campbell, D., Eds.; University of Toronto Press: Toronto, ON, Canada, 1973; pp. 115–127.
30. Bassetti, C.; Weder, B. SPECT during sleepwalking. *Lancet* **2000**, *356*, 484–485. [[CrossRef](#)]
31. Yellowless, D. Homicide by a somnambulist. *J. Ment. Sci.* **1878**, *24*, 451–458. [[CrossRef](#)]
32. Broughton, R.J.; Shimizu, T. Sleep, and violence: A medical and forensic challenge. *Sleep* **1995**, *18*, 727–730. [[CrossRef](#)] [[PubMed](#)]
33. Broughton, R.; Cartwright, R.; Doucette, D.; Edmeads, J.; Edwardh, M.; Ervin, F.; Orchard, B.; Hill, R.; Turrell, G.; Billings, R. Homicidal Somnambulism: A Case Report. *Sleep* **1994**, *17*. [[CrossRef](#)]
34. Pedley, T.A.; Guilleminault, C. Episodic nocturnal wanderings responsive to anticonvulsant drug therapy. *Ann. Neurol.* **1977**, *2*, 30–35. [[CrossRef](#)] [[PubMed](#)]
35. Zadra, A.; Pilon, M.; Montplaisir, J. Polysomnographic diagnosis of sleepwalking: Effects of sleep deprivation. *Ann. Neurol.* **2008**, *63*, 513–519. [[CrossRef](#)]
36. Desjardins, P.M.; Carrier, J.; Lina, J.-M.; Fortin, B.M.; Gosselin, N.; Montplaisir, J.; Zadra, A. EEG Functional Connectivity Prior to Sleepwalking: Evidence of Interplay Between Sleep and Wakefulness. *Sleep* **2017**, *40*. [[CrossRef](#)]
37. Gaudreau, H.; Joncas, S.; Zadra, A.; Montplaisir, J. Dynamics of slow-wave activity during the NREM sleep of sleepwalkers and control subjects. *Sleep* **2000**, *23*, 755–760. [[CrossRef](#)]
38. Lecendreux, M.; Bassetti, C.L.; Dauvilliers, Y.; Mayer, G.; Neidhart, E.; Tafti, M. HLA and genetic susceptibility to sleepwalking. *Mol. Psychiatry* **2003**, *8*, 114–117. [[CrossRef](#)]
39. Jones, E. *On the Nightmare*; Hogarth: London, UK, 1949.
40. Gastaut, H.; Batini, C.; Broughton, R. Étude électroencéphalographique des manifestations paroxystiques non-épileptiques au cours du sommeil nocturne. *Rev. Neurol.* **1964**, *110*, 309.
41. Fisher, C.; Byrne, J.; Edwards, A. A psychophysiological study of nightmares. *J. Psychoanal. Assoc.* **1970**, *18*, 747–782. [[CrossRef](#)] [[PubMed](#)]
42. Fisher, C.; Kahn, E.; Edwards, A. A psychological study of nightmares and night terrors: Physiological aspects of the stage 4 sleep terror. *J. Nerv. Ment. Dis.* **1973**, *157*, 75–98. [[CrossRef](#)] [[PubMed](#)]
43. Fisher, C.; Kahn, E.; Edwards, A. A psychophysiological study of nightmares and night terrors: The suppression of stage 4-night terrors with diazepam. *Arch. Gen. Psychiatr.* **1973**, *28*, 252–259. [[CrossRef](#)] [[PubMed](#)]

44. Tassinari, C.; Mancina, D.; Bernardina, B.; Gastaut, H. Pavor nocturnus of non-epileptic nature in epileptic children. *Electroencephalogr. Clin. Neurophysiol.* **1972**, *33*, 603–607. [\[CrossRef\]](#)
45. Gastaut, H.; Dongier, M.; Broughton, R. Electroencephalographic and clinical study of diurnal and nocturnal anxiety attacks. *Electroenceph. Clin. Neurophysiol.* **1964**, *17*, 475.
46. Hartmann, E. *Dreams and Nightmares: The Origin and Meaning of Dreams*; Perseus Publishing: New York, NY, USA, 2000; p. 328.
47. Hartmann, E.; Russ, D.; Van Der Kolk, B.; Falke, R.; Oldfield, M. A preliminary study of the personality of the nightmare sufferer: Relationship to schizophrenia and creativity? *Am. J. Psychiatry* **1981**, *138*, 794–797. [\[CrossRef\]](#)
48. Boydon, A.D.; Pott, M.; Starks, P.T. An evolutionary perspective on sleep terrors. *Evol. Med. Public Health* **2018**, *20*, 100–105. [\[CrossRef\]](#)
49. Marc, C. *De la Folie*; Ballière: Paris, France, 1840; pp. 658–668.
50. von Gudden, H. Die physiologische und pathologische Schlaftrunkenheit. *Arch. Für Psychiatr.* **1905**, *40*, 989. [\[CrossRef\]](#)
51. Ohayon, M.; Mahowald, M.W.; Leger, D. Are confusional arousals pathological? *Neurology* **2014**, *83*, 834–841. [\[CrossRef\]](#)
52. Roth, B.; Nevsimanova, S.; Rechtschaffen, A. Hypersomnia with “Sleep Drunkenness”. *Arch. Gen. Psychiatry* **1972**, *26*, 456–462. [\[CrossRef\]](#) [\[PubMed\]](#)
53. Feltin, M.; Broughton, R. Differential effects of arousal from slow wave versus REM sleep. *Psychophysiology* **1968**, *5*, 231.
54. Broughton, R.; Shimizu, T. Dangerous behaviours by night. In *Forensic Aspects of Sleep*; Shapiro, C., Smith, A.M., Eds.; John Wiley & Sons: London, UK; New York, NY, USA, 1997; pp. 65–83.
55. Broughton, R.; Billings, R.; Cartwright, R. Homicidal somnambulism: A case report. *Sleep* **1994**, *1*, 253–264.
56. Schenck, C.H.; Bundlie, S.R.; Ettinger, M.C. Chronic behavior disorders of human REM sleep: A new category of parasomnia. *Sleep* **1986**, *9*, 293–308. [\[CrossRef\]](#)
57. Jouvet, M.; Delorme, F. Locus coeruleus et sommeil paradoxal. *CR Soc. Biol.* **1965**, *159*, 895–899.
58. Snyder, S. Isolated sleep paralysis after time-zone changes (jetlag). *Chronobiologica* **1983**, *10*, 377–379.
59. Everett, H.C. Sleep paralysis in medical students. *J. Nerv. Ment. Dis.* **1963**, *136*, 283–287. [\[CrossRef\]](#)
60. Penn, N.E.; Kripke, D.F.; Scharff, J. Sleep Paralysis Among Medical Students. *J. Psychol.* **1981**, *107*, 247–252. [\[CrossRef\]](#)
61. Roth, B.; Bruhova, S.; Berkova, L. Familial sleep paralysis. *Schweiz. Arch. Neurol. Neurochir. Psychiatr.* **1968**, *102*, 321–330.
62. Nan’no, H.; Hiskikawa, Y.; Koida, H. A neurophysiological study of sleep paralysis in narcoleptic patients. *Electroenceph. Clin. Neurophysiol.* **1970**, *28*, 382–390. [\[CrossRef\]](#)
63. Dahlitz, M.; Parkes, J.D. Sleep paralysis. *Lancet* **1993**, *341*, 406–407. [\[CrossRef\]](#)
64. Lugaresi, E.; Cirignotta, F. Hypnogenic paroxysmal dystonia: Epileptic seizure or new syndrome? *Sleep* **1981**, *4*, 129–138. [\[CrossRef\]](#) [\[PubMed\]](#)
65. Lugaresi, E.; Cirignotta, F.; Montagna, P. Nocturnal paroxysmal dystonia. *J. Neurol. Neurosurg. Psychiatry* **1986**, *49*, 375–380. [\[CrossRef\]](#) [\[PubMed\]](#)
66. Oswald, I. Sudden Bodily Jerks on Falling Asleep. *Brain* **1959**, *82*, 92–103. [\[CrossRef\]](#) [\[PubMed\]](#)
67. Broughton, R.J. Some important underemphasized aspects of sleep onset. In *Sleep Onset Mechanisms*; Ogilvie, R., Harsh, J., Eds.; American Psychological Association: Arlington, VA, USA, 1994; pp. 19–36.
68. Silvetti, R.; Walters, A.S. Rhythmic movements in sleep disorders and epileptic seizures during sleep. *Sleep Sci. Pract.* **2020**, *4*, 5. [\[CrossRef\]](#)
69. Tassinari, C.A.; Gardella, E.; Cantalupo, G.; Rubboli, G. Relationship of Central Pattern Generators with Parasomnias and Sleep-Related Epileptic Seizures. *Sleep Med. Clin.* **2012**, *7*, 125–134. [\[CrossRef\]](#)
70. Reding, G.R.; Zepelin, H.; Robinson, J.E. Nocturnal tooth-grinding: All-night psychophysiological studies. *J. Dent. Res.* **1968**, *47*, 787–797. [\[CrossRef\]](#)
71. Glaros, A.G.; Rao, S.M. Bruxism a critical review. *Psychol. Bull.* **1977**, *84*, 767–781. [\[CrossRef\]](#)
72. Richmond, G.; Rugh, J.D.; Dolfi, R.; Wasilewsky, J.W. Survey of bruxism in an institutionalized mentally retarded population. *Am. J. Ment. Defic.* **1984**, *88*.
73. Ware, J.C.; Rugh, J. Destructive bruxism: Sleep stage relationship. *Sleep* **1988**, *11*, 172–181. [\[CrossRef\]](#)
74. Meletti, S.; Cantalupo, G.; Volpi, L.; Rubboli, G.; Magaudo, A.; Tassinari, C.A. Rhythmic teeth grinding induced by temporal lobe seizures. *Neurology* **2004**, *62*, 2306–2309. [\[CrossRef\]](#) [\[PubMed\]](#)
75. Lavigne, G.J.; Khoury, S.; Abe, S.; Yamaguchi, T.; Raphael, K. Bruxism physiology and pathology: An overview for clinicians. *J. Oral Rehabilitation* **2008**, *35*, 476–494. [\[CrossRef\]](#) [\[PubMed\]](#)
76. Mayer, P.; Heinzer, R.; Lavigne, G. Sleep bruxism in respiratory medicine practice. *Chest* **2016**, *149*, 262–271. [\[CrossRef\]](#) [\[PubMed\]](#)
77. Shapiro, C.; Tralanovic, N.N.; Fedoroff, J.P. Sexsomnia—A new parasomnia? *Can. J. Psychiatry* **2003**, *48*, 311–317. [\[CrossRef\]](#) [\[PubMed\]](#)
78. Andersen, M.L.; Poyares, D. Sexsomnia: Abnormal sexual behavior during sleep. *Brain Res. Rev.* **2007**, *56*, 271–282. [\[CrossRef\]](#)
79. Rodriguez, H.A.; Iranzo, A.; Giaz, C. Sexsomnia: Parasomnia associated with sexual behaviour during sleep. *Neurologia* **2014**, *29*, 146–152.
80. Ingravallo, F.; Poli, F.; Gilmore, E.V.; Pizza, F.; Vignatelli, L.; Schenck, C.H.; Plazzi, G. Sleep-Related Violence and Sexual Behavior in Sleep: A Systematic Review of Medical-Legal Case Reports. *J. Clin. Sleep Med.* **2014**, *10*, 927–935. [\[CrossRef\]](#)
81. Schenck, C.H. Update on sexsomnia, sleep related sexual seizures, and forensic implications. *NeuroQuantology* **2015**, *13*, 519–541. [\[CrossRef\]](#)

82. Soca, R.; Kennan, J.; Schenck, C.H. Parasomnia overlap disorder with sexual behaviors during sleep in a patient with obstructive sleep apnea. *J. Clin. Sleep Med.* **2016**, *12*, 1189–1191. [[CrossRef](#)]
83. Sayin, U.; Schenck, C.H. Neuroanatomy and neurochemistry of sexual desire, pleasure, love and orgasm. *Sexus* **2019**, *4*, 907–946.
84. Serman, B.; Shouse, M.N.; Passouant, P. *Sleep and Epilepsy*; Academic Press: New York, NY, USA, 1982.
85. Degen, R.; Rodin, E.A. *Epilepsy, Sleep and Sleep Deprivation*; Elsevier: Amsterdam, The Netherlands, 1991.
86. Janz, D. The grand mal epilepsies and the sleeping-waking cycle. *Epilepsia* **1962**, *3*, 69–109. [[CrossRef](#)] [[PubMed](#)]
87. Gastaut, H.; Roger, J.; Ouachi, S. An electro-clinical study of seizures of tonic expression. *Epilepsia* **1963**, *3*, 69–109. [[CrossRef](#)] [[PubMed](#)]
88. Scheffer, I.E.; Bhatia, K.P.; Lopes-Cendes, I.; Fish, D.R.; Marsden, C.D.; Andermann, E.; Andermann, F.; Desbiens, R.; Keene, D.; Cendes, F.; et al. Autosomal dominant nocturnal frontal lobe epilepsy. *Brain* **1995**, *118*, 61–73. [[CrossRef](#)] [[PubMed](#)]
89. Scheffer, I.E.; Bhatia, K.P.; Lopes-Cendes, I. Autosomal dominant frontal epilepsy misdiagnosed as sleep disorder. *Lancet* **1994**, *343*, 515–517. [[CrossRef](#)]
90. Phillips, H.; Scheffer, I.; Crossland, K.; Bhatia, K.; Fish, D.; Marsden, C.; Howell, S.; Stephenson, J.; Tolmie, J.; Plazzi, G.; et al. Autosomal Dominant Nocturnal Frontal-Lobe Epilepsy: Genetic Heterogeneity and Evidence for a Second Locus at 15q24. *Am. J. Hum. Genet.* **1998**, *63*, 1108–1116. [[CrossRef](#)]
91. Provini, F.; Plazzi, G.; Tinuper, P.; Vandi, S.; Lugaresi, E.; Montagna, P. Nocturnal frontal lobe epilepsy: A clinical and polygraphic overview of 100 consecutive cases. *Brain* **1999**, *122*, 1017–1031. [[CrossRef](#)]
92. Sharpless, B.A.; Zimmerman, J.A. Exploding Head Syndrome. In *Unusual and Rare Disorders: A Handbook for Clinical Practice and Research*; Sharpless, B.A., Ed.; Oxford University Press: Oxford, UK, 2016; pp. 39–52. [[CrossRef](#)]
93. Sachs, C.; Svanborg, E. The exploding head syndrome: Polysomnographic recordings and therapeutic suggestions. *Sleep* **1991**, *14*, 263–266. [[CrossRef](#)]
94. Kallweit, U.; Khatami, K.; Bassetti, C.L. Exploding head syndrome—More than “snapping of the brain”. *Sleep Med.* **2008**, *9*, 589. [[CrossRef](#)]
95. Broughton, R.; Tolentino, M.A.; Krelina, M. Excessive fragmentary myoclonus in NREM sleep: A report of 38 cases. *Electroencephalogr. Clin. Neurophysiol.* **1985**, *61*, 123–133. [[CrossRef](#)]
96. Lins, O.; Castonguay, M.; Dunham, W.; Nevsimalova, S.; Broughton, R. Excessive Fragmentary Myoclonus: Time of Night and Sleep Stage Distributions. *Can. J. Neurol. Sci.* **1993**, *20*, 142–146. [[CrossRef](#)]
97. Sobreira-Neto, M.A.; Pena-Pereira, M.A.; Tavares, E.S. Excessive fragmentary myoclonus in patients with Parkinson’s disease: Prevalence and clinic-polysomnographic profile. *Sleep Breath* **2015**, *19*, 997–1002. [[CrossRef](#)] [[PubMed](#)]
98. Nepozitek, J.; Sonka, K. Excessive fragmentary myoclonus: What do we know? *Prague Med. Rep.* **2017**, *118*, 5–13. [[CrossRef](#)] [[PubMed](#)]