ACADEMIC PRESS Available online at www.sciencedirect.com



Consciousness and Cognition 11 (2002) 377-390

Consciousness and Cognition

www.academicpress.com

# Sleep and dream suppression following a lateral medullary infarct: A first-person account

J. Allan Hobson\*

Laboratory of Neurophysiology, Massachusetts Mental Health Center, Harvard Medical School, 74 Fenwood Road, Boston, MA 02115, USA

Received 18 September 2001

# Abstract

Consciousness can be studied only if subjective experience is documented and quantified, yet first-person accounts of the effects of brain injury on conscious experience are as rare as they are potentially useful. This report documents the alterations in waking, sleeping, and dreaming caused by a lateral medullary infarct. Total insomnia and the initial suppression of dreaming was followed by the gradual recovery of both functions. A visual hallucinosis during waking that was associated with the initial period of sleep and dream suppression is described in detail. Since the changes in sleep and their recovery are comparable to results of animal experiments, it can be concluded that damage to the medullary brain stem causes extreme but short-lived alterations in conscious state and that substantial recovery occurs even though the damage to the brain stem endures.

© 2002 Elsevier Science (USA). All rights reserved.

# 1. Introduction

Interest in the effects of stroke on the subjective experience of sleep and dreaming has recently been rekindled by the work of Mark Solms on suppression of dreaming by cortical and subcortical forebrain structures (Solms, 1997). Solms is

1053-8100/02/\$ - see front matter © 2002 Elsevier Science (USA). All rights reserved. PII: S1053-8100(02)00011-9

<sup>\*</sup> Fax: 1-617-739-2224.

E-mail address: allan\_hobson@hms.harvard.edu.

quite right in asserting that the clinicopathological approach is an integral part of the investigation of the brain basis of dreaming and his interesting findings have reopened debate about the relative importance of brain stem and forebrain structures in the neurogenesis of dreaming (Hobson, Pace-Schott, & Stickgold, 2000; Solms, 2000).

This report of suppression of dreaming following infarction of the lateral medulla is meant to contribute to that debate but, by focusing on sleep generally and the way in which the subsequent changes in sleeping and dreaming evolved in parallel during the recovery and rehabilitation periods, it is also intended as a broader call for the close study of a wider range of poststroke effects which have been ignored in the neurological literature and which have a strong bearing on both sleep psychophysiology and sleep medicine.

The following account emphasizes the immediate and virtually total suppression of sleep that occurred in the acute poststroke phase, an effect that closely parallels the objective findings in cats with experimental medullary lesions (Batini, Moruzzi, & Palestini et. al., 1959; Moruzzi, 1972; Hobson, 1965). It also documents in detail the distinctive visual hallucinatory phenomena that were present during that insomniac period. Such observations are necessarily subjective but, because they are associated with localized neuropathology, they may help us begin to construct models of visual hallucinosis in relation to a brain-based theory of dreaming (Hobson et al., 2000).

The parallels with brain function in the poststroke evolution of sleep and dreaming during the recovery phase and their further normalization during the rehabilitation phase are also described. These long-term changes echo the important point, emphasized in the basic science of work of Villablanca (1968), that postlesion plasticity is continuous in both the neurobiological and behavioral domains.

## 2. Clinical history

The patient-author is a 67-year-old married father of five who has devoted his professional life to the scientific study of sleep and dreaming and especially to the link between neurophysiology and psychological phenomenology. His motivation to make a detailed account of his stroke experience was greatly aided by the constant attendance and support of his wife, Dr. Lia Silvestri, a clinical neurologist and sleep medicine expert. Dr. Silvestri recorded the author's comments and notes on a daily basis until he was able to operate a hand-held tape recorder. Because it seems artificial to report personal experiences in the third person, I henceforth use the firstperson voice.

# 2.1. Stroke onset

On Saturday, February 2, 2001, while traveling in Europe with my wife, I experienced the sudden onset of vertigo and sweating, which was particularly intense on the right side of the head. The sensation of the world spinning around me (from left to right and in a right-side downward-tilted orbit) could be relieved by resting my head on the café table where we had just finished breakfast—and by closing my eyes.

Incorrectly supposing that these effects might be due to the sleep deprivation incurred on the previous day's overnight flight from Boston to paris to Nice, I waited for the acute symptoms to subside and walked back to my hotel. Upon reclining, I immediately felt better, slept for 3 h and enjoyed a snack supper in my room. That night, after watching TV, I slept fitfully but enjoyed the room service breakfast the next morning. However, while returning the breakfast table to the hallway, I noticed a distinct ataxia, which told me that my troubles were not over. In fact, they had just begun.

By noon of February 3rd, a full 24 h after the initial attack of vertigo, I began to experience great difficulty swallowing. This problem was compounded by the massively increased volume of salivation which suddenly began. The combination made me feel like I was drowning in my own secretions. Together with the vertigo and ataxia, these two signs clearly suggested an emerging infarction process localized to the lateral medullary brainstem, a diagnosis my wife had already suspected and for which she had already put me on aspirin and nicergoline (a vasodilator).

# 2.2. Hospitalization

In the emergency ward of the Princess Grace Hospital in Monaco, an EKG revealed atrial fibrillation and suggested th at my brain stem could be undergoing infarction due to embolic thrombosis of the right vertebral artery near its posterior inferior cerebellar branch. A negative CAT scan ruled out intracerebral hemorrhage but three subsequent MRIs showed a clear, discrete infarct of the right lateral medulla in the area of the distribution of the posterior inferior cerebellar artery centered on the R restiform body, the adjacent medullary tegmentum, and the overlying cerebellum (see Figs. 1a and b).

Formal neurological examinations on Days 3 and 10 revealed the following signs of Wallenberg's syndrome: (1) cerebellar ataxia most severe in the right leg but also affecting the right arm; (2) vestibular deficits inclusing nystagmus and postural instability: especially with my feet together and eyes closed, I showed a tendency to fall forward and to the right; (3) Horner's syndrome with anisocoria R > L; (4) loss of sensation and paraesthesias R side of face; (5) lip droop R; (6) paralysis of pharyngeal muscles R; (7) paralysis of R vocal cord; and (8) mildly decreased sensibility to pain and markedly decreased sensibility to temperature change of L side of body below neck. A neurological consultant declared, in French, that I was a classic case (Hörnstein, 1974a,b).

There was no indication of damage to cranial nerves XI or XII. A surface EEG was normal and visual field testing were unremarkable. An ultrasound study of the cerebral vascular revealed blockage of the R vertebral artery.

Repeated electrocardiograms demonstrated persistent atrial fibrillation. An attempt to obtain a transesophageal EKG on Day 8 was unsuccessful because the benzodiazepine premedication caused a psychotic reaction: convinced that I was being attacked by soldiers with fierce medieval weapons, I forcefully repulsed both the physician and his probe.



Fig. 1. (a) Sagittal view of brain reveals extensive damage in vestibular and reticular medullary tegmentum. Cerebellar damage is also visible. (b) Coronal section through center of lesion in medulla shows its broad extent and documents the overlying cerebellar damage more clearly.

# 3. Hospital course

# 3.1. Acute phase (Days 1–10)

I was on the acute coronary care unit of the Princess Grace Hospital, where the internal medicine, nursing, and ancillary clinical care were all excellent. Because I speak French fluently I was comfortable in this setting and because my wife and I were confident that no definitive treatment was available anywhere in the world we decided to stay in Monaco until all the medical, logistical, and financial arrangements were made for my return to Boston. As one of the attendings said to me on rounds, "We are going to give you the best possible care and see what happens."

My care consisted of iv fluids, a nasogastric tube, and a bladder catheter. Medications directed at the heart failed to convert my fibrillation. Anticoagulants (heparin followed by coumadin) were used to decrease the risk of embolization.

## 3.2. Recovery phase (Days 10–40)

Once I was able to sit up and get out of bed and my cardiovascular signs had stabilized, I was transferred to the cardiology unit and began to make specific plans to go back to Boston.

Following return to the United States (by Air Ambulance on February 22, 2001), I was hospitalized in the stroke unit of Brighan and Women's Hospital, where I stayed until March 15th. The most importance therapeutic interventions that were made were as follows: installation of a gastric tube (G-tube or PEG) on March 5, 2001, which improved delivery of foodstuffs to tie stomach and stopped my weight loss (from 200 to 170); and electrical cardioversion on March 12, 2001, which re-instated a normal sinus rhythm. To reduce the risk of recurrent emboli or thrombosis, I was continued on coumadin/heparin anticoagulant therapy; an antibiotic treatment of a urinary tract infection led to removal of the catheter.

Although my neurological process was steady, I continued to have difficulty swallowing, salivation was persistent, and I could not maintain an upright posture without feeling dizzy and falling to the right. These symptoms persisted well after I was transferred to Spaulding Rehabilitation Hospital. Aspiration was thus a constant threat and I ultimately developed consolidation in the lower lobe of my left lung. On two occasions, 1 had chills, headache, and photophobia, for which I was placed an antibiotic medication which may have prevented the development of frank pneumonia.

## 3.3. Rehabilitation phase (Days 40–100)

At the Spaulding Rehabilitation Hospital, where I stayed from March 15th to April 15th, I was taught to walk, first with a walker and then with a cane. Now that I am at home, I navigate with no external support but I must assume a broad-based gait and change direction or level of ambulation very carefully to avoid the residual effects of lateropulsion. Heroic efforts to help me learn to swallow were un successful until about 8 weeks poststroke, the time at which most neurologists predict recovery. But even then swallowing was not so much recovered as relearned. (And this distinction still holds as I write this report 16 weeks poststroke.) Instead of swallowing as I previously did, I now force the food down through my pharynx by moving my tongue suddenly and vigorously backward. This usually clears the oropharynx of food and liquid which otherwise tends to pool there constantly threatening (and sometimes occasioning) aspiration and violent coughing. In order to avoid aspiration I actively regurgitate the softened food and try again to bypass what must be a still-paralytic dead spot in my peristaltic oryngopharyngeal musculature. Building on my progress with this technique I was gradually able to reduce the G-tube feedings and to have it removed on Monday, April 30, 2001.

Using this technique, I was able to make the following triumphant entry in my journal on April 9, 2001:

During the past week, my progress has been very impressive, especially in the domain of eating. I now have a really full diet, which is fully transferable to the outside world. It consists of juice for breakfast. I can drink juice very easily if I lay at a 45-degree angle. It goes right down. Probably because gravity helps it stay back. Then, I have an 8-ounce cup of vanilla yogurt with sugar, which is delicious. I then go to scrambled eggs, which I press with a fork so as to break up the lumps; the whole thing is held together with lashings of ketchup. It is really quite good. No coffee, no tea, no toast as yet. But that will come later.

Because my stroke was localized to the brain stem, have been spared the indignity of aphasia and other cognitive impairments of forebrain damage. This means that I have been able to communicate with a wide circle of well-wishers from all corners of the world and all the nooks and crannies of my life, an enormous psychological benefit.

Since the earliest days, in Monaco, I have been able to edit manuscripts (such as our *Behavioral and Brain Sciences* monograph), books, papers, and grant applications. Now I can write again and am contemplating my next major book project. Because my motor difficulties are mainly dynamic and occur only on assuming an upright posture, I function well while seated. As well as working at my desk at home, that includes driving my car, so I am now able to go in to the lab and confer with my colleagues, attend seminars, and pursue important hospital projects.

To an outside observer, my recovery may appear complete. Some of my friends even prefer my now-leaner lines! But at 16 weeks poststroke, I am still aware of how far I have to go to regain confident mobility, normal swallowing, and better control of salivation. And no neurologist is promising relief of the right facial anesthesia or the left somatic analgesia and temperature sensation loss. I am nonetheless happy to be sleeping, dreaming, and engaging in other gratifying visceral behaviors even if all of these functions are distinctly altered—possibly permanently.

# 4. Sleep and dream changes

#### 4.1. Sleep suppression in the acute poststroke period

Over the first 10 days of my hospitalization, in Monaco, I not only manifested many of the characteristic features of Wallenberg's syndrome but also suffered from total

insomnia, a symptom not previously reported in the neurological literature. Because the lesion is so precisely localized—and because the sleep suppression so clearly resembles the insomnia observed in cats with mediopontine transections—I thought it might be useful to describe these subjective effects. Here is the verbatim transcript of a note dictated to my wife on February 8, 2001, Day 6 of my hospitalization:

Time is exquisitively attenuated in the hospital because there is nothing to do except try to keep from drowning in my own saliva or falling off the bed due to vertigo. So I lay for hours in my bed, flat on my back with my eyes closed. The worst time is the night because I am alone, usually from 7 PM to 7 AM and because I cannot sleep a wink. The usual side position does not help; I'm just awake, my mind working actively in the dark all night long. Attempts to watch TV are frustrated by the banal programming and my double vision. Toward morning, at first around three o'clock and subsequently as late as 7, I suddenly feel irresistibly sleepy but this is not normal sleep onset. Instead I am drawn into a hallucinatory visual world of great complexity which only occasionally bears a faint semblance to real dreaming.

When I was acutely and severely insomniac, I recalled the dramatic arousal effects caused by mediopontine pretrigeminal transections of the brainstem which we have made in Lyons in 1963 to test Moruzzi's hypothesis that there was a synchronizing mechanism in the medullary brainstem. Cats with such lesions remained fully alert with strongly activated EEG's for about 10 days (Batini et al., 1959; Moruzzi, 1972; Hobson, 1965). Whether the current results bear on that phenomenon, they clearly indicate that major sleep disturbances can be precipitated by brainstem strokes.

Integrating the acute sleep changes with my neurological signs it seems reasonable to propose that it was damage to the pontomedullary brainstem that caused my insomnia. Among the relevant structures affected are the oculomotor (VI) and pontine reticular formation preoculomotor neurones, which resulted in temporary diplopia; the trigeminal (V), which resulted in a persistent R facial hemianesthesia; the facial (VII), which resulted in temporary right mouth droop; the vestibular (VIII), which resulted in the persistent vertigo and lateropulsion; the glossopharyngeal (IX), which led to the persistent difficulty swallowing; and the vagus nuclei (X), which led to R vocal cord paralysis and, together with damage to the nucleus ambiguous, probably contributed to the cluster of autonomic dysfunctions that I experienced: the persistent excessive salivation, constipation, and arterial hypotension.

# 4.2. Dream suppression and visual hallucinosis

It goes without saying that if sleep is totally suppressed, as it was for the first 10 days in my case, then dreaming cannot occur. During this acute poststroke period, I experienced the abnormal visual percepts described below. As far as dreaming itself is concerned, my journal notes suggest that even at  $3\frac{1}{2}$  months poststroke, I had not yet fully recovered the vivid dreamlife that I experienced prior to my stroke.

Between Days 1 and 10 I could visually perceive a vault over my supine body immediately upon closing my eyes. The vault resembled the bottom of a swimming pool but the gunitelike surface of the vault could be not only aqua, but also white or beige and, more rarely, engraved obsidian or of a gauzelike nature mixed with ice or glass crystals.

There were three categories of formed imagery that appeared on these surfaces. In the first category of *geologic forms* the imagery tended to be protomorphic and crude

but often gave way to the more elaborate structures of category two *inanimate sculptural forms*. The most amusing of these (which occurred on the fourth night) were enormous lucite telephone/computers. But there were also tables and tableaux in which the geologic forms sometimes took unusual and bizarre shapes. One that I recall is a TV-set-like representation of a tropical landscape. In category three, the most elaborate forms have *human anatomical elements*, including long swirling flesh, columns that metamorphosed into sphincters, nipples, and crotches, but these were never placed in real bodies. In fact whole body forms almost never emerged. Instead I saw profiles of faces and profiles of bodies which were often inextricably mixed with penises, noses, lips, eyebrows; torsos arose out of the sculptural columns of flesh and sank back into them again.

The most fully realized human images include my wife, featuring her lower anatomy and (most amusingly) a Peter Pan-like Robert Stickgold and two fairies enjoying a bedtime story. While visual disturbances are quire common in Wallenberg's syndrome, they have only been reported to occur in waking with eyes open. Blurring of vision (which I had), and the tendency of objects to appear to move called oscillopsia (which I did not have), are attributed to the disturbed oculomotor and vestibular physiology.

The severe suppression of my sleep continued for about 2 weeks despite some relief provided by antihistamines, which were begun on Day 8. On February 15, 2001, I wrote:

Among the strange psychic phenomena are the continued sense that normal sleep and dreaming are impossible. Last nigh I had a image of the antihistamines working on anterior hypothalamic sleep mechanisms but the lower brain system could not be definitively turned over.

#### 4.3. Vertigo and lateropulsion

During the immediate poststroke phase, my hallucinatory experiences also involved the vestibular domain as has been reported by others (Werner, Ciuffreda, & Tannen, 1989). These vertigo variants consist of the vivid impression of being launched through space. They were never associated with the protomorphic visual images described above but were instead accompanied by their own imagery. They could be precipitated by forced ambulation (when I was awake in the daytime) or they might occur spontaneously (while I was lying motionless in bed at night trying to sleep). The first, and most violent, episode occurred on the first night of my hospitalization, in Monaco. Here is my journal account of that episode which occurred immediately after vomiting my breakfast:

I suddenly felt as if the bed were rotated 90° in a counterclockwise direction and catapulted downward toward the front of the hospital and into the sea. The illusion that I was actually moving at very high speed for at least 100 m through space was so completely convincing—and so terrifying that I said to myself, "So this is what death is like." To my amazement, these sensations abated as rapidly as they had begun but they made a very lasting impression on me.

Less dramatic variations on this experience occurred three times during the acute phase and then abated. They were sometimes associated with sneezing attacks. I have always been a convulsive sneezer (as was my father) but ever since my stroke the clusters of up to five sneezes can occur at any time of day (whereas they were previously preferentially matinal) and they lack the inspiratory wind-up instead consisting of short explosive entirely expiratory phases.

In the acute phase poststroke, and for a much longer time thereafter (10–30 days), I was prone to the illusion of falling—or even hurtling—to the right when wellmeaning attempts were made to help me ambulate. In these cases, after I had assumed an erect posture, I suddenly felt that I was being propelled through space parallel to the ground and was incapable of righting myself despite my frantic and powerful efforts. These involuntary movements are called lateropulsions in the neurological literature, where they are attributed to vestibular (Dietrich & Brandt, 1992; Solomon, Galetta, & Liu, 1995) and/or cerebellar damage (Helmchen, Straube, & Buttner, 1994). Such attacks became less frequent and less intense but could still be provoked until about Day 30. Since then I have had a milder but still annoying tendency to lurch to the right and this can cause falling, a dangerous outcome since I am still on anticoagulant medication.

## 4.4. Partial recovery of dreaming and sensorimotor functions

Between Days 8 and 15 I began to have more dreamlike imagery associated with my fitful sleep but during this period there was no REM sleep dreaming (elaborate, rapidly evolving plot; movement; bizarreness; dynamic emotion; etc.).

The following are two descriptions of such imagery from Day 8:

I have the sensation of having paper all over my body which cannot be removed because there is a computer program that instantiates equal and opposite commands each time I try to remove one of them. This fruitless struggle goes on for hours and gradually replaces the alternating state of lucid thinking and lush visual hallucinations of the preceding 6 nights.

I see a female monkey whose head and paws are made out of silver foil containing brown flower petals. The monkey mama is floating in the air in front of me like a comical beatific protector.

On Day 10 I had a brief human character perception with the beginnings of dreamlike narrative structure:

Ed (Schott, my coworker in Boston) is filling in brain drawings for people who want to know how the brain works and he has made a huge quilt out of about thirty or forty drawings all of which were in color and connected together in a scrupulous way. I said "Look Ed, you don't need to do all this for the other people, why don't you keep this one for us and just send them a copy?

Such fragmentary mentation, which is formally similar to my dreams, was unsustained and the loss of typical REM sleep dreaming persisted for 30 days! On Day 31, at the Brigham and Women's Hospital in Boston, I had the following

experience:

Before I woke up I had a prolonged interesting dream experience in which I imagined that I was being treated by a woman or two women (possibly nurses) who were giving me magical fabric to put on my skin. This fabric released a kind of coolness and pleasurable sensation over my skin which I relished and I kept enjoying it as the fabric was moved from place to place on my hands, my chest, my belly, and my face. It was a little bit like the coolness that emanates from the ice packs (that I used in waking to count facial paraesthesias) but very gentle and not like any other sensation that I have ever experienced. Nor was this dream like any other I ever had because it just continued on and on and on in this wonderful analgesic almost narcotically pleasant mode.

I remember wondering what the mechanism of the heat-released cool pleasure was and I couldn't come up with any reasonable explanation. I knew that I couldn't reason well but even this recognition still didn't lead me to think that I was dreaming. Instead I attributed it to warmth of women who had obviously made an important discovery on how to comfort people. I would guess this went on for about thirty or forty minutes until I gradually woke up and then I could go back and forth for about five minutes in an out of this dreamy state. But by five o'clock, five-thirty in the morning I was too wide awake to do anything else but remember it well, contemplate it, and vow to tape record its contents.

To this report I added an interpretation: This "dream" means that something is changing my brain and I hope it means that my spontaneous sleep mechanisms are recovering to some degree. I hasten to emphasize, however, that this was unlike any REM sleep dream I ever had.

My first elaborate and sustained dream occurred on Day 38, when I was already beginning to walk and exercise at the Spaulding Rehabilitation Hospital. Here is my account:

It took place in a foreign country, which I thought might be Yugoslavia or Hungary. Lia and I were on a trip and we were crossing a bridge with a lot of other people. It was a high arched bridge of a medieval sort. The bridge suddenly separated from the land and became a boat swimming across a very small river to get to a village on other side. We had plans to stay in an old-fashioned inn.

There was already some discomfort and difficulty finding each other as we got the boat near to the shore. I caught glimpses of her. She was talking to someone else, a man. At one point, either before or just after we got off the boat, I noticed that she had given or sold to him a half-inch bit used with my large brace to drill holes in wood in Vermont. I was very surprised and somewhat hurt by this. I noticed also that the bit had been used to make a very perfect hole in the shoulder bag, which the man wore. It was a shoulder bag very much like mine.

In the dream, Lia explained to me that she had sold the drill but would give me the money. It still seemed to me odd that she would give a stranger one of my most precious tools without asking me. I was feeling very vexed and apprehensive. We got on the land, we walked around looking for the inn and we were separated from each other on several occasions. During one of the times that we were together, she made it clear to me that she needed to have a secret life.

When I was asking her about this man, it was clear that she meant that she needed to be free to have an affair with him if she wanted to. I found that very odd and very disquieting and tried to express my concerns. When we finally got to what appeared to be the inn, there was a strange scene in which she was again difficult to find. But I found her in what looked like a kitchen and she was preparing to cook some food, which struck me as odd, since this was such a flimsy excuse. I asked her when she would be finished and she looked at her watch and she said 45 min, to which I agreed, knowing that this was all the time she would need to make love with whichever stranger she had selected.

I then walked around the inn, which had a very peculiar structure. On one side, there was a row of seats roofed over as in a theater and beside each of the seats were exotic bouquets of flowers. I walked down from one level of this improbable architecture to the next, finally getting to the bottom and wondering in which room Lia and her lover were situated and how I could get to

the window to see it. But by going down, I was of course going down from the bedroom level as if to avoid my curiosity. I wandered all the way around the other side of the building, admiring the ancient medieval architecture, all of which was quite exotic and entirely impossible. When I came back to the place where I thought I might find Lia, and there indeed was her coat, the brown coat with the fur trim and hood that she wears so often and that I like so much but no sign of her and no sign of whatever man she might be with.

This was a REM sleep dream if there ever was one. The word count of 603 gives this assumption a very high probability. I cite the report in full to make several points. (1) The plot was long, sustained, and richly detailed. I was convinced, by the intensity of hallucinatory imagery that I was awake and never questioned that assumption despite abundant evidence that it could not be true given the bizarreness detailed below. My thought processes were thus markedly it impaired: my self-reflective awareness, judgement, and logic were poor as is typical I of REM sleep dreams. (2) The consistent and strong emotional binding of these disparate elements is what gave the dream its obvious meaning: in my state of impaired health I was worried about losing my most important companion, strongest supporter, my wife, and my lover. (3) The classic bizarreness of REM sleep dreams was present: the setting was unspecified (it could have been Hungary or Yugoslavia but was not either one), key personnel were unidentified, the behavior was odd (was the man of whom I was jealous a total stranger?), the drill bit and the hole in my shoulder bag make only Freudian sense, my wife's cooking was unlikely in a hotel, the architecture was incongruous; and so on.

Since recording this dream, on Day 38, I have been sleeping increasingly well, almost too well. At home from Day 75 onward, I now go to bed with my 5-year-old twins at about 9–10 PM and sleep until 7 AM and while I do have some fragmentary dream recall, I have not remembered another dream as well as this one. The possibility thus arises that I am now hypersomnolent and unable to recall my now-normal REM sleep dreams because I sleep so deeply. This hypothesis, like the hypothesis that my initial insomnia was stroke dependent, is testable.

## 5. Discussion

## 5.1. Methodologidal issues

Subjective reports are always suspect but sleep science has learned to deal with them, in part, by correlating specific aspects of the reports with anatomical and physiological data. The common occurrence of strokes makes this approach both compelling and informative, especially if our misgivings about subjective data can be overcome. One way to do this is to record the subjective experience of stroke victims more aggressively, more thoroughly, and more systematically.

To accomplish this goal, neurologists need to be sensitized to the research significance and technique of subjective data collection. Family members are almost always available—and willing to help—as in my case. Hand-held tape recorders are inexpensive and can be used to augment dictated reports.

In the absence of such affirmative action, the patient is reduced to a set of easily localizable signs of CNS damage and classic case status. That Wallenberg himself did

not inqure about sleep and dreaming is surprising because he was so widely respected as a very modest and thorough clinician (Pearce, 2000). In the 50 articles that I examined describing cases like mine, there was lot one mention of the phenomena I describe: the initial severe insomnia, the suppression of dreaming, the gradual but incomplete recovery of sleep and recovery of dreaming, and the subsequent hypersomnia. Even the one previous first-person account of the effects of lateral medullary infarction makes no mention of any change in sleep or dreaming (Werner et al., 1989).

Several facts of modern medical life add to neurological short-sightedness in making such descriptions difficult to obtain. Because hospitals and doctors are so overburdened, hurried, and procedure-oriented and because hospital stays are so short, attention to psychological detail is likely to decline further rather than increase. And yet patients, like myself, have very little to do in hospitals except introspect! We thus need to take advantage of the availability of such privileged self-observation. The first goal of this report is thus to show that it can be done.

## 5.2. Clinicopathological correlations

As already asserted, it seems very likely that the subjective experience reported here was a direct result of damage to my brain stem. And judging from the clinical picture, that damage was far more extensive in a functional sense than the lesion seen on the MRI. In other words, the initial suppressive effects of sleep and dreaming cannot be ascribed only to the lesion itself but also to the temporary dysfunction of adjacent structures.

This logic, which has great importance for all sleep lesion studies, including those of basic sleep research, runs as follows: The infarction (lesion) killed neurones in the restiform body, the cerebellum, the vestibular nucleus, the trigeminal nucleus, the glossopharyngeal nucleus, the vagus nucleus, and the nucleus ambiguous. It is this damage that produced the acute poststroke symptoms: vertigo, sweating, ataxia, hypersalivation, and dysphagia. And it is this damage which, being permanent, is responsible for the residual symptoms: ataxia, hypersalivation, dysphagia, facial anesthesia and paresthesia, and the contralateral temperature and pain insensitivity.

But these lesioned structures are probably not solely responsible for the sleep loss, the suppression of dreaming and the triggering of hallucinoid visual imagery. These effects are more likely due in part to temporary functional deficits in the pontomedullary reticular formation. Although unlesioned, these structures were clearly impaired during the acute poststroke period as evidenced, for example, by the diplopia, which signals malfunction of the oculomotor and preocular pontomedullary reticular neurones. Once these adjacent pontomedullary reticular structures regained functional integrity, sleep and dream functions also recovered.

Can we understand these effects in mechanistic detail?

With regard to the visual hallucinatory syndrome, we can offer only a provisional model of explanation. Whenever the balance of neuromodulatory systems is upset—whether by sleep deprivation, therapeutic drugs, drug abuse, or organic insult to the brain—visual hallucinosis can be produced. This means that the brain-mind is capable not only of producing formed visual imagery off line as it does in dreaming but can do so simply by tuning out external inputs (in my case, eye closure was enough to

set the imagery in motion) and thus altering the balance between external and internal stimuli (Hobson, 2001; Hobson et al., 2000).

As far as the sleep and dream suppression are concerned, we know that the pontomedullary reticular systems are exquisitely state-dependent. The normal architecture of sleep and especially REM sleep are, in turn, dependent on a complex and precise orchestration of activity in the reticular formation and the structures with which it interacts (including the locus coeruleus, the raphé nuclei, and the hypothalamic sleep switch, which mediates circadian control of the NREM-REM sleep cycle) (Hobson & Pace-Schott, 2002). What exactly goes wrong when a stroke or an experimental lesion disrupts these systems we do not yet know. But the model of primary and secondary effects discussed in the next section suggests an experimental paradigm that could link sleep research to the pathophysiology of stroke in a novel way.

# 5.3. Experimental lesions and strokes

When we make a lesion in an experimental animal, we almost certainly produce primary (neuronal death) and secondary (neuronal dysfunction) effects just as occurred in my stroke. To wit, experimental transection of the medulla at the medio-pontine level produces in intense insomnia lasting about 10 days but this is followed over the subsequent 20 days by recovery of normal sleep (Hobson, 1965). And, as Villablanca (1968), has pointed out, the longer one waits and the longer one observes the more one sees recovery and/or compensation of function. It is currently impossible to decide between simple recovery and compensation, implying plasticity. But it now seems probable—on the basis of my case data and that of the experimental literature—that the explanation of the insomnia caused by medullary lesions is *not* as likely to be disenablement of a putative synchronizing mechanism in the lower brain as it is due to secondary damage of pontomedullary structure adjacent to the primary lesion.

These observations lead us to conclude that any inferences about structure– function relationships need to be made with extreme caution. Before concluding that this or that sleep stage or its associated mental experience has been eliminated by a lesion we need much longer observation periods than most experimenters have used and much more searching psychological probes than neuropsychologists have yet employed. In the case of functions, like dreaming, which depend on subjective reports, we need to be sure that the subjects' capacity to report has not been damaged. In my case, the rehabilitation phase hypersomnolence was an obstacle to dream recall and dream reporting. And of course we need to obtain objective recordings to corroborate subjective reports. Did I really recover my REM sleep? Was it the same as my REM sleep prestroke? These are important, questions which, until answered, limit our interpretive power.

## 6. Conclusion

From the data presented here, I feel safe in concluding that as long as my brain stem was in the throes of acute and postacute infarction I was unable to sleep and to dream in a normal fashion. Once recovery began and my brain stem had recovered its ability to support sensorimotor functions, my dreaming recovered too. I have no doubt that normal dreaming requires a normal forebrain and that severe damage to the forebrain leads to a loss of dreaming that may even be permanent. But based on my experience I also have no doubt that a normal forebrain cannot sustain normal dreaming as long as the pontomedullary brainstem is dysfunctional.

## References

- Batini, C., Moruzzi, G., & Palestini, M., et al. (1959). Effects of complete pontine transections on the sleep-wakefulness rhythm: The midpontine pretrigeminal preparation. *Archives of Italian Biology*, 97, 1–12.
- Dietrich, M., & Brandt, T. (1992). Wallenberg's syndrome: Lateropulsion, cyclorotation, and subjective visual vertical in thirty-six patients. *Annals of Neurology*, *31*(4), 399–408.
- Helmchen, C., Straube, A., & Buttner, U. (1994). Saccadic lateropulsion in Wallenberg's syndrome may be caused by a functional lesion of the fastigial nucleus. *Journal of Neurology*, 241, 421–426.
- Hobson, J. A. (1965). The effect of chronic brainstem lesions on cortical and muscular activity during sleep and waking in the cat. *Electroencephalography and Clinical Neurophysiology*, 19, 41–62.
- Hobson, J. A. (2001). The dream drugstore. Cambridge, MA: MIT Press.
- Hobson, J. A., Peace-Schott, E. F. (2002). Basic mechanisms of sleep: New evidence on the neuroanatomy and neuromodulation of the NREM–REM cycle. In D. Charney, K. Davis, C. Nemeroff (Eds.), *American College of Neuropsychopharmacology, fifth generation of progress* (pp. 1857–1877). New York: Lippincott, Williams, and Wilkins.
- Hobson, J. A., Pace-Schott, E. F., & Stickgold, R. (2000). Dreaming and the brain: Toward a cognitive neuroscience of conscious states. *Behavioral and Brain Sciences*, 23(6), 793–842.
- Hörnstein, G. (1974a). Wallenberg's syndrome. Part I. General symptomology, with special reference to visual disturbances and imbalance. *Acta Neurologica Scandanavica*, 50, 434–446.
- Hörnstein, G. (1974b). Wallenberg's syndrome. Part II. Oculomotor and oculostatic disturbances. Acta Neurologica Scandanavica, 50, 447–468.
- Moruzzi, G. (1972). The sleep-waking cycle. Ergebnisse Physiologie, 64, 1-165.
- Pearce, J. M. S. (2000). Wallenberg's syndrome [historical note]. Journal of Neurology, Neurosurgery, and Psychiatry, 68(5), 570.
- Solms, M. (1997). The neuropsychology of dreams: Clinico-anatomical study. Hillsdale, NJ: Erlbaum.
- Solms, M. (2000). Dreaming and REM sleep are controlled by different brain mechanisms. *Behavioral and Brain Sciences*, 23(6), 843–850.
- Solomon, D., Galetta, S. L., & Liu, G. T. (1995). Possible mechanisms for horizontal gaze deviation and lateropulsion in the lateral medullary syndrome. *Journal of Neuro-Ophthalmology*, 15(1), 26–30.
- Villablanca, J. (1968). Cortical control of thalamic spindle wave. *Experimental Neurology*, 20, 432–442.
- Werner, D. L., Ciuffreda, K. J., & Tannen, B. (1989). Wallenberg's syndrome: A first-person account. Journal of the American Optometric Association, 60(10), 745–747.