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Editor’s Column

Many of you have asked how often we print this publication. At the SRS executive meeting in Nashville (June) it was formally decided that we will publish every quarter. We are trying to put the Bulletin on the world wide web so that you can access it directly. This will also save mailing and printing costs. At this time we are still in the process of finding a web-site, server etc.

In this issue we have published the winning essays from the SRS Student Essay Contest. A record-breaking number of high-schools participated and many students sent in essays. As you know, the essay contest is one way that we are reaching out to the community and conveying the message that sleep research is important. Clearly, our message is getting through. What is more important is that the next generation of sleep researchers is likely to come from such young people. We will give each of the winners a copy of this issue of the SRS Bulletin. We also urge all of you to make copies of this issue and send it to your high school along with the notification of the essay contest. This is one of the real successes of our organization and we want more students participating next year.

President’s Column

Actually, SRS does a good job with the essay contest, but we individually could do more. When I have been invited to schools to talk about the animal research issue, I find a very interested audience when the students learn I am a sleep researcher. Then it is easy to introduce the idea that all sorts of research has contributed to our understanding of sleep and its disorders and that animals have contributed in important ways. I hope you will participate in this vital endeavor.
Letter to the Editor

From: Avi Karni, Laboratory of Neuropsychology, NIMH, NIH Bldg. 49, Rm.1B80, Bethesda MD 20892.

Memory consolidation during REM sleep: perchance not a dream?; a response to R.P. Vertes.

I agree with Robert Vertes (SRS Bulletin 1995, 1(2): 27-32) that there appears to be little evidence that "magical processes" relating to memory consolidation take place during sleep. I think, however, that on his way to make (what is to me) a reasonable point, Vertes has misread our study design and results (Karni et al, 1994), as well as our interpretation of these findings.

The design of the Karni et al study had a much simpler and a more straightforward logic than what Vertes ascribes in his critique. Performance was measured, by psychophysical means, for subjects training on a simple target-from-background discrimination task (first described in Karni & Sagi, 1991) with a novel (unique) set of stimuli on the evening before sleep (either normal or selective sleep-stage disrupted). Performance of the same task was then remeasured, using that very same set of stimuli, on the next morning. What we found was that across a time interval in which sleep - either normal, REM stage deprived or SW stage deprived - was interposed, a large gain in performance occurred for (during) normal as well as SW deprived sleep, but not during REM deprived sleep. We have previously shown (Karni & Sagi, 1993) that provided that about 8 hours are allowed to elapse after the initial training session, a large and long-lasting gain in the performance of this task can be found. As no immediate effects of training (i.e., immediately following, 1, 2, 4, 5 and, for some subjects, even 6 and 8, hours after the training session) were found, our results indicated that a time dependent process of plasticity in the adult visual processing pathways is initiated during the training session and requires about 8-10 hours to evolve.

Vertes is wrong in his reading that only during the control phase of the study the test stimuli were always presented within a single quadrant of the display, and he is also mistaken in thinking that, subsequently, performance was compared to this initial control measurement. For each of the experiment's phases, baseline performance before sleep was compared to performance after sleep, using the very same stimulus configuration. What subjects saw (trained on) on the initial (pre-sleep) session were the very same stimuli (in terms of element orientation and target locations) on which they were retested on in the next morning's session.

For each of the study phases, however, either a new target location was chosen and/or a switch in the background elements' orientation was made to create a novel set of test stimuli, so as to initiate a new, independent learning process. These were introduced in the pre-sleep training/measurement session. This design was used to counter the possibility of a carry-over effect from one study phase across to the other for the very same reason that Vertes raises: "it might be argued that some critical learning occurred from the first to the second deprivation". One advantage of our perceptual learning paradigm as a probe for investigating adult skill learning is that a switch in stimulus configuration amounts to "starting with a fresh slate" for each study phase. The reason for that may lie in the fact that basic physical attributes of the sensory input presented during training determine which of very discrete populations of neurons will undergo experience-dependent plasticity (Recanzone et al, 1992; Weinberger et al, 1993; Bertini et al, 1995). Thus, in our paradigm (this is an important characteristic of many different perceptual skill learning paradigms) learning is specific for physical attributes of the stimulus such as, for example, the target's location. In other words, learning is retinotopically local: when one trains with the target consistently presented in one quadrant of the display, no performance gains occur for visual-field locations removed by more than 3 degrees of visual angle from the trained visual-field location. Thus new, independent, training is required for learning to occur for each visual-field location, and as a direct consequence of this characteristic of perceptual learning paradigms, one can initiate several parallel learning processes within the (visual system of the) same subject.

Vertes' criticism stems from his (mis)reading that the switch in stimulus condition was done on the morning after the sleep intervals of each study phase. This is why he finds it surprising that the "SWS deprived subjects showed immediate (?) improvement". Presumably, by the same token, one can understand why Vertes was not surprised by the improvement after normal sleep? Vertes correctly notes that a counter-
balanced design was used, which makes his interpretation of the data even more awkward. His interpretation of subject NK's results is difficult to defend because a double night of deprivation was also employed in the case of subject IA during both the REM and the SW deprivation phases. But then, as Vertes' criticism follows a misreading of the basic study design, the whole line of argument is not very relevant.

As I have explained in my short paper (SRS Bulletin 1995, 1(2): 32-34), written without being aware of Vertes' paper, we use the term "consolidation" to denote an active, time-dependent process underlying the emergence (evolution) of long-term, experience-driven, changes in basic visual processing. "This is a reasonable use of the term not only because the improved skills, once gained, were not forgotten even after an interval of several years., but also in analogy to the use of the term in studies of developmental mammalian-brain plasticity, [in which] the effects of experience on neuronal response properties were shown to develop long after the inducing stimulus (experience) has been withdrawn" (Karni et al, 1994). What our findings suggest, is that REM sleep is a necessary requirement for the consolidation of experience dependent plasticity in the adult cortex during sleep. We do not know whether it is also a sufficient condition for this type of consolidation. To borrow Hobson and Stickgold's summation (1995): "REM sleep (but not non-REM sleep) can subserve [...] plasticity as effectively as waking". Our conjecture is that neuronal mechanisms subserving the consolidation of experience-driven cortical plasticity, are active during normal (REM) sleep as during waking; either because both REM sleep and the waking state share a common property that is necessary for consolidation, or (which we think less likely for reasons of parsimony) through two different neuronal mechanisms.

This interpretation, I think, is not in disagreement with Vertes' proposal that "the primary function of REM is to provide endogenous stimulation to the brain which serves to maintain requisite levels of CNS activity throughout sleep" which is quite common ground. Nor do we make any claims about "mental phenomena or quasi-conscious processes". Indeed, we suggest that our use of a very basic non-declarative procedural (skill) learning paradigm (which is completely lacking semantic content) is the foremost factor for a quite straight-forward interpretation of our results in a reductionistic framework. Procedural memory (learning "how" memory) of the type induced by training in our visual discrimination task does not depend on classical (hippocampus, limbic system related) memory circuits (declarative, learning "what" memory), and can be formed, without awareness even in profoundly amnesic patients. As texture discrimination learning is determined by the specific retinal input presented during the pre-sleep practice session, the role of REM sleep may reside in providing a critical milieu for the transformation of the activity-dependent neural change, presumably initiated during the pre-sleep session, into a more efficient and stable (consolidated) form. All we can say from the limited point of view afforded by our results, is that there is a reasonable argument to be made for the idea that sleep is a brain state, perhaps not necessary, but sufficient for the consolidation of some types of memory (Karni, 1995). I think this does support the hypothesis that a process of memory consolidation occurs in sleep.

References
Reply to Avi Karni’s Letter to the Editor, SRS Bulletin

Robert P. Vertes, Center for Complex Systems, Florida Atlantic University, Boca Raton, FL 33431

I welcome Dr. Karni’s response to my recent article in SRS Bulletin (Vertes, 1995). The role of sleep in memory consolidation is an important topic and deserves our continuing attention. These exchanges will undoubtedly sharpen our approach to the problem.

My article dealt with several issues related to sleep and memory. Understandably, Dr. Karni’s letter focuses on my treatment of their recent article (Karni et al., 1994). Karni’s main objection to my brief summary/critique of their work was that I misread their design and results and hence misinterpreted their findings. Karni did not address what I believe to be the most serious problem with their study which was their small subject pool: only four subjects (of a total of 6 subjects) completed the entire experiment.

With respect to my misreading of the design of their study, I do not believe that I did. I will acknowledge, however, that I may not have adequately conveyed my main concerns with their study. I will try again.

As I previously indicated, their perceptual task (Karni et al., 1994) involved the identification of a test stimulus consisting of three adjacent lines oriented differently than a background pattern of lines. In the initial (control) phase of the study, the test stimulus was presented within one quadrant of the display (e.g., upper left). The measure of improved performance was shorter latency identification of the test stimulus. The effects of sleep state disruption were evaluated by training/testing subjects with the stimulus in a new quadrant (e.g., lower left), sleep depriving them (SWS or REM), and then re-testing them with the stimulus in this quadrant (i.e., lower left). To control for possible non-specific effects of sleep deprivation (e.g., stress, fatigue), subjects were also re-tested both before and after a period of sleep deprivation with the stimulus in the original quadrant (i.e., upper left).

Karni states that; "Vertes' criticism stems from his (mis)reading that the switch in stimulus condition was done on the morning after (italics ours) the sleep intervals of each study phase. This is why he finds it surprising that the 'SWS deprived subjects showed immediate improvement'. Karni is mistaken. We previously stated that the switch in quadrants was made before and not after a night of sleep deprivation. We said: "Subjects were tested with the stimulus in the new quadrant; sleep deprived ....; and then re-tested with the stimulus in the new quadrant (i.e., the same quadrant in which it was presented in the single session before sleep deprivation)" (italics added here).

It appears that our principal area of disagreement involves the question of whether the switch in quadrants represents a completely new task as claimed by Karni ("a switch in stimulus configuration amounts to 'starting with a fresh slate' for each study phase") or whether one phase of the experiment could influence subsequent phases, as we contend. We suggest that training/testing in one quadrant could adversely affect performance in another quadrant; that is, performance in the 'new' quadrant would, at least initially, be poorer with, than without (naive), prior training. In accord with this interpretation, the data of Karni et al. (1994) shows that switches in quadrants always resulted in significantly longer latencies to identify the test stimulus during the first test session in the new quadrant (see Fig. 1C). Karni would likely attribute this to beginning with a 'new slate'. Karni would also likely argue that the poorer performance following the switch of quadrants would dissipate over one training session. In contrast, we would suggest the latencies might continue to be significantly elevated (i.e., poorer performance) for additional test sessions following the switch to a new quadrant. We would contend that this is, in fact, what their data shows for the REM deprived group; that is, long latencies (and equally long) for the first two sessions following the switch in quadrants (i.e., before and after REM deprivation). Further, we would argue that subjects might perform poorly after the first REM deprivation period, but significantly better with subsequent REM deprivations (i.e., with repeated training/testing in the new quadrant). This was the case for one subject (NK), but as pointed out by Karni, not for another subject (IA). Finally, we still question why the SWS deprived group showed an essentially immediate improvement in performance; that is, the first session after sleep deprivation (or the second session following the switch of quadrants).

In conclusion, an underlying and fundamental assumption of the study of Karni et al. (1994) is that learning in one quadrant (or part of the retina) is completely independent of learning in another.
quadrant. Although Karni, in his letter, cites recent evidence to support this, we feel that this position is tenuous. We have suggested that learning in one quadrant could adversely affect learning in another quadrant; alternatively, learning in one could facilitate learning in another. In any case, the study of Karni et al. (1994) was based on the assumption that there is no interaction among separate regions of the retina -- a 'clean slate' in their terms. We feel that it may be problematic to structure a study driven by this assumption, especially one that purports to show that "a process of human memory consolidation, active during sleep, is strongly dependent on REM sleep" (Karni et al., 1994).

REFERENCES


SLEEP RESEARCH SOCIETY HIGH SCHOOL ESSAY CONTEST AWARDS

By Gina Poe, SRS Executive Committee Trainee at Large, Nashville, TN (June 2, 1995)

The Sleep Research Society announced the winners of the 1995 Essay Awards Program at the SRS Executive Committee Meeting. As Executive Committee Member in charge of the 1995 High School Essay Awards Program, I am honored to announce the outcome of the contest.

146 entries were received from high school students from 20 states and Canadian Provinces and all essays were judged on the basis of content, scientific application and originality. The 146 essays mark the highest number of entries received in a single year since the program's inception in 1992.

Five first place awards were presented to:


Helen Florez, "Sleep Deprivation and its Unknown Dangers", Cooper City High School, Cooper City, FL.

Jennifer Hall, "Slumber Interrupters", Allen High School, Allen, TX.

Elisabeth Laderoute, "The Importance of Understanding Sleep Disorders", Garden City High School, Garden City, NY.

John Meliska, "Melatonin and Sleep", Carbondale Community High School, Carbondale, IL.

In recognition of this achievement, a cash award of $250 and a certificate of excellence will be awarded to each of these students. Additionally, the school library of each winning entry will receive a copy of The Encyclopedia of Sleep and Dreaming (Carskadon, ed., New York: Macmillan, 1993).

All essay entrants received a sleeping brain t-shirt and all teachers of entrants received a copy of the SRS syllabus, "Basics of Sleep Behavior." The Program Committee hopes that these items may serve as a foundation for future study of sleep in high school classrooms.

Thanks goes to Katharine Sharkey for her valuable suggestions regarding ways in which SRS members can personally contact high schools and promote participation in this program. Thanks to Douglas Nitz for his assistance in coordinating the judges, and thanks to all those SRS members who wrote to, or visited your alma maters and area high schools to promote the Essay Awards Program.

Special thanks to Gregory Mader in the APSS office in Minnesota for his tireless efforts and excellent work to coordinate the Awards Program and make it run smoothly.

Individuals interested in the 1996 Essay Awards Program should contact the Sleep Research Society at 1610 -14th Street Northwest, Rochester, Minnesota, 55901 after October 1, 1995.
Sleep Apnea
John Dennis
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Cold Spring Harbor, New York

The word apnea is the Greek word for "want of breath." Sleep apnea affects six million people. A person with sleep apnea has an impairment to some part of their respiratory system causing them to cease breathing for periods of 10 to 20 seconds while they sleep. The blood receives less oxygen and the brain signals the person's body to gasp for breath. This can happen hundreds of times in a single rest period and can result in three quarters of the person's sleeping time spent not breathing.

There are three different kinds of sleep apnea. The first kind, the most common, is known as obstructive sleep apnea. The impairment to the respiratory system is caused by the partial blockage of the air passage. This can happen to a person with an enlarged tongue, enlarged tonsils, a small jaw, or any other physical aspect that could cause the air passage to the lungs to be blocked. The second kind is a rarer form called central sleep apnea. Central sleep apnea is caused when the diaphragm and chest muscles temporarily fail. People with central sleep apnea have no air blockage and do not snore loudly, but suffer the same symptoms as people with obstructive sleep apnea. The third type is called mixed sleep apnea. A person who suffers from mixed sleep apnea simply has both obstructive and central sleep apnea.

Sleep apnea has many repercussions, both nighttime and daytime. The very unsteady breathing results in very loud snoring and abnormal, uncomfortable sleeping. A person with sleep apnea may experience a headache immediately after awakening. The person can also become irritable, depressed, and can find it hard to concentrate. Sleep apnea sufferers may also have the tendency to fall asleep in the middle of the day, a potentially life-threatening problem. People with sleep apnea are two to five times more likely to be in a car accident than those without sleep apnea. A person suffering from sleep apnea receives air in spurts, therefore the blood receives oxygen unevenly causing cardiovascular complications such as high blood pressure and hypertension.

Studies have shown that there are many traits characteristic of sleep apnea sufferers including: gender, weight, age, alcohol intake, and even neck size. The typical sleep apnea sufferer is a middle-aged, overweight man. Eight times as many men have sleep apnea than women. Increased alcohol intake can greatly worsen the effects of sleep apnea. Experts say that sleep apnea can be caused by hormonal problems including hypothyroidism and acromegaly. Older, more overweight sleep apnea patients have an increased chance of hypertension.

Only 10 to 25 percent of sleep apnea cases are ever diagnosed. To diagnose sleep apnea, a series of tests are run on a sleeping patient. This test is called a polysomnography. A polysomnography detects many things including differences in breathing and heart rate, which would show up as a result of sleep apnea. For mild cases of sleep apnea the treatment may be as simple as not sleeping on one's back or taking a decongestant to clear one's nose. Before 1981, there was only one treatment for more serious cases of sleep apnea. This is a tracheostomy, a hole surgically cut in the throat with a tube attached. This completely clears the airway and provides relief from the problems of sleep apnea, but now it is only used in very extreme cases. A type of surgery is now possible called an uvulopalatopharyngoplasty. In a UPPP, certain tissues in the back of the patient's throat are removed to increase the size of the air passage. This process is shown to only be effective in only half the cases of sleep apnea since the tissue removed may only be partially responsible for blocking the air passage. The most common treatment for sleep apnea is nasal continuous positive airway pressure, or CPAP. A mask attached to a motor goes over the patient's face and regulates the amount and the pressure of the air that travels into the patient's nose. CPAP opens up collapsible upper airways to restore normal breathing during sleep. CPAP is shown to relieve all the symptoms of sleep apnea. Since there are no side-effects to CPAP, this form of therapy can be used without having completely diagnosed sleep apnea. CPAP therapy can prove to be ineffective, especially since many patients prefer the symptoms of sleep apnea to the uncomfortable mask. Another form of treatment is surgery to the patient's jaw. The jaw can be moved forward surgically to increase the size of the air passage. The jaw can also be moved forward through use of a special dental device. Dental devices are also made that hold the tongue back or raise the soft palate.
Sleep apnea can range from mild forms to extremely serious. Problems associated with sleep apnea can range from slight discomfort to severe hypertension and cardiovascular problems. Mild forms of sleep apnea can be cured easily while serious cases require methods that are not always effective and have not yet been perfected. The technology for treating this problem is advancing all the time and hopefully soon we will have the cure for sleep apnea.

Bibliography


Sleep Deprivation and Its Unknown Dangers
Helen Florez
Cooper City High School
Cooper City, Florida

People know that sleep is essential, yet millions of Americans seem to be sleep deprived, according to "Wake up America" (1993). The reason that most people don't sleep enough is because of technology. Ever since the invention of the light bulb, people are able to stay up during all hours of the night. Nowrozzi (1992) says that more than seventy-five percent of Americans use alarm clocks on a routine basis in order to wake-up. Almost the same amount of people sleep about two or three hours more on the weekends than they do on the weekdays. Both of these are indications of severe sleep deprivation. According to Alperstein (1994) routine duties can be performed normally after a bad night's rest, but tasks that require creativity or coordination are more apt to be affected. That is why the number of hours a trucker or pilot is allowed to work without rest, are regulated by the government.

I did a research project and concluded that the effects of sleep deprivation can be devastating. I took eight subjects and had them fill out a survey. All of them seemed to be slightly sleep deprived on a regular basis. Though we do learn to combat sleepiness, in order to perform at our top levels we have to work harder than if we were well rested. I then asked these eight subjects to stay awake for a total of twenty-four hours. After the first twenty hours, I gave them each two tests, one for memory and the other for math. For the memory tests, I read out loud twenty words and then asked them to write down as many as they could remember in 150 seconds. I then gave them a math test which was comprised of three addition, three multiplication, and four subtraction problems. Once they were finished, I wrote down how much time it took them to complete the test. I did this after twenty-two and then twenty-four hours without sleep. For my control group, I had them take similar tests to those described above, only it was one week later and they were not sleep deprived.

These are the results. The control group got an average of 9.375 problems correct on the math test. But once they were sleep deprived for twenty and twenty-two hours it went down to an average of 8 and 8.125 problems correct. And finally, after twenty-four hours without sleep, they only got an average of 7.5 problems correct. The time that it took the control group to complete the math test was an average of 143 seconds. After twenty and twenty-two hours without sleep it went up to 233 and 230 seconds. And after being awake for twenty-four hours it generally took them 247 seconds to complete the test. The control group remembered about 10.25 words out of the twenty that were read out loud. After twenty and twenty-two hours without sleep they only recalled an average of 8.5 words. And after they were awake for twenty-four hours they only remembered an average of 5.875 words, which is almost half of what they recalled when they were alert. This means that if you are sleep deprived, you will do 20% worse on mathematical computations than if you were alert. It will take you about one minute and forty-four seconds longer. And your memory will hold 57% less information than if you were vigilant.
I noticed that after twenty hours without sleep, people felt as though they were able to function normally, but the test results show that even then they began to perform more poorly. After twenty-two hours they were a little drowsy and they had no energy, but they still felt that they could pull through the tests OK. But after twenty-four hours without sleep, they were extremely tired. Some were not able to keep there eyes fully opened. They were extremely agitated and moody. And a lot of them complained that they were cold. When I talked to the subjects a few days later, most of them complained that they still felt the effects one or two days later. They were not able to concentrate properly and all felt somewhat drowsy.

I am concluding that the signs of sleep deprivation are very apparent in this experiment. And the subjects were not kept awake as long as some doctors and nurses are sometimes. The subjects were not responsible for hundreds of lives like a pilot or captain are. But that is what they would all experience if they stayed awake that long. I think it is very important that employees make sure that they are well rested before they go to work, or else they could be putting themselves and others in danger.

Sleep is biologically necessary, but people don't regard it as important as food or water. Without sleep, mental focus and performance can be abraded (Toufexis 1990). People's attention spans can't hold up for long. They are also hot-tempered, irritable, and more likely to get angry at the slightest offense. Evidence keeps piling up that sleep deprivation has become one of the most widespread health problems that the U.S. has to confront.

Bibliography


Slumber Interrupters
Jennifer Hall
Allen High School
Allen, Texas

Though in theory it's as natural to the human body as eating and breathing, the act of sleeping is becoming increasingly difficult for modern Americans. According to Emlyn E. North of the Menninger Perspective, over 50 million adults in the U.S. report "trouble with sleeping", and about one fifth of those receive medical attention for their troubles. As somnologists Lynne Lamberg astutely pointed out in an article published in American Health Magazine, "Sleepers make strange bedfellows," and sleeping disorders are almost more prevalent than their absence.

There are at least 84 distinct sleep disorders affecting Americans, ranging in seriousness from ordinary anxiety-induced insomnia to recently identified REM Behavior Disorder in which sufferers nightly "act out their dreams" in violent and/or self-destructive episodes. Whether cataloged as "severe" or not, the most common effect of sleep disorders is lack of sleep, and lack of sleep does more than just exhaust you. Sleep loss can cause myriad pernicious consequences including, according to Cornell University professor James Mass Ph.D., illness, ongoing sleep problems, emotional problems, and memory loss, and when one
considers that pilots, firefighters, and surgeons are just as susceptible to sleep disorders as any other American, the implications of the prevalence of sleep disorders becomes distressingly apparent.

Perhaps the most common type of sleep disorder, those of initiating and maintaining sleep, increase in severity and occasion of incidence as people age (North 26). Two of the most interesting of this type of disorder are sleep apnea and sleep related myoclonus. Sleep apnea causes breathing to become shallow or stop as a person falls asleep. The sufferer awakes suddenly-sometimes hundreds of times in a single night-in order to restore the flow of air. More often than not a person with sleep apnea "trains" their nervous system to resume sleep quickly and may not recall the disturbances when fully conscious, but the many awakenings take a severe toll on their ability to maintain wakefulness during the day.

Sleep related myoclonus, a condition believed to be brought on by a sedentary lifestyle, is nighttime twitches of the leg muscles causing fragmented sleep and, if untreated, chronic insomnia. Both sleep apnea and sleep related myoclonus are easily cured once identified; sleep apnea with a mechanical device that pressurizes air, and myoclonus with exercise and/or muscle relaxers (Lambert 67-69).

The best known of the second type of sleep disorder, those characterized by excessive somnolence, is narcolepsy. Victims of this disease fall asleep suddenly and without apparent relationship to their sleeping habits. Narcolepsy usually arises during late adolescence and is frequently accompanied by day-time bouts of cataplexy, a weakness which causes one to collapse when they experience strong emotion (Kemper 22). Though it seems paradoxical, narcoleptics tend to fall asleep most often in stressful and/or exciting situations (e.g. in the middle of an important meeting or test, during the climax of a movie, or at bat in a baseball game). Amphetamines are frequently used by narcoleptics to control their tendency to fall asleep or experience weakness, but there is no known natural remedy (such as frequent napping or getting a certain amount of sleep each night) or any permanent cure (North 26).

A third type of sleep problem, REM Behavior Disorder, remains an enigma to many somnologists. According to neurologist Mark Mahowald, director of the Minnesota Regional Sleep Disorders Center in Minneapolis, it is a rare multiple personality disorder which only manifests itself during sleep (Kemper 82). Normal sleepers experience a form of mild paralysis during rapid eye movement sleep, but victims of this bizarre disorder flail about wildly, usually in motions directly corresponding to the events of their dreams (Blakemore 27-31). REM Behavior Disorder can be extremely dangerous-both to the victim (a teenage sufferer threw himself through a glass patio door)- and their bed partners (an adult male victim nearly strangled his wife believing he was saving her from drowning). Though currently there is no cure for REM Behavior Disorder, clonazepam, a sedative type drug, is an effective suppressant of these nocturnal episodes.

Shakespeare said, "Sleep knits up the raveled sleeve of care", but for sufferers of sleep disorders, trying to achieve normal, refreshing, uninterrupted sleep may cause more stress than it relieves. Luckily, sleep disorder clinics and treatment centers are becoming increasingly prevalent as the rates of sleep disorder incidence continue to grow (Abbot 72). Sleep related studies and research now underway will allow medicine and science to continue to treat and cure sleep disorders of the future. Human beings spend at least a third of their life sleeping, more than on any other single activity; research on sleep disorders allows those who suffer from them to spend that time- as well as their hours of wakefulness-more peacefully.

Works Cited


5. Sleep Deprivation and Its Unknown Dangers
The Importance of Understanding Sleep Disorders  
By Elisabeth Laderoute  
Garden City High School  
Garden City, New York

Few people are immune from sleep problems. In a recent poll only 5% of adults reported that they never had trouble sleeping. This leaves an overwhelming majority of 95% who have experienced sleep problems in one form or another. In a study done during the past year, when asked about their recent sleep experiences, people complained greatly about their sleep problems. The figures speak for themselves; 60 million American adults, or between 30 and 35 percent of the population, say they have had some trouble sleeping. About half of this group characterize their sleep problems as severe or constant. Sleep problems annually cause 10 million Americans to consult their physicians. Despite the fact that sleeping pills can be dangerous and more often than not may contribute to a sleeping problem, 21 million prescriptions for sleeping pills are written each year. Considering that Americans spend approximately a third of their life in a state of crucial sleep, it is important to understand the sleep cycle and the ramifications sleep disorders may bring.

Sleep disorders can be divided into two basic categories; the first is insomnia, the second, all other sleep disorders. Insomnia is a condition in which a person has difficulty getting sufficient sleep. About 30 million people in the United States suffer from this problem. One can think of insomnia as consisting of two different and usually separable symptoms: difficulty in falling asleep and difficulty in remaining asleep. The term "insomnia" is actually a misnomer because it implies a complete lack of sleep. Sleep professionals sometimes refer to insomnia as DIMS, an acronym for "disorders of initiating and maintaining sleep."

Difficulty falling asleep is a more common type of insomnia among people under 40 or 50. Difficulty staying asleep becomes more common after that age. Insomnia becomes a significant problem only if it interferes with daytime mood or functioning. The seriousness of insomnia is measured not by how little a person sleeps, but by how well the person feels and functions the next day. Despite the genuine distress sleepless nights may bring, it is equally useful to consider the accuracy of perception of how well one sleeps. People with insomnia tend to overestimate how long it takes them to fall asleep and to underestimate how long they sleep. One study at Stanford University observed overnight the sleeping pattern of a group of self-reported insomniacs in the sleep laboratory. The next morning these subjects estimated on average that it had taken them roughly an hour to fall asleep. The subjects also reported that they had slept only four and a half hours. However, the surveys recorded that the subjects, on average, had taken about 15 minutes to fall asleep and had slept for six and a half hours. Therefore, insomnia is often as much a psychological problem as a physical ailment. Subjects appear to perceive the onset of sleep at a later time than people with normal sleeping patterns. Furthermore, they don't perceive themselves asleep until they are well into stage two. These findings are not intended to minimize the reality and distress of insomnia. However, the evidence is useful to know that in some instances, especially when one feels anxious about losing sleep, he may overestimate the amount of sleep loss.

Experts estimate that in three-fourths of all cases of insomnia, the cause is the result of psychological difficulties. Depression is a common cause of insomnia. About 20% of women and 10% of men experience an episode of serious depression at some time in their lives. Poor sleep, especially a pattern of awakening frequently during the night or in the early morning, can often be the first sign of severe depression. Depression related insomnia is not lifelong or long-lasting. The onset is often insidious but sleeping troubles seem to become worse over a period of two, three, or four months. The most common depression related sleep problem is awakening in the predawn hours with an inability to return to sleep. The successful treatment of depression will improve insomnia.

Anxiety is another psychological cause that clearly produces difficulty in falling asleep among insomniacs. Worry over an upcoming event can often make it difficult to fall asleep rather than remain asleep. One spends time thinking, either being excited or worried, about what is going to happen. Anxiety is a very common cause that keeps millions of Americans up each night.

Besides the mentioned psychological causes of insomnia, medical problems and medications can affect sleep. Disorders of every organ system in the body can
lead to insomnia. Many illnesses have symptoms—such as pain, itch, or shortness of breath—that may hardly be noticeable during the day but interfere with sleep at night. Sleep apnea is one very important but often unrecognized medical cause of insomnia. Sleep is disturbed repeatedly because the breathing mechanism is not working properly. Observed subjects with apnea have shown that breathing may seize from 10 second to minutes. This is an incredible phenomena when we think about consciously trying to hold our breath in a wakeful state. Other medical causes of insomnia can include angina, hypertension, asthma and heartburn.

Another frequent and poorly recognized cause of insomnia is medical drugs. Hardly any of the wonderful drugs of modern medicine are free of side effects and problems. The price we pay for therapeutic drugs is often sleep. There are drugs that actually have a stimulant effect and that will produce difficulty in sleeping as a pharmacological effect, especially if taken in the evening. These include amphetamines (Dexedrine, Ekatrol) and methylphenidate (Ritalin). Caffeine can also interfere with sleep, although sensitivity to caffeine differs tremendously from person to person.

The causes for insomnia are many. Whether psychological or medical, insomnia persists as a serious problem which affects millions of Americans. With medical research and technology improving each day, there is hope for insomniacs who endure the sleepless nights.

As humans we spend our lives in two basic states—wakefulness and sleep. Those of us who live to be a hundred will have spent perhaps 30 years in the state of sleep. It is evident that sleep is an important and vital factor of life. Many important biological phenomena occur during sleep, namely cell regeneration. Few people however, are immune from sleep problems. It is important therefore that scientists have come a long way in understanding the importance of sleep. However much more information is needed before we can truly sleep well at night.

Bibliography


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MELATONIN AND SLEEP

John Meliska

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Much research has been conducted in the past to understand jet-lag, insomnia, circadian rhythm dysfunction, and other disorders associated with sleep. These problems have afflicted many people, and have affected productivity. Jet-lag, a common condition, is characterized by insomnia, fatigue, weakness, sleepiness, gastrointestinal complaints, irritability, and malaise (Moline, Pollak, Monk, et al., 1992). Recent studies have indicated that the hormone, melatonin, may be therapeutically useful for the control of circadian clock dysfunction, such as jet-lag, shift work syndrome, and other sleep disorders (Cassone, 1990). The hormone melatonin is important in the regulation of sleep and the circadian rhythm of the human body. Melatonin is secreted by the pineal gland, which is located in the brain (Wilson & Foster, 1985). The pineal gland and melatonin are an important part of the circadian system in humans (Cassone, 1990).

According to William Dement in his book, Some Must Watch While Some Must Sleep, circadian rhythms can be classified as cycles that are completed under natural conditions in a twenty-four hour period. In humans, the pineal gland is sensitive to light or darkness that may or may not be consciously noticed. Melatonin is secreted into the blood stream almost immediately after exposure to darkness, and is stopped with exposure to light (Wilson & Foster, 1985). Melatonin secretion occurs most frequently at night, where levels in the blood can be 3 to 10 times more than the amounts.
present in the day (Cassone, 1990; Wilson & Foster, 1985; Pierpaoli, Regelson, Fabris, 1994). Melatonin then induces sleepiness, and can increase the amount of rapid eye movement (REM) sleep. It has been reported that melatonin can create a "sensation of well being and moderate elation," though large doses can cause headaches and cramps (Wilson & Foster, 1985). This consistent release of melatonin makes it an important part of the body's circadian rhythm. When the body's circadian rhythm is interrupted, undesirable physiological effects can occur, e.g., insomnia. The malaise accompanying the condition may not be the result of sleep deprivation, but more likely the body responding to the interruption of its regular routine (Dement, 1974). The process of realigning the circadian system to a new routine is an active one. It usually takes several days because the biological rhythms controlled by the circadian system are not simply reactions to changes in activity and rest (Monk, Moline & Graeber, 1988).

To better understand the causes and possible methods of treatment, much recent research has been directed toward the study of melatonin. In one study, a group of travelers engaging in flights from London to San Francisco and back were given melatonin before they slept. A control group was given placebo in a similar fashion. The results to the study are as follows: "Sleep quality was significantly improved in the melatonin group and correlated negatively with jet-lag ratings. Melatonin treated subjects tended to be more alert than placebo subjects, especially at bedtime. They were also less depressed. Jet-lag was significantly less in melatonin- treated subjects" (Arendt, Aldhous, English, et al., 1987). In other studies, melatonin administration has ameliorated the psychological and physiological effects of jet-lag. Melatonin has also had positive effects in synchronizing the circadian rhythms of totally blind patients, whose sleep patterns are frequently disrupted (Cassone, 1990).

However, melatonin may have certain connections with depression in humans. It was previously stated that melatonin can cause enhanced REM stages; therefore, it most probably is present in the body during REM. According to Wilson & Foster in the Williams Textbook of Endocrinology, exposure to melatonin lowers plasma leptinizing hormone levels and suppresses growth hormone secretions. Wilson and Foster go on to state that REM sleep also inhibits growth hormone, and that growth hormone is most commonly present in Stages III or IV of sleep. What may occur in the body is that melatonin is secreted before and in the onset of sleep. Then with the approach of Stages III and IV, growth hormone is present. Once the body reaches REM, a time also associated with melatonin, growth hormone is absent once again. Perhaps melatonin is responsible for interfering with growth hormone. In a recent study, significantly less growth hormone was secreted by patients suffering from depression (Jarrett, Kupfer, Miewald et al., 1994). Perhaps these patients had too much melatonin that interfered with their growth hormone production. Wilson & Foster (1985) noted that administering melatonin to depressed patients increased self-rating of depression, and increased degree of insomnia.

In another recent study, the use of sleep deprivation was used as a treatment for depression. The patients were kept awake, and subjected to bright and dimlight treatment. Subsequently, there was a 50% decrease in the Hamilton Rating Scale for depression by some of the patients (Rao, Muller-Oerlinghausen, Mackert & Strebel, 1992). This treatment involving light exposure would prevent melatonin from being released into the blood. During the winter months, people are exposed to much less light because of the shorter days. Accompanying these winter months is a greater amount of depression in people. With this little amount of light, melatonin will be released more frequently, which may be the cause of the increase of depression in people. Many situations exist where limited exposure to light may produce depression.

In conclusion, the hormone melatonin is secreted by the pineal gland during the night under natural conditions. Melatonin is important in the balance of circadian rhythm, and has been shown to be an effective treatment for sleep dysfunction. There may also exist a link between the presence of melatonin and depression. The prominence this hormone has in the functions of sleep and in the balance of our lives is fascinating, and should receive further study.

Bibliography


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**Schools Participating in the Essay Contest**

Entries were received from the following high schools:

**CANADA**
Grimsby Secondary, Grimsby, Ontario

**United States**
La Canada High School, La Canada, California
La Jolla High School, La Jolla, California
Palm Springs High School, Palm Springs, California

Cooper City High School, Cooper City, Florida
Westminster Schools, Atlanta, Georgia

Illinois Mathematics and Science Academy, Aurora, Illinois
Carbondale Community High School, Carbondale, Illinois
Naperville North High School, Naperville, Illinois

Central Noble High School, Albion, Indiana
Winfield High School, Winfield, Kansas
North Dartmouth High School, North Dartmouth, Massachusetts
OSMTECH Academy, Clarkstown, Michigan
Edina High School, Edina, Minnesota
Marquette High School, Chesterfield, Missouri
Uta Halee High School, Omaha, Nebraska

Academy of Saint Aloysius, Jersey City, New Jersey
Pingry School, Martinsville, New Jersey
Montclair High School, Montclair, New Jersey
Hills High School, Oakland, New Jersey

W.C. Mepham High School, Bellmore, New York
Canandaigua Academy, Canandaigua, New York
Cold Spring Harbor High School, Cold Spring Harbor, New York
Walter Panas High School, Cortlandt Manor, New York
Croton Harmon High School, Croton-on-Hudson, New York

Garden City High School, Garden City, New York
Rye Neck High School, Mamaroneck, New York
Hendrick Hudson High School, Montrose, New York
Blind Brook High School, Rye Brook, New York

Conestoga Senior High School, Berwyn, Pennsylvania

East Providence Senior High, East Providence, Rhode Island
Smithfield High School, Smithfield, RI

Abbeville High School, Abbeville, SC

Allen High School, Allen, Texas
Bryan Adams High School, Dallas, Texas
Chester W. Nimitz High School, Irving, Texas
Dr. Ralph H. Poteet High School, Mesquite, Texas
J.J. Pearce High School, Richardson, Texas

Provo High School, Provo, Utah
Sequim High School, Sequim, Washington

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Judges

A two-tiered judging system was employed. The following individuals served as judges with the thanks of the Program Committee:

Joel Bennington, Ph.D.
Linda Dickerson, Ph.D.
Wendy Hunt, V.M.D.
Christopher Leonard, Ph.D.
Ann Miller

David Rector, Ph.D.
Christopher Richard, Ph.D.
Martha Rosenthal, Ph.D.
James P. Shafferly, D.Phil.
Teresa Steinenger, Ph.D.

NEWS AND COMMENTS

NATIONAL SLEEP FOUNDATION GRANTS

The National Sleep Foundation has established a fellowship program to fund pre-or postdoctoral scientists in the study of sleep or circadian rhythms. Applicants must be U.S. citizens or resident aliens in recognised programs of study or laboratories with strong mentorship in the appropriate area. Fellowships are available for basic, applied or clinical research. Minorities are encouraged to apply. **Deadline for receipt of applications is January 1, 1996**

Application materials and additional information may be obtained from:

The National Sleep Foundation
1367 Connecticut Avenue, NW
Suite 200
Washington, D.C. 20036
Telephone: 202-785-2300
Fax: 202-785-2880
natsleep@haven.ios.com

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CLUB HYPNOS

Members who will be attending the Society for Neuroscience meeting this fall should plan to attend the first annual reception held by Club Hypnos on Tuesday, 11/14 from 5:30 to 7:30. The Board authorized Adrian Morrison and Michael Chase to organize this SRS-sponsored event as a way to publicize opportunities in sleep research. We are especially eager to have uncommitted students get to know our field. Look for more details in the final meeting program.

Obituaries

**Lucien Jassy, M.D.,** Medical Director of the Sleep Disorders Center at Mercy Hospital in San Diego, passed away on Friday, August 25, 1995. He was 47. Cause of death was unexpected heart attack. Lucien was a member of ASDA.

**Nobuyuki Okudaira, M.D.,** Tokyo Japan. He was 51. He was a long standing member of SRS, and a fine sleep researcher and clinician.
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