Pinel Lecture Notes - Chapter 1 | Pinel Chapter 2 | Pinel Chapter 3 | Pinel Chapter 4 - Neural Conduction and Synaptic Transmission | Pinel Chapter 5 - Research Method in Biopsychology | Pinel Chapter 6 - Human Brain Damage & Animal Models | Pinel Chapter 9 | NS Development | Pinel Chapter 10a - Eating | Pinel Chapter 11 - Hormones & Sex | Pinel Chapter 12 - Sleep | Chapter 13 - Drug Action and Addiction | Chapter 15 - Neuroplacticity | Biopsychology of Stress and Illness

Lecture 12a THE PHYSIOLOGICAL AND BEHAVIORAL CORRELATES OF SLEEP AND DREAMING

Outline

- 1. Three Psychophysiological Measures of Sleep
- 2. The Five Stages of Sleep EEG
- 3. REM Sleep and Dreaming
- 4. Sleep Disorders
 - a. Insomnia
 - b. Hypersomnia
 - c. REM-Sleep-Related Disorders

Lecture Notes

1. Three Psychophysiological Measures of Sleep

- in the 1930s, it was discovered that although <u>EEG</u> waves are generally high-voltage and slow during sleep, there are periods during sleep when they are similar to the low-voltage fast waves of wakefulness
- in 1953, Aserinsky and Kleitman discovered that <u>rapid eye movements</u>
 (REMs) occurred under the eyelids of sleeping subjects during the
 periods of low-voltage fast EEG activity
- in 1962, Berger and Oswald found that there was a dramatic decline of **EMG** activity in the muscles of the body core during these same sleep periods
- since these three discoveries, EEG, EOG (electrooculogram), and EMG have been monitored in most sleep experiments

2. The Four Stages of Sleep EEG (use Digital Image Archive Figure CH12F02.BMP)

- just before a subject falls asleep, the EEG is typically punctuated by bursts of alpha waves (large-amplitude, regular, 8-to-12-Hz waves indicative of relaxed wakefulness)
- when the subject falls asleep, the EEG progresses in sequence through initial stage 1, stage 2, stage 3, and stage 4

- initial stage 1 sleep EEG is low-voltage, fast activity similar to, but slightly slower than, that of wakefulness
- stage 2 sleep EEG is of higher voltage and slower than stage 1; its most obvious characteristics are K complexes (a single large negative wave followed by a single large positive wave) and sleep spindles (1-to-2-second waxing-and-waning bursts of 12-to-15-Hz waves)
- stage 3 sleep EEG is defined by the occasional presence of delta waves,
 the largest and slowest EEG waves (1 to 2 per second)
- **stage 4 sleep EEG** is defined by a predominance of delta waves
- once the sleeping subject has gone through initial stage 1 to stage 4, he or she goes back through the stages to stage 1 EEG
- but when the subject returns to stage 1 EEG, it is referred to as <u>emergent</u> <u>stage 1 EEG</u>, as it is every other time that the subject enters stage 1 during that night's sleep; it is called "emergent" stage 1 because it emerges from the other stages
- it is important to distinguish between initial stage 1 EEG and emergent stage
 1 EEG because only emergent stage 1 EEG is associated with REMs and low levels of muscle tonus in core muscles
- the stage during which emergent stage 1 occurs is often called <u>REM sleep</u> or <u>paradoxical sleep</u> (it was initially considered a paradox that subjects slept while their EEGs suggested that they were awake); stages 2, 3, and 4 together are often referred to as <u>slow-wave sleep</u>; stages 3 and 4 together are often referred to as <u>delta sleep</u>
- the progression of EEG stages changes during a typical night's sleep; notice: (1) that each cycle is about 90 minutes long, (2) that as the night progresses less time is spent in stages 3 and 4 and more is spent in REM sleep, and (3) that there are brief periods of wakefulness, which are normally forgotten in the morning (use Digital Image Archive Figure CH12F03.BMP)

3. REM Sleep and Dreaming

- the discovery of REMs in 1953 by Kleitman and his colleagues led to the obvious hypothesis that REM-sleep periods were periods of dreaming; this hypothesis was confirmed by waking subjects up during various stages of sleep and asking them if they had been dreaming; about 80% of awakenings from REM sleep led to dream reports, and only 7% of awakenings from nonREM-sleep

stages led to dream reports

- in the years since this discovery, a number of common beliefs about dreaming have been objectively tested by using EEG, EMG, and EOG indices of dreaming:
 - (1) Are <u>external stimuli</u> incorporated into dream sequences? (yes, dripping water was in 14 out of 33 cases);
 - (2) Do dreams run on <u>"real time"</u>? (yes, subjects awakened 5 or 15 minutes after the beginning of a dream could guess the correct interval on the basis of the contents of their dreams);
 - (3) Does everybody dream? (yes, even people who claimed that they did not dream had normal amounts of REM, and they reported dreams if they were awakened during REM--although less frequently);
 - (4) Are <u>penile erections</u> indicative of dreams with sexual content? (no, penile and clitoral tumescence occurs during all dreams, regardless of sexual content);
 - (5) Are <u>somnambulism</u> and <u>sleep talking</u> the acting out of dreams? (no, they usually occur during stage 4);
 - (6) Are dreams the **reliving** of stressful or sexual events of the previous day? (there is no good evidence for this)
- The Freudian theory of dreams, that dreams represent unacceptable wishes, was based on science and beliefs of the 1890's and has no support from today's science base
- Hobson's has proposed an <u>activation-synthesis theory</u> of dreaming, based upon the idea that the information passed on to the cortex during REM is random, and dreams are the cortex's effort to make sense of these random signals
- A few people have <u>lucid dreams</u> in which the dreamer knows that they are dreaming and can change the course of their dreams; lucid dreams are usually positive experiences

4. Sleep Disorders

 sleep disorders are of two major kinds: <u>insomnia</u> (complaints of too little sleep) and <u>hypersomnia</u> (complaints of too much sleep and tiredness)

a. Insomnia

- insomnia is often <u>iatrogenic</u> (physician induced); a person with minor difficulties in sleeping is often given sleeping pills (usually <u>benzodiazepines</u> such as Valium or Librium); <u>tolerance</u> develops to benzodiazepines so the patient starts to take larger and larger doses, and when they try to stop taking them, severe insomnia occurs as a <u>withdrawal symptom</u>; this causes them to return to their drug taking (see the case of Mr. B. in BIOPSYCHOLOGY); they become locked in a vicious circle
- another cause of insomnia is <u>sleep apnea</u>; in some patients, spasms of the throat muscles block air intake several times a night; if they do not recall their many awakenings, their excessive sleepiness the next day can lead to a diagnosis of hypersomnia
- nocturnal myoclonus is another cause of insomnia; subjects are briefly
 awakened each night by sudden twitches of the legs; it can lead to a
 diagnosis of hypersomnia if the patient does not recall the awakenings
 because he or she tends to be excessively sleepy during the day
- <u>restless legs</u> is a hard-to-describe tension in the legs that keeps people from falling asleep; this leads to a diagnosis of insomnia
- because benzodiazepines have muscle relaxant, anxiolytic, and anticonvulsive effects in addition to their hypnotic effects, they are often prescribed for nocturnal myoclonus and restless legs; unfortunately, they are rarely effective
 - one large-scale study showed that people seeking help for insomnia claim that they sleep an average of 4.5 hours per night, but they actually sleep an average of 6.5 hours per night; it used to be the practice to assume that people who complained of insomnia but slept more than 6.5 hours per night were neurotic, and they were diagnosed as **pseudoinsomniacs**;

this practice stopped when psychophysiological studies found that many of the people who had been diagnosed as neurotic were suffering from sleep-disturbing problems such as sleep apnea or nocturnal myoclonus; it seems that undisturbed sleep rather than sleep per se is what we need to feel rested the next day

b. Hypersomnia

- the most interesting disorder of hypersomnia is **narcolepsy**; narcoleptics tend

- to fall asleep in totally inappropriate situations (while having conversations, eating, or scuba diving), they usually sleep for 10 or 15 minutes and then awaken refreshed
- narcoleptics sleep only about one hour more per day than is average but often sleep at inappropriate times
- sleep promoting conditions are called <u>soporific</u>

c. REM-Sleep-Related Disorders

- narcoleptics go directly into REM sleep rather than going through other sleep stages, thus narcolepsy is often categorized with REM-sleep-related disorders
- cataplexy is commonly associated with narcolepsy; it is a sudden loss of muscle tone, sometimes triggered by an emotional event; in its extreme form, the sufferer drops as if shot and stays there for a minute or two while remaining conscious; the observation that narcoleptics, unlike normal people, go directly into REM sleep when they fall asleep has led to the view that a cataleptic attack is the muscle relaxation of REM sleep encroaching on wakefulness
- the <u>nucleus magnocellularis</u> of the caudal reticular formation normally becomes active during REM sleep to enforce muscle relaxation during dreaming; these cells become active during cataleptic attacks in dogs; presumably it is failure of the nucleus magnocellularis that causes the REM-sleep disorder in which people act out their dreams.
- hypersomnia is treated with <u>stimulants</u>; cataplexy is treated with <u>tricylic</u> <u>antidepressants</u> because they block REM sleep
- patients treated with tricyclic antidepressants and some individuals with brain damage do not experience REM sleep; there seem to be no adverse effects

Suggested Websites for Lecture 12a:

Sleep: http://www.ninds.nih.gov/healinfo/DISORDER/SLEEP/brain-basics-sleep.HTM

for a comprehensive review of sleep, sleep stages, sleep pathology; complete with figures of EEG stages in sleep, neural structures involved in sleep, and dreaming and REM.

The Sleep Well: http://www.stanford.edu/~dement/index.html

the site maintained by Dr. Daniel Dement, a pioneer in sleep research.

The Science of Sleep:

http://www.real.com/rafiles/npr/password/nf6m2201-5.ram

from National Public Radio, a RealTime audio interview with Dr. Clifford Saper, Dr. David White and Dr. Craig Heller on the Science of Sleep.

LECTURE 12 b -- WHY DO WE SLEEP?

Outline

- 1. Why Do We Sleep?
 - a. Recuperation Theory
 - b. Circadian Theory
- 2. Circadian Sleep-Wake Cycles
- 3. The Effects of Sleep Reduction
 - a. Total Sleep Deprivation
 - b. REM-Sleep Deprivation
 - c. Sleep Reduction
- 4. Recuperation and Circadian Models Combined

1. Why Do We Sleep?

 all mammals and birds sleep; even fish, reptiles, amphibians, and insects go through periods of inactivity and unresponsiveness that are remarkably like mammalian

sleep

- this suggests that sleep fulfills some very important function, but what is it?

a. Recuperation Theory

- the essence of various <u>recuperation theories</u> of sleep is that being awake <u>disrupts homeostasis</u> in some way and that sleep is required to restore it
- this is the way that most people think about sleep

b. Circadian Theory

 according to the <u>circadian theory</u> of sleep, sleep is <u>not a response to an</u> <u>internal</u>

imbalance:

sleep is an <u>adaptive</u> response that evolved to <u>conserve energy</u> and to <u>protect</u> organisms from <u>mishap</u> and predation;

the <u>urge to sleep</u> evolved to be <u>greatest</u> during the night for <u>animals who do</u> <u>not see well</u> in dim illumination

- the circadian theory of sleep considers sleep to be like sexual behavior; it is adaptive
 - and there is a **strong drive** to engage in it, but its **purpose** is **not** to correct an inner

deficiency

- two lines of research have a direct bearing on whether sleep is fundamentally recuperative or circadian:
 - (1) the study of circadian sleep cycles and
 - (2) the study of the effects of sleep reduction

2. Circadian Sleep-Wake Cycles

- almost every physiological function in surface-dwelling animals displays some kind
 - of circadian rhythmicity;

the sleep-wake cycle is the most obvious

- the <u>light-dark cycle</u> is an important factor in the timing of most <u>circadian</u> <u>rhythms</u>;
 - environmental cues (such as the light-dark cycle) that <u>entrain</u> circadian rhythms are

called **zeitgebers**

- however, sleep-wake cycles still cycle regularly in a constant environment;
 - <u>regular biological cycles</u> in constant environments are called <u>free-running</u> <u>cycles</u>;

the duration of a free-running cycle is its **free-running period**

(use Digital Image Archive Figure CH12F03.BMP;

a free-running sleep-wake cycle of a man living in an unchanging environment;

notice that the free-running period is **25.3 hours**;

free-running periods are usually greater than 24 hours)

 even animals raised from birth in unchanging laboratory environments display highly

regular free-running cycles;

circadian cycles thus do not appear to be learned

- the fact that such <u>regularity is precisely maintained</u> despite large day-today
 - variations in physical and mental activity is **strong support for the circadian theory** of sleep;
 - in fact, several studies have found a <u>negative correlation</u> between the <u>length</u> of a
- period of <u>wakefulness</u> and the <u>length</u> of the <u>following period of sleep</u>, even under free-

running conditions

(use Digital Image Archive Figure CH12F06.BMP);

this is the **opposite** of what the **recuperation** theory would **predict**;

- it seems that we are <u>programmed to go to sleep every 24 hours</u> or so and that if we <u>stay awake longer</u> than usual during a particular 24-hour cycle, <u>there is less time left for sleep</u>
- the <u>suprachiasmatic nuclei</u> (SCN) of the hypothalamus appear to contain the

circadian timing mechanism;

- SCN <u>lesions abolish</u> most <u>circadian cycles</u>, and the <u>neurons</u> of the <u>SCN</u> display <u>circadian patterns of activity</u>
- <u>light zeitgebers</u> entrain circadian cycles via a path from the retina to the SCN, the

retinohypothalamic tract (use Digital Image Archive Figure CH12F16.BMP);

- lesions of the retinohypothalamic tract eliminate the ability of light to entrain circadian rhythms
- Alternating <u>shift work</u> and <u>jet lag</u> are situations where zeitgebers are <u>phase</u> <u>advanced</u> (moving to an earlier shift or flying east) or <u>phase delayed</u> (moving to a later

shift or flying west);

people <u>must adjust</u> their natural sleep-wake cycles or <u>endure problems</u> such as sleep disturbances, fatigue, and performance decrements;

phase advances are more difficult adjustments than are phase delays

3. The Effects of Sleep Reduction

 the second line of research that has a direct bearing on the question of whether sleep

is fundamentally circadian or recuperative is the research on sleep reduction

- the recuperation theory <u>predicts</u>:
- (1) that long periods of wakefulness will result in **debilitating physiological deficits**,
- (2) that these deficits <u>will grow steadily worse</u> as the sleep deprivation continues, and
- (3) that <u>after</u> the sleep deprivation has <u>ended</u>, much of the <u>lost sleep will be</u> <u>regained</u>

a. Total Deprivation

- the above predictions are seriously <u>challenged</u> by the documented cases of individuals such as Miss M., the elderly lady, who seemed to <u>sleep only out of boredom</u>- and then only about 1 hour per night
- there are also many studies in which subjects have been <u>totally deprived of</u> <u>sleep</u> for

several days;

these studies have **not confirmed the predictions** of the recuperation theory:

(1) <u>no marked physiological disturbances</u> other than <u>increased</u> <u>sleepiness</u> have

been discovered in sleep-deprived subjects although there have been many

attempts to document them;

(2) during sleep deprivation, subjects <u>do not become progressively</u> more tired:

their <u>sleepiness follows the usual circadian cycle</u>, and there appears to be <u>no</u>

increase in sleepiness after the third day;

- (3) after the termination of several days of total sleep deprivation, subjects sleep
- about 3 hours extra the first night and about an hour extra the next night, but then

they return to their previous sleep schedule

 one might expect that sleep deprivation would produce severe performance deficits,

particularly on complex tasks; study after study has shown this not to be the case;

sleep-deprived subjects display some performance deficits but primarily on **boring**.

passive tasks, requiring continuous attention;

such deficits have been attributed to **microsleeps** (2-or-3-second periods during

which subjects remain sitting or standing but their eyelids droop, they are **unresponsive** to external stimuli, and they **have a sleep EEG**)

- several experts who have reviewed the sleep-deprivation literature have reached the
- same general conclusion: "there is yet no evidence that sleep deprivation does anything

to humans other than to increase their tendency to fall asleep"

the recuperative theory also has <u>difficulty explaining</u> why some animals <u>sleep so</u>

<u>little</u> (horses sleep 2 hours per day) while <u>others sleep so much</u> (cats sleep 14 hours per

day and giant sloths sleep 20 hours per day)

b. REM-Sleep Deprivation

because REM sleep is associated with dreaming, there has been much interest in the

effects of the selective deprivation of REM sleep

- there are **two major effects** of REM sleep deprivation:
 - (1) REM deprivation is enforced by waking subjects up each time that REMs begin

to occur; as a period of REM deprivation progresses, <u>subjects must be</u> awakened more

and more frequently to prevent them from having periods of REM sleep; and

(2) <u>after REM deprivation</u> is curtailed, <u>REM rebound</u> is often seen; subjects get

more than their normal amount of REM sleep for the next two or three nights

- one of the main challenges for anyone suggesting that REM sleep is critical to normal functioning must explain a fact about why, patients who have taken tricyclic
- <u>antidepressants</u>, (which block REM sleep at common therapeutic doses) regularly for

years, have experienced no adverse psychological side effects that can be attributed to

REM deprivation

c. Sleep Reduction

- several studies have maintained subjects on programs of sleep-reduction
- a study by Friedman, Mullaney and their colleagues is the most thorough of such

studies;

8 subjects were asked to <u>reduce their sleep in 30-minute steps</u> every few weeks

until they felt that they did not want to reduce it further;

then they slept at this shortest time for 1 month and at the shortest time plus 30

minutes for 2 months;

finally, the subjects' natural sleep time was measured 1 year after they had returned to sleeping for as long as they pleased

- the results were:
- (1) the achieved **minimum duration of nightly sleep** was 5.5 hours for two subjects, 5.0

hours for four subjects and 4.5 hours for two subjects;

- (2) there was a major **increase in sleep efficiency** (less time to fall asleep, fewer awakenings, more stage 4 sleep);
- (3) there were <u>no deficits</u> on any of the numerous performance and psychological tests given throughout the experiment;

(4) a year after the period of sleep restriction, <u>all subjects were naturally</u> <u>sleeping</u>

between 7 and 18 hours less per week

- 4. Recuperation and Circadian Models Combined (use Digital Image Archive Figure CH12F10.BMP)
- experimental evidence seems to come down strongly in favor of the circadian theory

but several <u>recent findings support the theory that both circadian and</u>
<u>recuperative factors</u>

are important;

- thus some of the sleep that we get may serve a recuperative function, and some simply serves to conserve energy and keep us safe
- support for this combination theory has taken a long time to accumulate because in

most sleep-deprivation studies only the total duration of sleep has been recorded, rather

than the duration of the various sleep stages

- the following lines of evidence suggest that <u>slow-wave sleep serves a</u> <u>recuperative</u>

function but that stage 2 and REM sleep do not:

- (1) sleep-deprived subjects <u>regain much of their lost stage 4 sleep</u> after the period of sleep deprivation;
- (2) subjects who reduce their sleep time do so by <u>reducing their REM sleep and</u> sleep;
- (3) **short sleepers** typically get as much **stage 3 and stage 4 sleep as long sleepers**;
- (4) <u>extra sleep</u> obtained in the <u>morning</u> contains almost <u>no stages 3 and 4</u>, and it does not reduce the time spent sleeping the next night;
- (5) after sleep deprivation, the proportion of slow waves in the EEG is

<u>higher</u>

 the evidence from sleep-reduction studies suggests that most individuals can adapt to

sleep schedules of about 51/2 hours per night

What do you think of this conclusion?

Do you think that it would be worth the effort of reducing your sleep time to create

more waking hours for yourself?

What kind of individuals would have the least difficulty reducing their sleep time?

 How might you rearrange your schedule during the day to make sleep reduction more

feasible?

Suggested Websites for Lecture 12b:

Biological and Circadian Rhythms:

http://www.epub.org.br/cm/n04/mente/cloks_i.htm

From the Brain and Mind site, an overview of the neural structures involved in establishing circadian rhythms; good figures, and links to related sites.

See also:

http://www.sfu.ca/~mcantle/rhythms.html

From Dr. Ralph Mistleberger at Simon Fraser University; see the overview of biological

rhythms and his links to other "sites with rhythm".

You might also look at:

http://www.srbr.org/

the site maintained by the Society for Research on Biological Rhythms.

Sleep Deprivation: http://www.mhsource.com/edu/psytimes/p980301b.html

A provocative article by Dr. Stanley Cohen, highlighting the possible negative effects of long-term sleep deprivation.

Melatonin and Sleep:

http://www.sciam.com/explorations/040196explorations.html

From Scientific American, a critical look at the "drug of darkness", the neurohormone and putative sleep aid melatonin.