Vanguard University
School for Professional Studies
Degree Program

"BIOLOGICAL PSYCHOLOGY"
PSYD 340

Student Guide

6/09
DS 01/10

VANGUARD UNIVERSITY
EDUCATIONAL TARGETS AND GOALS

Course content and activities have been designed to meet Vanguard University's Educational Targets and Goals in the areas of Intellectual Engagement, Spiritual Formation, Professional Excellence, Aesthetic Expression, Responsible Stewardship, and Sociocultural Responsiveness. The complete list of Targets and Goals follows.

INTELLECTUAL ENGAGEMENT

Students will

- Learn to think critically and evaluate evidence rationally
- Acquire and continue to use skills for learning
- Utilize research methods for the expansion of knowledge and problem solving
- Integrate learning with Christian faith and living and
- Develop the ability to communicate the fruits of learning and research clearly and effectively.

SPIRITUAL FORMATION

Students will

- Understand Christian existence as a journey that integrates human experience with personal faith
- Gain an appreciation for the value of participation in communities of believers and
- Develop and maintain a biblically based and theologically sound Christian lifestyle of personal and social responsibility

PROFESSIONAL EXCELLENCE

Students will

- Understand current theories and practices in their respective academic disciplines in the context of the liberal arts and sciences
- Develop lifelong skills for communicating and performing professionally
- Achieve technological competence in acquiring and processing information
- Acquire interpersonal ability to work harmoniously with others, and
- Internalize a strong sense of professional ethics

AESTHETIC EXPRESSION

Students will

- Understand various sources of aesthetic sensitivity and expression as inherent human endowments and part of God's creation
- Gain an awareness, understanding, appreciation, and expression of the fine and performing arts and
- Develop interpretive frameworks of aesthetic truths and values for personal wholeness and community enrichment

RESPONSIBLE STEWARDSHIP

Students will

- Adopt a lifestyle of personal health and well-being
- Appreciate the value of family and other meaningful relationships
- Exhibit the responsibilities of citizenship in society
- Gain a global outlook in caring for the environment and in promoting social justice and economic empowerment and
- Promote the church's mission through community service

SOCIOCULTURAL RESPONSIVENESS

Students will

- Demonstrate a capacity to challenge personal prejudices, appreciate cultural diversity, and learn from other cultures
- Develop a commitment to pursue peace, justice, and reconciliation in a pluralistic society and
- Celebrate the differences of race, ethnicity, gender, and age within the biblical vision of inclusiveness and the equal value of all people.

COURSE DESCRIPTION

PSYD 340 • Biological Psychology (3 units)

Prerequisite: PSYD 366. A study of human behavior in terms of the nervous system and its control of the activity of the muscles, glands, and the biochemistry of the body, with special emphasis on perception, sensory and motor functions, motivation, emotion, learning, and memory. Neurological impairment and disorders are examined.

This course is an introduction to the concepts of biological psychology. Beginning with a basic understanding of brain structures, the student will gain understanding of how brain structure relates to brain function and behavior. Content of this course includes brain and nervous system structure, neuronal communication, drugs and hormonal influences on the brain, visual sensation and perception, sleep, sexual behavior, lateralization, language, and psychological disorders. Learning activities include text reading, lecture, video, video reactions, session quizzes, session writing assignments, and comprehensive objective evaluation. Learning activities support Vanguard University's Educational Targets and Goals for students.

STUDENT LEARNING OUTCOMES

The student will demonstrate a competent knowledge base in the following areas.

- Identify basic brain structures and related functions.
- Describe contemporary research methods for studying brain and behavior
- Understand sensory and motor divisions of the nervous system
- Describe structure and function of neurons.
- Understand how neurons communicate
- Describe the role of neurotransmitters
- Understand how drugs and hormones influence behavior
- Understand basic brain development
- Understand brain structure and function in visual sensation and perception
- Understand the stages of sleep and the importance of REM sleep
- Understand biological and social origins of sexual behavior
- Understand lateralization of brain function and language development
- Understand biological elements of common psychological disorders
- Learn terminology associated with Biological Psychology

TEXT

Required text:

Freberg, L. A. (2010). *Discovering Biological Psychology* (2nd ed.). Belmont, CA; Wadsworth. ISBN-13: 978-0-547-17779-3; ISBN-10: 0-547-17779-8

Note: A "Companion Site" is provided by the publisher of this text, providing valuable study aids for each chapter of the text. To register for the Companion Site, go to http://www.cengage.com/login/ and click "Create My Account." To access the Companion Site, go to http://www.cengage.com/highered/, enter "freberg" in the search box at the top, and click on Student "Companion Site." Select a chapter from the drop-down menu and click on the resource you wish to access (for example, "Tutorial Quiz").

STUDENTS: Please note that during Session 1 there will be a 25-point quiz covering chapters 1 and 2 of the text.

Within the first hour of class (Sessions 1-4), a quiz covering material assigned for that Session will be given. The quizzes may include term definitions, multiple-choice, true/false, fill-in-the-blank, short-answer essay questions, and drawing and/or labeling of diagrams. To prepare for the weekly quizzes, students are encouraged to study the assigned text chapters and use the resources on the "Companion Site."

COURSE POLICIES

- **ATTENDANCE AND TARDY POLICY**
  You must attend class on time and remain present until dismissed. Class attendance is necessary in order to complete the course. The School for Professional Studies relies on the dynamics of class interaction and group processing in order to integrate and apply the learning of academic content. This model also emphasizes the development and practice of interpersonal communication skills and teamwork (e.g., group problem solving and negotiation). The format therefore necessitates class attendance. In practical terms, one course session is equivalent to three weeks of traditional semester course work.

  Students who miss more than one class meeting (or more than five class hours) in any given course will automatically receive a failing grade and need to retake the course to obtain a passing grade. If an instructor deems that a student's second absence was under extremely unavoidable and unusual circumstances (i.e., an auto accident), the professor may file an academic petition on behalf of the student to the Director of SPS. If the academic petition is approved, the student will be given a "W" (Withdrawal) in place of a failing grade. The student will still be required to retake the course.

  Students who arrive late disturb the class. At the professor's discretion, students who arrive late may not receive participation points for the unit covered. Students who are habitually late may be asked to drop the course.

- **CLASS PARTICIPATION**
  You must be prepared and participate in all discussions. Criterion: Is the student engaged in classroom discussions? Does the student demonstrate an ability to handle assigned material with a degree of proficiency? (E.g., demonstrate the type of questions and issues consistent, and reflecting a familiarity with the assigned material). Participation evaluated according to quality, not quantity, of participation. Attendance will be scored, and no participation points will be awarded if the student is absent.

- **SUBMISSION OF FINAL EXAMS / PAPERS**
  The School for Professional Studies office does not assume responsibility for any final papers. No homework or final papers will be accepted for professors in the SPS office, nor will final papers be returned to students through the SPS office.

  The method for the submission of homework and the final exam or final paper will be determined by the professor. The professor will discuss the method which will be employed during the first night of class. All exchanges of papers will be between the student and the professor.

- **LATE PAPER POLICY**
  You are responsible for submitting assignments on time (by 6:00 pm). Unless authorized by the Professor in advance, no credit will be given for assignments not turned in when due.

- **ACADEMIC DISHONESTY**
  Work submitted for assessment purposes must be the independent work of the student concerned. Plagiarism, or copying and use of another's work without proper acknowledgment, is not permitted. Nor is it permissible for any former or present student to allow another student to refer to, use as a sample, or in any way copy or review their work. If a student needs guidance, he or she must seek the Professor's assistance.

- **DISABILITY SERVICES**

  For students with documented medical or psychological disabilities, please contact the Coordinator of Disability Services to request reasonable accommodations. The Coordinator of Disability Services is located in the Counseling Center on the second floor of the Scott Academic Center and can be reached at extension 4489 or by email at disabilityservices@vanguard.edu

  For students with a documented learning disability who would like to request appropriate accommodations, please contact the Director of Learning Skills, located upstairs in Scott Academic Center at extension 2540 or by email at disabilityservices@vanguard.edu

- **DIVERSITY STATEMENT**

  The School for Professional Studies intends to foster a Christ-centered community that promotes appreciation and respect for individuals, enhances the potential of all members, and values differences in gender, race, abilities, and generation. As such, we endeavor to communicate with honesty, to speak with encouraging and edifying words, and to create a safe environment in our classes and interactions.

STUDENT EVALUATION

%	Points	Grade	Significance	GPA
93-100%	930-1000	A	Exceptional	4.00
90-92.9%	900-929	A-		3.67
87-89.9%	870-899	B+		3.33
83-86.9%	830-869	B	Good	3.00
80-82.9%	800-829	B-		2.67
77-79.9%	770-799	C+		2.33
73-76.9%	730-769	C	Satisfactory	2.00
70-72.9%	700-729	C-		1.67
67-69.9%	670-699	D+		1.33
63-66.9%	630-669	D	Poor	1.00
60-62.9%	600-629	D-		0.67
00-59.9%	000-599	F	Failure	0.00

Students in this course will be evaluated by the university's 4.0 grading system. Grades will be assigned based on the points earned in the class as follows.

Attendance	
(40 pts each for Sessions 1-5)	200 pts
Session Quizzes	
(25 pts per quiz, Sessions 1-4)	100 pts
Session Assessments	
(100 points per assessment, Sessions 2-5)	400 pts
Video Reactions	
(25 pts each for Sessions 1-4)	100 pts
Comprehensive Exam (Session 5)	200 pts
Total	1000 pts

Session Quizzes: Within the first hour of class (Sessions 1-4), a quiz covering material assigned for that Session will be given. The quizzes may include term definitions, multiple-choice, true/false, fill-in-the-blank, short-answer essay questions, and drawing and/or labeling of diagrams. To prepare for the weekly quizzes, students are encouraged to study the assigned text chapters and use the text resources on the "Companion Site."

Session Assessments: Hard copies of Session Assessments are due in class by 6:00 p.m. (Session 2 through Session 5). Each Session Assessment has five questions, with each question worth 20 points. Responses to the Session Assessment questions are to be typed and double-spaced with proper spelling and punctuation. Use APA style general document guidelines: one-inch margins, 12-pt font size, Times Roman or Courier typeface. Typical length of Assessment responses will be 1-2 pages per question. For each question, re-write the question at the beginning of your response. Session Assessments will be evaluated on thoroughness, clarity, and accuracy of the content.

Video Reactions: In-class written reactions to each session's video (Sessions 1-4) will be completed. The video reactions should summarize the key content of the video, relate the content of the video to material presented in class, and should include a personal reaction to the content of the video. Each video reaction will be worth a maximum of 25 points. Notes should be taken during the videos; video reaction forms will be distributed immediately following each video. Students will be given 15-20 minutes to complete their video reactions.

Comprehensive Exam: An objective exam worth 200 points will be given during the final ninety minutes of Session 5. All major topics covered in class will be represented on the exam. Additional information about the type of questions to be included on this exam will be provided by the instructor. In order to preserve for future classes the integrity of the exam questions used, the exam booklets will be collected by the instructor and will not be returned to students. Students who wish to examine their work on the exam may schedule a meeting with the instructor to review their work.

LOGISTICS CHART

Hr	Session 1	Session 2	Session 3	Session 4	Session 5
1	Professor's Introduction Review Student Guide Quiz 1 Lecture: Chapters 1,2	Sharing Quiz 2 Lecture: Chapters 3,4	Sharing Quiz 3 Lecture: Chapters 6,10	Sharing Quiz 4 Lecture: Chapters 11,13	Sharing Lecture: Chapter 16
2	Lecture: Chapters 1,2 **Break**	Lecture: Chapters 3,4 **Break**	Lecture: Chapters 6,10 **Break**	Lecture: Chapters 11,13 **Break**	Lecture: Chapter 16 **Break**
3	Lecture: Chapters 1,2 Brain Video	Lecture: Chapters 3,4 Brain Video	Lecture: Chapters 6,10 Brain Video	Lecture: Chapters 11,13 Brain Video	Exam
4	Brain Video, Reaction	Brain Video, Reaction	Brain Video, Reaction	Brain Video, Reaction	Exam

Session ONE
Chapters 1 and 2

Chapter 1: Introducing Biological Psychology
Chapter 2: The Anatomy of the Nervous System

ACTIVITIES
Introduction of Professor and Students
Overview of Biological Psychology: Course Description
Overview of Course Expectations
Student Guide
Attendance
Assessments
Quizzes
Video reactions
Comprehensive Exam
Quiz: Chapters 1 and 2
Lecture: Chapters 1 and 2
Break
Lecture: Chapters 1 and 2
Video
Secret Life of the Brain: The Baby's Brain: Wider Than the Sky
Less than a month after conception, brain cells are developing at the astonishing rate of 500,000 per minute. The brain will ultimately comprise billions of cells linked by trillions of connections, the most complex thing in the universe. How does it organize itself? What are the roles of genetics and environment in brain development? The first hour traces the formation of the infant brain through the age of one, the period during which it is most open to molding through external influence and experience.
Video Reaction (in-class)

CHAPTER 1
Introducing Biopsychology
Lecture Outline

I. Biological Psychology as an Interdisciplinary Field (p. 3)
   A. **Biological psychology** is an interdisciplinary area of study involving psychology, biology, biochemistry, the neural sciences, and related fields.
   B. Researchers have a specific interest in the relationships between the nervous system and behavior.
      These relationships between biology and behavior are reciprocal in nature. Biology can influence behavior as is the case of increased levels of testosterone being associated with increased aggression and likewise behavior can influence biology as is the example when watching your favorite sports team lose produces a decrease in the level of circulating testosterone.

II. Highlights in the Biopsychology Timeline (pp. 3-7)
   A. Key Points
      1. While some periods of enlightenment regarding the relationship between the nervous system and behavior emerged among the Egyptians and Greeks, the major advancements in biopsychology such as the understanding of electrical activity and functional neuroanatomy have been relatively modern and recent being established within the last 200 years.
      2. We take for granted that the brain and nervous system are the sources of intellect, reason, sensation, and movement. This disarmingly simple fact has not been universally accepted throughout human history.
   B. Trepanation, the drilling of holes in the skull, may represent a prehistoric understanding of the brain's role in behavior. (p. 3)
   C. Egyptians discarded the brain during mummification, yet provided modern-sounding descriptions of structure and the effects of brain injury including the irreversible nature of brain trauma. (pp. 3-4)
   D. Greeks understood that the brain was the organ of sensation. (p. 4)
      1. Hippocrates viewed the brain as the source of intellect, but this role was given to the heart by Aristotle and to the ventricles by Herophilus.
      2. Galen made many accurate anatomical observations, but continued the misunderstanding of the role of the ventricles and the central nervous system as a fluid-filled network of interconnected tubes. (p. 4)
   E. René Descartes (1596-1650) (pp. 4-5)
      1. Continued the notion that fluids produced movement.
      2. Proposed **"mind-body" dualism**, which maintains that the body is mechanical and the mind is neither physical nor suited to scientific observation as opposed to the modern monism philosophical perspective that the mind is a product of physical neural activity.
   F. Discovery of the light microscope by Anton van Leeuwenhoek in 1674. (p. 5)
   G. In the late 1700's, Luigi Galvani and Emil du Bois-Reymond established electricity as the mode of communication used by the nervous system. (p. 5)
      Figure 1.3 is an interesting depiction of Galvani's basement laboratory where he connected wires from a rooftop antenna to the legs of frogs to demonstrate that electrical disturbances could stimulate the leg muscles of the frogs.

H. Charles Bell (1774–1842) and François Magendie (1783–1855) demonstrated that sensory and motor information travels in separate pathways. (p. 5)

I. Camillo Golgi, an Italian neuroanatomist, developed new staining techniques allowing the visualization of the structure of single neurons. Santiago Ramón y Cajal, a Spanish neuroanatomist, made classic trace drawings of neural circuitry leading to the proposal of the Neuron Doctrine, which stated that the nervous system is composed of separate nerve cells rather than the interconnect network of continuous fibers proposed by Golgi. In 1906, Golgi and Cajal shared the Nobel Prize for their neuroanatomical work and theories. (pp. 5-6)

J. **Phrenology**, the correlation of bumps on the skull with personal traits and intellectual abilities, was misguided in most respects, but modern in its acceptance that different cognitive functions may be localized to specific areas in the brain. (p. 6)

K. Work by Paul Broca, who examined the postmortem brains of patients with language deficits, and Fritsch and Hitzig, who observed contralateral muscle movements when stimulating the motor cortex of a rabbit and dog, further established the concept of localization of function in the brain. (p. 6)

L. Hughlings-Jackson proposed that the nervous system acted as a hierarchy, with simpler processing carried out by lower levels and sophisticated processing carried out by the cortex. (p. 6)

M. Charles Sherrington coined the word "synapse," and conducted research on reflexes and the motor systems. Otto Loewi demonstrated chemical signaling at the synapse. Sir John Eccles, Bernard Katz, Andrew Huxley, and Alan Hodgkin furthered our understanding of electrical signaling. (p. 6)

III. Research Methods in Biopsychology (pp. 8-20)

A. **Histology** refers to the study of microscopic structures and tissues. (pp. 8-9)

1. Tissue to be viewed must be fixed by freezing or formalin and sliced thinly by a **microtome**.
2. Stains are applied to highlight structures of interest, such as the structural analysis of single cells (**Golgi silver stain**), clusters of cell bodies within a central nervous system nucleus (**Nissl stain**), axonal pathways (**myelin stain** or **horseradish peroxidase**), or specific proteins found in a particular cells (antibodies).

B. **Autopsy** (to view for oneself) is the examination of a body after death. (p. 9)

C. Imaging (pp. 9-11)

1. **Computerized Tomography (CT)** uses x-ray technology to provide structural information about the brain and ventricles.
   Disadvantages of CT technology include the inability to discriminate between active and inactive regions, and repeated exposures to x-rays.
2. **Positron-Emission Tomography (PET)** provides information about brain activity based on the utilization of radioactive glucose or oxygen, the two primary nutrients needed to produce neural signals.
   Disadvantages of PET technology include a high expense of the machine and the injection of radioactive substances into the participant.
3. **Magnetic Resonance Imaging (MRI)** uses magnetism and radio frequency waves to provide high resolution images of structure.
   Although the use of high-power magnetic fields is generally considered safe, as larger magnets are employed questions arise about their effect on the physiology

of the body. MRI technology provides significantly higher resolution images than CT; however, MRI technology is much more expensive than CT technology.

4. **Functional Magnetic Resonance Imaging (fMRI)** uses a series of images taken in a short period of time to analyze brain activity by measuring the flow of blood and oxygen use in the central nervous system.

D. Recording (pp. 12-15)
Noninvasive recording techniques, such as EEG, evoked potentials, and MEG, are often used on humans; whereas, invasive recording techniques such as single-cell recordings are typically reserved for use in animal research models.

1. **Electroencephalography (EEG)**, useful in examining states of consciousness, such as sleep and epilepsy, is recorded through scalp electrodes providing information about the relative activity of large groups of neurons close to the surface of the brain.
   Recent advances using computerized EEG recordings are allowing the diagnosis of many disorders, such as schizophrenia, dementias, epilepsy, and attention deficit disorder.
2. **Evoked potentials**, a specialized use of EEG technology, represent the brain's response to environmental stimuli.
3. **Magnetoencephalography (MEG)**, in which the brain's tiny magnetic output is assessed, provides information about the activity of particular areas of the brain. Often the activity measured through MEG is superimposed on a MRI structural scan of the brain.
4. Single-cell recordings, using surgically implanted microelectrodes, allow researchers to observe the responses of individual neurons and apply electrical stimulation.

E. Stimulation of the brain, used to assess the functions of particular areas, may be accomplished by using surface electrodes during neurosurgery, surgically implanted electrodes, or magnetism (**repeated transcranial magnetic stimulation** or rTMS). (pp. 15-16)

F. **Lesions**, which are also used to assess function, may result from naturally occurring or deliberate brain injury.
Electrical or heat lesions destroy small areas of neurons and pathways, whereas chemical lesions destroy small areas of either neural cell bodies or axons allowing researchers to selectively examine the function of nuclei or pathways in a particular area. The term, **ablation**, is used instead of lesion when sections of the central nervous system are surgically removed. (pp. 16-17)

G. Biochemical Methods (pp. 17-18)

1. Drugs of interest may be administered in a variety of ways, including injection, eating, inhaling, chewing, or direct administration through micropipettes.
2. **Microdialysis**, in which chemical samples are removed through micropipettes, allow researchers to identify chemicals present in a very small area of interest.

H. Genetic Methods (pp. 18-19)
Why Does This Matter: Nature versus Nurture (p. 18)

1. The effect of the amount of genetic concordance on behavior is examined by comparing monozygotic and fraternal twins. Studies comparing twins, including those adopted by different sets of parents, provide some insight into relative contributions of genetics and environment.
2. Genetically modified (GM) animals have had a defective gene (**knockout gene**) inserted into their chromosomes, allowing researchers to correlate changes in behavior and physiology with the associated genes affected by the knockout technique.

I. Stem cells from embryos, adult tissues, and umbilical cord blood might provide the means to repair neural damage. (pp. 19-20)

IV. Research Ethics (pp. 21-25)

A. The ethical use of experimental methodology, including animal and human subjects, is subjected to multiple levels of review (university review boards, national organizational review boards, and peer review at the time of publication) to ensure compliance with federal, state, and local laws and ethical use guidelines. (pp. 21-22)

B. Human subjects guidelines provide for lack of coercion, informed consent, and privacy protection. (pp. 22-23)

Why Does This Matter: The Death of Jesse Gelsinger (p. 22)

C. Animal subjects guidelines focus on the necessity of using animals to answer a research question, the provision of excellent housing and health care, and the minimizing of the number of animals used and any pain and suffering that is necessitated by the research protocol. (p. 23)

D. Research on the Internet raises challenges in the areas of informed consent and privacy. (p. 24)

E. The use of stem cells has stimulated extensive ethical debate. (pp. 24-25)

CHAPTER 2

The Anatomy and Evolution of the Nervous System

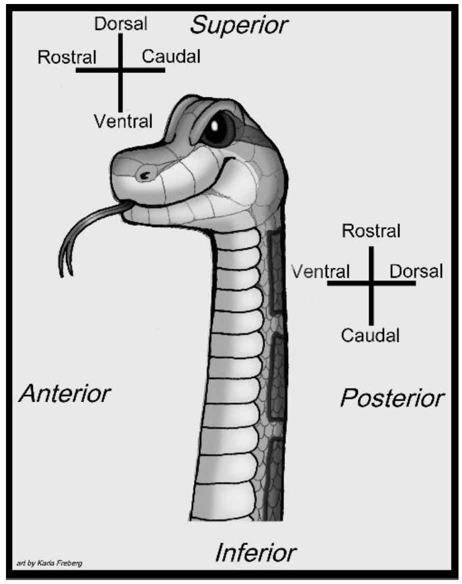
LECTURE OUTLINE

I. Anatomical Directions and Planes of Section (pp. 27-29)

A. Anatomical directions help us locate structures in the nervous system.

Common directional terms must be established before undertaking a description of the nervous system. The anatomical directional terms may become confusing due to a 90-degree bend in the **neuraxis** of humans. Comparing the use of the terms between a four-legged animal and a human is a very useful tool to minimize confusion. (pp. 27-28)

You can think of the anterior-posterior set naming the walls, floor, and ceiling of a room, whereas the rostral-caudal set applies to the animal in the room, as in this image



1. **Rostral** structures are located toward the head within the body region or the front of the skull within the head region.
2. **Caudal** structures are located toward the tail (feet in humans) within the body region or the rear of the skull within head region.
3. **Dorsal** structures are located toward the back within the body region or the top of the skull within the head region.
4. **Ventral** structures are located toward the belly within the body region or the bottom of the skull within the head region.
5. In humans, the dorsal parts of our brain form a 90-degree angle with the dorsal parts of the spinal cord.
6. **Anterior** structures are in front of the animal, which means the forehead and belly of a human. In other words, looking at the human spinal cord, anterior and ventral are equivalent.
7. **Posterior** structures are in back of the animal, which means the back of the head and the back of a human. In other words, looking at the human spinal cord, posterior and dorsal are equivalent.
8. **Superior** structures are located at the top of the animal, which means the top of the head in humans. Looking at the human spinal cord, the thoracic division is both superior and rostral to the lumbar division.
9. **Inferior** structures are located at the bottom of the animal. In the human spinal cord, the lumbar division is both inferior and posterior to the thoracic division.
10. **Ipsilateral** structures are on the same side of the midline, and **contralateral** structures are on opposite sides of the midline.
11. Structures near the **midline** are **medial**, and structures away from the midline are **lateral**.
12. In limbs, **proximal** structures are closer to the body center, and **distal** structures are farther away.

B. Anatomists make particular cuts or sections in the nervous system in order to view the structures in two rather than three dimensions. (pp. 28-29)

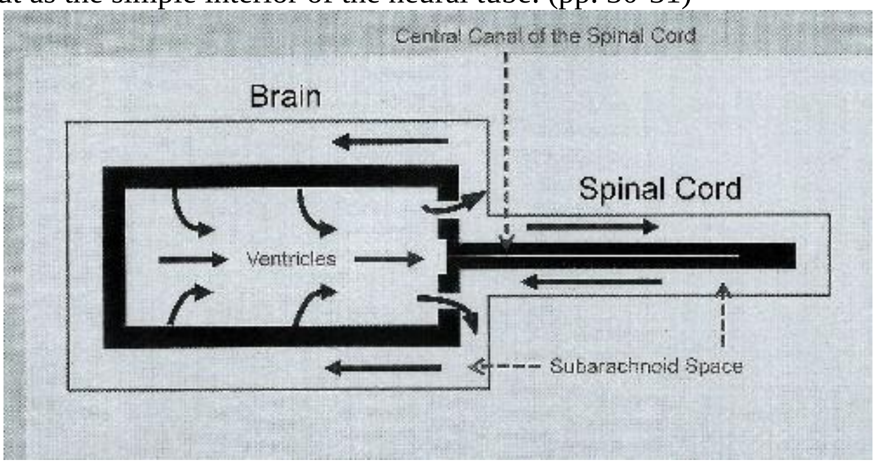
1. **Coronal** or frontal sections divide the brain from front to back in a vertically cut plane as if from ear to ear. (Mnemonic: Think of a woman wearing a tiara—coronal means crown)
2. **Sagittal** sections are parallel to the midline and give us a "side" view of the brain in a vertically cut plane as if from the front to back of the head. A special sagittal section cut direction on the midline of the brain is called a **midsagittal** section. This is a common view used to describe the corpus callosum, the **brainstem** and midbrain structures, and the ventricle system. (Mnemonic: Sagittarius is usually depicted as a man getting ready to shoot an arrow from a bow, seen from the side)
3. **Horizontal / axial** sections divide the brain from top to bottom in a plane that is parallel to the floor in a human standing upright.

II. Protecting and Supplying the Nervous System (pp. 29-32)

A. The **Meninges** (pp. 29-30)

1. Three layers of meninges protect the central nervous system: the **dura mater**, the **arachnoid**, and the **pia mater**.
2. Only the dura and pia mater layers are present in the peripheral nervous system.

B. The **Cerebrospinal Fluid** circulates through the four **ventricles**, the **central canal** of the spinal cord, and the **subarachnoid space**, floating and cushioning the central nervous system. Although the ventricular system curves and folds as the brain matures, it starts out as the simple interior of the neural tube. (pp. 30-31)



The Cerebrospinal Fluid (CSF) can be thought of as an ultra-filtered version of the plasma found in circulating blood. The CSF is generated by the **choroid plexus** primarily in the lateral ventricles and it flows in a pattern from the left and right lateral ventricles into the medial third ventricle through the narrow cerebral aqueduct of the midbrain into the fourth ventricle between the brainstem and the cerebellum and finally into the central canal of the spinal cord and the surrounding subarachnoid space where it is absorbed back into the blood supply. A primary function of the CSF is to protect the brain through floating the brain rather than attaching it to the skull.

Hydrocephalus is the condition resulting from a blockage of CSF flow through the central nervous system. The blockages usually occur at the narrow passages in the ventricle system such as the cerebral aqueduct. These blockages are commonly associated with development, tumor growth, or swelling of the brain due to trauma.

C. The Blood Supply: The brain is supplied with blood through the carotid and vertebral arteries. (pp. 31-32)

The neurons of the central nervous system use large amounts of energy and thus require a constant supply of oxygen and glucose among other nutrients. In fact, the average adult brain represents only 5% of the total body weight; however, the brain uses more than 20% of the body's total cardiac output. There is no storage of oxygen or glucose within the central nervous system and so an uninterrupted supply is critical. Significant neural death occurs within 3 minutes of the central nervous system not receiving any new blood supply. In the event of a cardiac arrest, effective and immediate CPR first-aid helps keep oxygenated blood flowing to the central nervous system to prevent brain damage.

The **carotid arteries** have very large diameters whereas the upstream cerebral arteries and capillaries become very small in diameter. Debris, such as blood clots or plaque deposits, which become dislodged and pass through the carotid artery but block the smaller cerebral arteries or capillaries are common causes of strokes (discussed in detail in Chapter 15). The effect of a stroke maybe localized to particular regions of the brain depending on whether the posterior, middle, or anterior cerebral arteries are affected.

III. The **Central Nervous System** (pp. 33-47)

A. The **Spinal Cord** (pp. 33-35)

1. The spinal cord may be divided into cervical, thoracic, lumbar, and sacral segments.
   The spinal cord segments are named according to vertebral bones surrounding the spinal cord. The incoming afferent sensory nerves and outgoing efferent motor nerves exit the **vertebral column** between each vertebral bone resulting in 31 discrete nerve segments. The area that is innervated by each of the 31 spinal nerves is called a dermatome. The motor cortex and somatosensory cortex respectively located in the frontal and parietal lobes are organized in a medial to lateral fashion by ascending dermatome from the toes to the head.
2. In addition to carrying messages to and from the brain, the spinal cord provides a variety of protective and motor **reflexes**.
   The **withdrawal reflex** is a commonly understood reflex that involves only three neurons. The afferent sensory neuron enters the dorsal spinal cord and forms a synapse in the **gray matter** of the dorsal horn with both an ascending sensory neuron that travels to the brain in the dorsal column **white matter** and an interneuron. In turn, the interneuron along with descending motor neurons traveling in the ventral white matter form a synapse with the outgoing ventral motor neuron to complete the reflex circuit. The level of incoming sensory signals determines activation of the interneuron with lower levels of input ascending to the somatosensory cortex without activating the reflex circuit.

B. The Hindbrain (pp. 35-37)

In terms of evolution, the development of brain regions follows the order of **hindbrain** then **midbrain** then **forebrain**, with the **cerebral hemispheres** being the most recent brain structure to develop. Similarly, functions associated with each brain region begin with the most basic life sustaining functions and progress to more complex functions through the ascending brain regions.

1. The hindbrain consists of the **medulla**, **pons**, and **cerebellum**. The medulla is also known as the **myelencephalon**, and together, the pons and cerebellum make up the **metencephalon.**
   In addition to containing **nuclei** representing the first central nervous system synaptic processing of incoming somatosensory, vestibular, auditory, and taste neural signals, the medulla and pons also contain several nuclei that control life sustaining functions such as heart rate, respiration, and the vomiting reflex.

   In addition to its known role in coordinating neural signals from the sensory and motor systems, the cerebellum also likely plays a role in cognitive functions such as attention, learning, and memory. The specific role of the cerebellum in advanced cognitive functioning has yet to be fully characterized.

2. Running through the medulla and pons at the midline is the **reticular formation**, which helps control arousal.
   Due to the associated life sustaining functions, damage to hindbrain and the reticular formation in particular is likely to cause coma or death. Trauma to the brain often produces a swelling response that can apply pressure on the hindbrain inducing a transient coma until the pressure is relieved.

C. The Midbrain: The midbrain, also known as the **mesencephalon**, contains the remaining section of the reticular formation, the **periaqueductal gray**, the **red nucleus**, the **superior colliculi**, the **inferior colliculi**, and the **substantia nigra**.
The periaqueductal gray is involved in gate control theory of natural pain management as part of the descending pathway responsible for the release of opioid peptides in response to incoming pain signals in the spinal cord. (pp. 37-38)

The red nucleus is involved in the motor output pathway and is the efferent nucleus receiving information from the lateral interpositus nucleus of the cerebellum to produce the conditioned eyelid response to an auditory tone in the example of classical conditioning learning.

The substantia nigra is a midbrain nucleus specifically targeted during the neural degeneration of Parkinson’s disease. The loss of the pathway from the substantia nigra to the **basal ganglia** produces the primary motor symptoms of Parkinson’s disease.

The superior and inferior colliculi are respectively involved in the ability to orientate the body toward visual and auditory stimuli. Animals that rely on visual and auditory tracking to detect prey have proportionally larger superior or inferior colliculi.

D. The Forebrain (pp. 38-46)

1. The **diencephalon** contains the **thalamus** and **hypothalamus**.
   A common misconception is that the thalamus serves a “relay” nucleus with little or no processing of incoming sensory information. The thalamus also participates in states of consciousness and arousal (see Chapter 11) and learning and memory (see Chapter 12).

The hypothalamus is best described as the central regulator of the internal physiological state of our body including the homeostatic functions of circadian rhythms, thermoregulation, reproduction, and ingestive behavior. The hypothalamus also controls the release of hormones from the **pituitary gland** and regulates the activation of the autonomic nervous system.

2. The telencephalon contains the cerebral cortex, basal ganglia, and **limbic system** structures.

   The basal ganglia consist of the anterior and medially located **caudate nucleus,** the **putamen** and **globus pallidus** located anterior and lateral to the thalamus, and the **subthalamic nucleus** located below the thalamus. The basal ganglia border the lateral ventricles and degeneration of the basal ganglia, such as that occurring in Huntington's disease, is often identified through brain scans revealing enlarged lateral ventricles. Pressure on the basal ganglion from CSF in the lateral ventricles produces the shuffling gait symptom of normal pressure hydrocephalus.

   The limbic system consists of medial subcortical structures collectively involved in memory or the interpretation and expression of emotion. Some limbic structures such as the **amygdala** and **septal area** appear to have specific emotional functions (fear, rage, attack, and aggression), while other areas such as the **cingulate cortex** have broader functional roles. The anterior cingulate cortex (ACC) also participates in decision-making, error-detection, anticipation of reward, and empathy. The posterior cingulate cortex (PCC) participates in eye movements, spatial orientation, and memory, and is one of the first structures affected by Alzheimer's disease. The **hippocampus**, **parahippocampal gyrus**, **mammillary bodies**, and **fornix** form tightly connects circuits involved in the formation of declarative memories (see Chapter 12). Emotion, the sense of smell, and the formation and recall of memories have strong associations between one another, thus the **olfactory bulbs** are often associated as limbic system structures.

3. The cerebral cortex is made up of six layers that cover the outer surface of the cerebral hemispheres.
   a) The "hills" of the cortex are referred to as **gyri (plural of gyrus)**, and the "valleys" are referred to as **sulci (plural of sulcus)** or **fissures**. The extent of the convolution of the cerebral cortex into gyri and sulci is directly related to the amount of cortical surface area contained within the skull. As the skull restricts the available volume for brain matter, the "wrinkling" of the cortex allows a greater surface area and thus more cortical neurons. Most cognitive functions are associated with the cerebral cortex and there is a positive correlation between the degree of cortical convolution and the cognitive abilities of various species.
   b) The cerebral cortex is divided into four lobes: the **frontal lobe** associated with motivation, personality, emotion, cognitive tasks such as executive function and judgment, and the motor system; the **parietal lobe** associated with somatosensation, association cortex, and advanced visual processing such as how to correctly respond to a visual stimulus; the **temporal lobe** associated with the auditory system, language comprehension, and

association cortex involved with memory storage; and the **occipital lobe** which is almost exclusively reserved for processing of visual stimuli.

c) The cerebral cortex also can be divided into **sensory** (somatosensation in the anterior parietal lobe, audition in the superior temporal lobe, vision in the occipital lobe, olfaction in the ventral frontal lobe, and gestation in the insular cortex at the junction of the temporal and parietal lobes), **motor** (posterior frontal lobe), or **association cortex** based on its function. Most of the sensory areas of the brain are located towards the back (caudal areas), whereas most of the motor areas of the brain are located rostrally.

d) The two cerebral hemispheres are connected by the **corpus callosum** and the **anterior commissure**.

e) Some functions, such as language, appear to be localized on one hemisphere or the other.
While there is good evidence of the lateralization of function for some specific tasks such as language comprehension and production respectively in Wernicke's area of the left temporal lobe and **Broca's area** of the left frontal lobe, the lateralization of other more intricate cognitive functions is much less clear (see Chapter 13). In general, the right hemisphere reacts more to negative emotional stimuli (avoidance), whereas the left hemisphere reacts more to positive stimuli (approach; see Chapter 14).

IV. The **Peripheral Nervous System** (pp. 47-52)

A. The **Cranial Nerves**: Twelve pairs of cranial nerves exit the brain and provide sensory and motor functions to the head and neck. (pp. 47-48)
A simple summary of the nerves is as follows: CNI, **olfactory nerve**, smells; CNII, **optic nerve**, sees; CNIII, **oculomotor nerve**, moves eyes, constricts pupils, accommodates; CNIV, **trochlear nerve**, moves eyes; CNV, **trigeminal nerve**, chews and provides facial sensations; CNVI, **abducens nerve**, moves eyes; CNVII, **facial nerve**, tastes and produces facial expressions; CNVIII, **auditory-vestibular nerve**, hears and provides balance / posture sensations; CNIX, **glossopharyngeal nerve**, tastes and provides sensory / motor functions of throat; CNX, **vagus nerve**, provides input to and sensations from the chest (heart, liver, digestive tract); CNXI, **spinal accessory nerve**, moves the head, neck, and shoulders; CNXII, **hypoglossal**, moves the tongue. Students might benefit from mnemonics for remembering these in order: <u>O</u>n <u>O</u>ld <u>O</u>lympus <u>T</u>owering <u>T</u>ops, a <u>F</u>inn and <u>G</u>erman <u>V</u>iewed <u>S</u>ome <u>H</u>ops.

B. The Spinal Nerves (pp. 48-49)

1. Spinal nerves contain dorsal **afferent nerve** roots and ventral **efferent nerve** roots of the **somatic nervous system** that merge outside the cord to form **mixed nerves**. A common pitfall is confusing the afferent and efferent terms. An easy association is that "a" comes before "e" in the alphabet and often we think of incoming "afferent" sensory information arriving before an outgoing "efferent" motor response to that sensation. Another mnemonic is to think of "a" for "access" and "e" for "exit."
2. Efferent nerves are myelinated, whereas afferent nerves may or may not be myelinated (see Chapters 7 and 8).

C. The **Autonomic Nervous System** provides sensory and motor innervation to glands, organs, and smooth muscle. (pp. 49-52)
Two subcomponents of the autonomic nervous system, the sympathetic and parasympathetic nervous system, act in opposition to one another with three primary differentiations: action on target organs, anatomical pathway, and type of neurotransmitter released.

1. The **sympathetic nervous system** operates during times of arousal and prepares the body for fight-or-flight reactions. The sympathetic nervous system is connected at the level of the ganglion producing a system known as the **sympathetic chain**. This connectivity results in the sympathetic nervous system being activated simultaneously as a whole unit allowing fast and complete reactions to an emergency. Norepinephrine is the primary neurotransmitter released by the sympathetic nervous system to its target organs.
2. The **parasympathetic nervous system** operates during times of rest and restoration. Unlike the sympathetic nervous system, the parasympathetic neurons are not connected with individual neural pathways to each target organ. This allows for differential activation of specific targets in response to particular needs, such as activation of the digestive system in response to the ingestion of food. Acetylcholine is the primary neurotransmitter released by the parasympathetic nervous system to its target organs.
3. The hypothalamus controls the autonomic nervous system by way of connections in the midbrain tegmentum.

V. Evolution of the Human Brain and Nervous System (pp. 53-57)
Why Does This Matter: Is Evolution Still Shaping Human Beings? (p. 53)

A. Natural Selection and Evolution (pp. 53-54)

1. Natural selection is the process by which favorable traits become more common in subsequent generations due to organisms' different reproductive success.
2. Fitness is the likelihood that an organism will reproduce successfully compared to other members of the same species.

B. Evolution of the Nervous System (pp. 54-55)
Why Does This Matter: Do Animals Have Minds? (p. 55)

1. The nervous system has been a relatively recent development in the course of evolution.
2. Single-cell organisms originated 3.5 billion years ago. Organisms developed neural nets approximately 700 million years ago, followed by organisms with ganglia located in a head region approximately 250 million years ago. Human brains appeared approximately 7 million years ago.
3. **Chordates** are the only animals possessing a true spinal cord and brain.
4. As the brain evolved, it became larger. In particular, the encephalon or cerebral cortex expanded and due to the capacity limit of the skull, the cerebral cortex became more convoluted.

C. Evolution of the Human Brain (pp. 55-57)
New Directions: Neuroscientists Search for Self-Awareness in the Brain (p. 58)

1. Human beings have experienced very rapid brain growth over the last five million years, possibly in response to the challenges of using tools, language, social behavior, and learning to plan for the future.
2. It is unclear is why such major cultural changes over the last 200,000 years, such as agriculture, urbanization, and literacy, have not produced additional changes in brain size.
3. One explanation of the late of recent changes in brain size is that a ceiling effect may have been reached where the advantages of further increases in brain size may be offset by childbirth difficulties and the large amount of resources required by the nervous system.

Assessment 1 (Due Session 2, 6:00 p.m.)

1. (20 points) Describe, compare, and contrast the following research methods: imaging; recording; stimulation; lesion.
2. (20 points) What are the relative strengths and weaknesses of the major imaging methods?
3. (20 points) What are the major considerations for the protection of human research participants?
4. (20 points) What are the major structures and functions of the hindbrain, midbrain, and forebrain?
5. (20 points) Neatly sketch (freehand, not traced) a midsagittal section of the brain, using different colors for different major structures. Clearly label the following: thalamus, hypothalamus, cerebellum, pons, medulla, reticular formation, corpus callosum, locus coeruleus, midbrain, lateral ventricle, frontal lobe, parietal lobe, occipital lobe. (Drawings will not be evaluated based on artistic ability, but attention to detail is expected.)

Session TWO
Chapters 3 and 4

Chapter 3: Cells of the Nervous System
Chapter 4: Psychopharmacology

ACTIVITIES
Quiz: Chapters 3 and 4
Lecture: Chapters 3 and 4
Break
Lecture: Chapters 3 and 4
Video

Secret Life of the Brain: The Child's Brain: Syllable From Sound
The explosion of language in young children provides one of the most dramatic illustrations of the young brain at work. How do we learn to talk? How do we learn to read? Unlike adults, in whose brains most linguistic activity is restricted to the left hemisphere, very young children respond to language with the entire brain. But what happens when the brain is physically compromised? And what are the physical roots of language disorders such as dyslexia?

Video Reaction (in-class)

CHAPTER 3
Cells of the Nervous System

LECTURE OUTLINE

I. Neurons and Glia

Neurons are the primary functioning cells of the central nervous system. **Glia** (Greek for glue) are the primary support cells of the central nervous system. (pp. 61-72)

A. The Structure of Neurons (pp. 61-67)

1. Most neurons have four distinct structural components associated with specific functions: **dendrites** (input), **cell body** or **soma** (processing), **axon** (signal transmission), and **axon terminal** (output).
2. The neural membrane is composed of a two-molecule-thick layer of phospholipids dividing the fluid inside the neuron (**intracellular**) from the fluid surrounding the neuron (**extracellular**).
3. Embedded within the phospholipid membrane are ion channels and pumps, which are specialized proteins that allow chemicals to pass into and out of the neuron. These channels and pumps allow selective **permeability**, meaning that the direction and type of chemicals allowed in or out of the neuron is regulated.
   a) There are two important categories of ion channels. A **ligand-gated channel** opens or closes its selective ion pore in response to a specific **neurotransmitter** binding to a receptor site. A **voltage-dependent channel** has a selective ion pore that opens in response to a specific intracellular voltage.
   b) Two important pumps are the **sodium-potassium pump** and the **calcium pump**. Both pumps are critical in maintaining the ionic concentration gradient necessary for neural signaling.
4. The neural **cytoskeleton** (**microtubules**, **neurofilaments**, **microfilaments**) provides structural support and the ability to transport needed substances within the neuron. **Tau** molecules holding the microtubules in place disconnect in Alzheimer's disease, forming neurofibrillary tangles and damaging the structural integrity of the neuron.
   Why Does This Matter: The Connection Between Neurofibrillary Tangles and Amyloid in Alzheimer's Disease (p. 64)
5. The cell body contains important organelles (**nucleus**, **nucleolus**, **ribosomes**, **endoplasmic reticulum**, **Golgi apparatus**, **and mitochrondria**) that participate in the basic metabolism and protein synthesis of the cell. In particular, the nucleus contains the DNA that directs the functioning of the cell and the mitochondria constructs adenosine triphosphate (ATP), the major energy source of neurons, from oxygen and pyruvic acid in sugar (glucose). In addition, the cell body is a site of synapses with other neurons.
6. The axon is responsible for transmission of the neural signal from the cell body to the axon terminal. The junction of the axon to the cell body is called the **axon hillock** and it represents the beginning of voltage-dependent channels responsible for transmitting the electrical signal down the axon. The axon is insulated by fatty

cells called **myelin**. The electrical signal is regenerated by the voltage-dependent channels at spaces between the myelin called the **nodes of Ranvier**.

7. Neurons communicate with other cells at a junction known as the **synapse**. Typically, dendrites and cell bodies receive input and axons transmit output as either a chemical or electrical signal across the synapse. The connectivity between **dendritic spines** and axon terminals is affected by learning,or changes due to experience.

B. Structural Variations in Neurons: **Unipolar**, **bipolar**, and **multipolar** neurons are classified by the number of branches they possess and represent functional differences. (p. 68)

C. Functional Variations in Neurons: Neurons may be classified as **sensory neurons**, **motor neurons**, or **interneurons**. (p. 68)
Figure 3.8 on p. 69 depicts several structural and functional variations of neurons.

D. Glia are generally classified based on size. (pp. 69, 70-72)
   1. Macroglia include astrocytes, oligodendrocytes, and Schwann cells.
      a) Astrocytes, named for their star-like shape, provide structural support for neurons, contribute to the blood-brain barrier, isolate the **synaptic gap**, gather molecules from the synapse, and may participate in the transmission of information. In response to central nervous system damage, astrocytes move in to clean up dead neurons and in turn form scar tissue that inhibits central nervous system neural regrowth and connectivity.
      b) Oligodendrocytes myelinate the central nervous system through branching processes that wrap around several neurons forming a connective network not conducive to regeneration following neural damage.
      c) Schwann cells myelinate the peripheral nervous system with each glial cell insulating a segment of one axon.
   2. **Microglia** remove debris resulting from damage to neurons.

II. The Generation of the Action Potential (pp. 73-79)

A. The differential ionic composition of the intracellular and extracellular fluids is essential in the process of neural signaling. (pp. 73-74)
   1. In a neuron at rest, the intracellular fluid contains large amounts of potassium and smaller amounts of sodium and chloride.
   2. The extracellular fluid contains large amounts of sodium and chloride and smaller amounts of potassium.

B. Two factors provide the force to move molecules during neural signaling. (pp. 74-76)
   1. In **diffusion**, molecules move from areas of high concentration to areas of low concentration in order to establish equilibrium.
   2. **Electrical force** moves like-signed ions away from each other and opposite-signed ions towards each other.

C. The **resting potential** of neurons averages about -70mV. **Depolarization** occurs when events, such as ion movement between the extracellular and intracellular fluid, results in a decrease in the membrane potential towards 0mV. (p. 76)
Why Does This Matter: Lethal Injection (p. 76)

D. The action potential (pp. 77-79)
   1. When a cell body is depolarized to **threshold**, an action potential is produced in the axon.

2. Action potentials are "all-or-none" meaning that an action potential is automatically produced once threshold has been reached but no action potential can be produced if threshold is not achieved.
3. As shown in Figure 3.15, the initial opening of voltage-dependent sodium channels allows sodium ions to enter the neuron accounting for the rise of the action potential. After a brief delay voltage-dependent potassium channels open allowing potassium ions to exit the neuron accounting for the fall seen in a recording of an action potential.
4. The **absolute refractory period** is a short amount of time following activation of sodium channels during an action potential in which it is impossible to send another action potential. The **relative refractory period** occurs during the period of **hyperpolarization** following an action potential. Action potentials are possible during the relative refractory period but a stronger input is required to reach threshold.

III. The **propagation** of the action potential refers to the reproduction of the signal down the axon. (pp. 79-81)
  A. Action potentials move down the length of unmyelinated axons by the relatively slow process of **passive conduction** as shown in Figure 3.17.
  B. Since myelinated axons are insulated, the action potential only needs to be reproduced at the nodes of Ranvier. This process is called **saltatory conduction** and is very rapid as shown in Figure 3.17.

IV. The Synapse (pp. 82-91)
The human brain contains about 100 billion neurons with each neuron forming an average of 1,000 synapses. The result is a prediction of more synapses in one human brain than the stars in the galaxy.

  A. **Electrical synapses** are characterized by very tiny synaptic gaps (3.5 nm) between the pre- and postsynaptic neurons that are essentially connected by special ion channels. Electrical synapses allow very fast, excitatory signals and while rarely found in the nervous system, electrical synapses are commonly found in the heart and liver. (pp. 83-84)
  B. **Chemical synapses** (20 nm) involve the release of neurotransmitter chemicals as the signal between pre- and postsynaptic cells. The use of chemicals as the signal between neurons permits selective activation and a wide variety of responses depending on the specific location and action of receptors for the different chemicals. (pp. 84-91)
    1. Neurotransmitters are released through the process of exocytosis.
      a) When an action potential reaches the axon terminal, voltage-dependent calcium channels open allowing a large influx of calcium into the axon terminal.
      b) The entry of calcium stimulates the movement of vesicles to the terminal membrane, where they fuse with the membrane and release neurotransmitters.
      c) Often vesicles do not release all of the contained neurotransmitter. After fusing with the membrane, vesicles are pinched off, refilled, and recycled.
      d) Most receptor proteins are found on the postsynaptic cells; however, autoreceptors are found on the presynaptic cell and provide feedback to the

axon terminal regarding the amount of neurotransmitter present in the synapse.

2. Receptor proteins on the postsynaptic cell can be either **ionotropic** or **metabotropic**.
    a) Ionotropic proteins open selective ion pores when activated by neurotransmitters.
    b) When activated by a neurotransmitter, metabotropic proteins release an intracellular **second messenger** signal called a **G protein**. The G protein in turn may produce a variety of effects including opening or closing of ion pores or even influencing the expression of proteins by the cell.
3. As summarized in Figure 3.21, the action of neurotransmitter molecules in the synaptic gap may be terminated through diffusion away from the gap, by deactivation of the neurotransmitter by enzymes, or by reuptake for reuse by the presynaptic neuron.
4. Postsynaptic potentials are small, local, **graded potentials** that can last 5-10 msec.
    a) **Excitatory postsynaptic potentials (EPSPs)** produce slight depolarizations by opening channels that allow sodium to enter the cell.
    b) **Inhibitory postsynaptic potentials (IPSPs)** produce slight hyperpolarizations by opening channels that allow either chloride to enter or potassium to exit the cell.
5. **Neural integration** of incoming EPSPs and IPSPs determines whether or not the postsynaptic neuron will generate its own action potential.
    a) In **spatial summation**, the input from many synapses is added together to determine whether or not an action potential will be produced.
    b) In **temporal summation**, a single, very active synapse may provide sufficient input within a short period of time producing an action potential.

C. Neuromodulation: One neuron may influence the ability of another neuron to send a signal by forming a synapse directly with the axon of the other neuron (**axo-axonic synapse**). The axo-axonic synapse may either facilitate or inhibit the release of neurotransmitter from the affected axon terminal. (p. 91)

V. New Directions: Tetanus and the Lack of Inhibition (p. 90)

A. Tetanospasmin hitches a ride to the central nervous system from a wound site using the retrograde transport system within axons.

B. The toxin binds to receptor sites for gamma-aminobutyric acid (GABA), the most common inhibitory neurotransmitter in the central nervous system, and can’t be dislodged.

C. Without normal inhibitory input from GABA, the motor system becomes over-excited causing muscles to go into sudden, involuntary contractions, or spasms leading to respiratory or cardiac arrest.

CHAPTER 4
Psychopharmacology

LECTURE OUTLINE

VI. Neurotransmitters, Neuromodulators, and Neurohormones (pp. 95-103)

A. **Neurotransmitters** affect adjacent cells across the synapse. **Neuromodulators** diffuse away from the synapse to target cells some distance away. **Neurohormones** reach even more distant target cells by circulating through the blood supply. (p. 95)

B. Any chemical released by one cell at a synapse that produces a response in a target cell may be referred to as a neurotransmitter. In addition, most neurotransmitters are manufactured within neurons, released in response to the arrival of an action potential, have an observable effect on the postsynaptic cell, and are deactivated following release by reuptake or by the action of enzymes. (p. 95)

C. Types of Neurotransmitters (pp. 96-103)

1. There are two major classes of neurotransmitters. **Small-molecule neurotransmitters** include acetylcholine, dopamine, norepinephrine, epinephrine, serotonin, melatonin, glutamate, GABA, and ATP. **Neuropeptides** acting as neurotransmitters include substance P and endogenous endorphins, among about fifty others. The small-molecule neurotransmitters can be synthesized anywhere in the neuron including the axon terminal; whereas, neuropeptides are synthesized in the cell body and transported to the axon terminal for release.

2. Cholinergic neurons that release **acetylcholine (ACh)** are found at the neuromuscular junction, in the autonomic nervous system, and within the central nervous system.

   a) The enzyme choline acetyltransferase (ChAT) combines choline, from dietary fats, and acetyl coenzyme A to from ACh. **Acetylcholinesterase (AChE)** is the enzyme that breaks down ACh in the synapse.

   b) Two major subtypes of ACh receptors are the nicotinic receptors and the muscarinic receptors. **Nicotinic receptors** are fast ionotropic receptors activated by both ACh and nicotine. Nicotinic receptors are used at the neuromuscular junction and also in the central nervous system. **Muscarinic receptors**, primarily found in the central nervous system, are metabotropic receptors activated by both ACh and muscarine.

   Why Does This Matter: Genes and Addiction to Nicotine (p. 98)

3. The small molecule class of neurotransmitters called the monoamines is divided into two subgroups, the **catecholamines, dopamine (DA), norepinephrine (NE),** and **epinephrine (E)**, and the **indoleamines**, **serotonin (5-HT)** and melatonin.

4. The catecholamines are synthesized from a single precursor, tyrosine. There is a progression of enzymatic conversions that produce **l-dopa**, then dopamine, next norepinephrine, and finally epinephrine.

5. Dopamine is particularly involved with systems controlling movement, reinforcement, and planning.

6. Norepinephrine pathways increase arousal and vigilance both in the central nervous system and as the primary neurotransmitter of the sympathetic nervous system.
7. The primary indoleamine, serotonin, is synthesized from the precursor tryptophan commonly found in grains, meats, and diary products. Serotonin acts as a neurotransmitter in the regulation of mood, sleep, and appetite.
8. **Glutamate** is the most frequently used excitatory neurotransmitter in the central nervous system. Multiple subtypes of glutamate receptors are organized into three categories: kainate, NMDA, and AMPA. The NMDA and AMPA receptors are thought to underlie some learning functions and work together to trigger long lasting changes in neurons.
9. **Gamma-aminobutyric acid (GABA)** is the major inhibitory neurotransmitters in the central nervous system.
10. ATP and its byproducts, particularly adenosine, act as neurotransmitters in both the peripheral and central nervous systems.
11. At least 40 different peptides serve as neurotransmitters, neuromodulators, and neurohormones, including substance P, endorphins, insulin, cholescystokinin (CCK), oxytocin, and vasopressin (ADH).
12. Gaseous neurotransmitters may diffuse through membranes and interact with internal receptors to transmit information. **Nitric oxide (NO)** is involved in neural communication, maintenance of blood pressure, and is the target of the drug Viagra to produce penile erection. Gases may also be used to communicate from postsynaptic neurons to presynaptic neurons.

VII. Drug Actions at the Synapse (pp. 105-108)

A. Agonists and Antagonists (Note: Pharmacologists usually apply these terms more strictly to describe drug actions at a receptor only, but most behavioral neuroscientists use the terms more broadly to describe drug actions elsewhere at a synapse). (p. 105)
   1. **Agonists** boost the activity of a neurotransmitter. An agonist may act on a neurotransmitter in a variety of ways including mimicking the neurotransmitter, blocking reuptake, or increasing synaptic release.
   2. **Antagonists** have a variety of methods to interfere with the action of a neurotransmitter, including reducing synaptic release, blocking the receptor site, and increasing the degradation of the neurotransmitter.

B. Drugs affect behavior by interacting with a variety of processes occurring at the synapse. (105-108)
   1. Manipulating neurotransmitter production affects the amount of neurotransmitter available for release.
      a) Agonistic example: Consuming milk before bedtime provides extra tyrosine that may boost production of serotonin that is involved in sleep control.
      b) Antagonistic example: The drug AMPT interferes with catecholamine synthesis reducing the levels of dopamine, norepinephrine, and epinephrine.
   2. Some antagonists, such as **reserpine**, interfere with the storage of neurotransmitters, in this case monoamines, in synaptic vesicles.
   3. Drugs may produce agonist and antagonist effects on neurotransmitter release.
      a) Black widow spider venom acts as an agonist to enhance ACh release at the neuromuscular junction resulting in convulsions. Paralysis follows when the

overstimulation of ACh depletes the supply for the neuromuscular junction synapses.

b) Botulin toxin acts as an antagonist interfering with ACh release producing the disease, **botulism**, leading to paralysis. A deactivated version of the toxin, Botox, is used to paralyze muscles in order to reduce facial wrinkles.

4. Receptor effects account for the majority of drug interactions.
   a) Nicotine mimics the action of ACh at nicotinic receptors.
   b) A toxin derived from plants in the Amazon, c**urare**, blocks nicotinic receptors and was traditionally used as a paralytic agent on darts and arrows in hunting.
   c) Alcohol, **benzodiazepines**, and barbiturates act as sedatives by potentiating the effects of GABA at the $GABA_A$ receptor.
5. Reuptake effects and enzyme degradation constitute the second largest effect of drugs on neurotransmitters.
   a) Cocaine, amphetamine, and methylphenidate (Ritalin) inhibit reuptake of dopamine acting as an agonist in the central nervous system reward and reinforcement areas.
   b) **Reuptake inhibitors** act as agonists by interfering with presynaptic absorption of neurotransmitters. The selective serotonin reuptake inhibitor (SSRI) fluoxetine (Prozac) inhibits serotonin reuptake increasing the presence of serotonin in people diagnosed with major depressive disorder.
   c) Organophosphates, developed as chemical weapons and still used today in pesticides, interfere with AChE, the enzyme responsible for breaking down ACh at the neuromuscular junction synapses. Although this will initially boost the action of ACh, continued stimulation of receptors can harm them.

VIII. Basic Principles of Drug Effects (pp. 108-113)
Why Does This Matter: The Hazards of Designer Drugs (p. 111)

A. Administration of Drugs: The mode of administration of a drug influences the concentration of the drug in the blood within a given time, which in turn influences the overall behavioral effects of the drug. Three protective mechanisms, the blood-brain barrier, enzymes in the liver, and the **area postrema**, developed to counter the ingestion of toxins may act to reduce the effects of administered drugs. (pp. 108-109)

B. Individual Differences in Response: Gender, size, and genetics influence individual differences in responses to drugs. (p. 109)

C. **Placebo effects** occur when inactive substances appear to produce behavioral and cognitive effects due to a user’s expectations. **Double-blind experiments** attempt to control placebo effects with neither the subject nor the experimenter knowing whether the administered chemical is the control (placebo) or actual drug. (p. 109)

D. Tolerance and Withdrawal (pp. 110-111)
   1. **Tolerance** (the need to administer more of a drug to produce the same effects) may occur due to changes in enzymes, changes at the level of the synapse, and even changes due to unconscious learning such as classical conditioning.
   2. **Withdrawal** occurs when some drugs are discontinued, leading to behavioral effects that are typically the opposite of the effects produced by the drug.

E. **Addiction** is characterized by a compulsive need to readminister a drug. (pp. 111-113)

New Directions: Dopamine Agonists, Parkinson's Disease, and Addictive Behaviors (p. 112)

1. The dopamine reward system, including the **nucleus accumbens**, evolved as a natural reinforcement mechanism activated in response to pleasant experience including eating, drinking, and sexual behavior. Many drugs have an effect of the nucleus accumbens implicating it in addiction.
2. Addiction is the result of complex physical and environmental variables, making it extremely challenging to treat. Research continues to explore physiological mechanisms to combat addiction such as methadone, Antabuse, and vaccinations.

IX. Effects of Psychoactive Drugs (pp. 113-121)

A. Stimulant drugs increase alertness and mobility. (p. 113-116)

1. **Caffeine**, the most commonly used stimulant, is an antagonist of adenosine, and inhibitory neurotransmitter in the central nervous system. Caffeine's behavioral effects due to blocking the inhibitory effects of adenosine include alertness, improvement of reaction time, and a reduction in headaches due to a reduction in blood flow to the brain. Caffeine is considered addictive producing both withdrawal and tolerance effects. The ability to metabolize caffeine develops at approximately eight months following birth. Therefore, it is prudent for pregnant and nursing mothers to avoid caffeine consumption.
2. The second most commonly used stimulant is **nicotine** which acts as an agonist of the nicotinic ACh receptors and dopamine receptors in the nucleus accumbens. Addiction to nicotine is attributed to its action on the nucleus accumbens and associated withdrawal symptoms.
3. **Cocaine** and **amphetamine** are among the most addictive drugs known. Cocaine acts as a dopamine reuptake inhibitor, while amphetamine acts to increase presynaptic dopamine and norepinephrine release.
4. Club Drugs: Ecstasy and GHB
   a) **Ecstasy (MDMA)** is structurally similar to methamphetamine and hallucinogens acting as an agonist of serotonin. Recent research in both animal and human subjects has revealed the propensity of MDMA to destroy serotonergic neurons. The behavioral consequences of this reduction in the serotonin supply in the central nervous system include sleep disturbances and depression.
   b) **Gamma-hydroxybutyrate (GHB)** is a sedative, hypnotic drug acting as a GABA agonist. Initially marketed as a diet aid and muscle building supplement, GHB has been used as a "date rape" drug and it has both serious withdrawal symptoms and dangerous interactions with alcohol.

B. Opiates produce profound pain relief, and in some forms, a remarkable sense of euphoria. (pp. 116-117)

1. **Morphine** and **codeine**, natural opiates derived from the opium poppy, and heroin, a synthetic opiate interact with receptors for endogenous **endorphins**.

C. Marijuana or cannabis contains over sixty different **psychoactive** compounds but **tetrahydrocannabinol (THC)** is primarily responsible for the behavioral effects, including mild euphoria, perceptual distortion, hallucination, and depression. (pp. 117-119)

Why Does This Matter: Medical Marijuana (p. 119)

1. Marijuana's active ingredients interact with existing cannabinoid receptors that are normally activated by endogenous cannabinoids such **anandamide**.

D. Other Hallucinogens (pp. 119-120)

1. Many diverse substances produce hallucinations, including the *Amanita muscaria* mushroom, **mescaline**, **phencyclidine (PCP)**, and **lysergic acid diethylamide (LSD)**.
2. The exact mechanisms for the production of hallucinations are not well understood, but appear to arise from a high level of brain excitation. The modes of action for hallucinogens vary from GABA agonism to glutamate antagonism to unknown effects on serotonin.

E. Alcohol (pp. 120-121)

1. Alcohol particularly seems to interact with the $GABA_A$ receptor, where it boosts the inhibition normally provided by GABA. Alcohol also stimulates dopaminergic pathways, which may lead to euphoria and addiction, and inhibits NMDA glutamate receptors, likely accounting for memory deficits during intoxication.
2. Alcohol produces rapid tolerance at both the GABA receptor level and in the production of liver enzymes responsible for alcohol metabolism.
3. Chronic alcohol use damages organs such as the liver and kidneys as well as damaging the frontal lobes and hippocampus of the brain. Moderate consumption of specific types of alcohol may have selective beneficial effects, such as the report of a daily glass of red wine preventing cardiovascular disease.

F. **St. John's Wort** (p. 121)

1. Americans are frequently turning to herbal remedies as opposed to prescription medicines, and many of these supplemental herbs are as potent or more so than the more regulated and researched prescription medicines.
2. Acting as a selective serotonin reuptake inhibitor, hypericin found in St. John’s Wort is somewhat effective in cases of mild depression, but interacts negatively with other medicines, including chemotherapy.

Assessment 2 (Due Session 3, 6:00 p.m.)

1. a. (10 points) Describe the basic structure of neurons.
   b. (10 points) Neatly sketch (freehand, not traced) a typical neuron in the CNS, using different colors for different major structures. Clearly label the following: soma, nucleus, dendrites, axon, axon terminals, oligodendrocytes, Nodes of Ranvier. (Drawings will not be evaluated based on artistic ability, but attention to detail is expected.)
2. (20 points) Describe the changes that take place within the neuron by which an action potential is produced.
3. (20 points) Describe the process by which a signal is transmitted (at a chemical synapse) from one neuron to another.
4. (20 points) Describe the major characteristics of the following small-molecule neurotransmitters: acetylcholine (ACh), dopamine, norepineprine, epinephrine, serotonin, melatonin, glutamate, GABA.
5. (20 points) Describe the major effects on neurotransmitters and the major psychological effects of the following drugs: cocaine, MDMA, marijuana, alcohol.

Session THREE
Chapters 6, 10

Chapter 6: Vision
Chapter 10: Sexual Behavior

ACTIVITIES
Quiz: Chapters 6 and 10
Lecture: Chapters 6 and 10
Break
Lecture: Chapters 6 and 10
Video

Secret Life of the Brain: The Adult Brain: To Think By Feeling
The brain is the seat of both intellect and emotion, and this episode chronicles the critical balance between these processes and explores what happens when the balance is lost. Scientists draw insight from the stories of a stroke victim and a sufferer of post-traumatic stress disorder, and break new ground in the struggle to understand and treat depression.

Video Reaction (in-class)

CHAPTER 6
Vision

LECTURE OUTLINE

X. Characteristics of Light (pp. 155-157)
   A. The Advantages of Light as a Sensory Stimulus: The visual spectrum is abundant, and travels quickly, thereby minimizing the delay in a visual event and sensory reception, and it travels in straight lines minimizing distortion. (p. 155)
   B. The Electromagnetic Spectrum (p. 156)
      1. Human vision responds to a very small portion of the **electromagnetic radiation** spectrum between 460 and 750 nanometers (nm) in wavelength.
      2. Electromagnetic radiation can be described in terms of waves, where wavelength is the distance between peaks and **amplitude** is the height of the wave.
      3. Wavelength is processed as color, and amplitude is processed as **brightness**.
      4. Electromagnetic radiation can also be described as the movement of large numbers of particles known as **photons**.
   C. **Absorption**, **Reflection**, and **Refraction**: Light waves are absorbed by some objects, reflected by others and reflected by others thus determining the colors that we perceive. Light waves may also be refracted or bent by molecules in air and water. (pp. 156-157)

XI. The Structure and Functions of the Visual System (pp. 158-175)
   A. Protecting the Eye: Eyes enjoy the protection of the bony **orbits** of the skull, cushioning fat deposits within the orbit, eyelids, and tears. (p. 158)
   Why Does This Matter: Why Do We Cry? (p. 158)
   B. The Anatomy of the Eye (pp. 159-161)
      1. The tough, outer, white covering of the eye is known as the **sclera**.
      2. Light first enters the eye through the transparent **cornea**, which performs the initial bending of the light towards the **retina**.
      3. Light then travels through the **aqueous humor** of the **anterior chamber** and passes through the opening of the **pupil** controlled by the **iris**. After the pupil light travels through the crystalline lens, which through **accommodation** aids in focusing the image as it continues through the **vitreous humor** of the **vitreous** chamber before reaching the retina.
      4. The retina is a thin layer containing visual interneurons and **photoreceptors**.
         a. Blood vessels and the optic nerves exit the eye through the **optic disk** producing a blind spot where there are no photoreceptors.
         b. The **macula** is an area of the retina that is not covered with blood vessels, and a pit within the macula, known as the fovea, is responsible for **central vision** as opposed to **peripheral vision**.
         c. There is a high density of **cone** photoreceptors in the fovea and little if any **rod** photoreceptors which are relegated to the peripheral retina. Acuity is best when an image falls on the fovea and macula; however, sensitivity to dim lights is best in the periphery.
   C. The Layered Organization of the Retina is illustrated in Figure 6.9. (pp. 161-162)
      1. As we look towards the back of the eye, the first layer of the retina is the **ganglion layer**, whose cells give rise to the axons forming the optic nerve.

2. The ganglion cells form connections with amacrine and bipolar cells in the **inner plexiform layer**.
3. The cell bodies of the **amacrine**, **horizontal**, and **bipolar cells** are located in the **inner nuclear layer**.
4. The bipolar cells form connections with horizontal cells and photoreceptors in the **outer plexiform layer**.
5. The cell bodies of the photoreceptors are located in the **outer nuclear layer**.

D. Photoreceptors (pp. 162-164)
   1. Photoreceptors are named rods and cones on the basis of the shape of their **outer segments**, which contain **photopigments**, the chemicals that interact with light to release an intracellular signal.
   2. The 120 million rods in the human eye are responsible for **scotopic vision**, which occurs in dim light, while the 6 million cones are responsible for **photopic vision**, which occurs in bright light.
   3. In general, **transduction** is the process of converting a physical stimulus into a biological signal.
      a. Transduction of light to neural signals begins when a molecule of photopigment absorbs a photon.
      b. Absorption of light causes the photopigment to break apart.
      c. This event triggers the release of enzymes that break down **cyclic guanosine monophosphate (cGMP)**, which in turn causes sodium channels in the outer segment to close.
   4. Photoreceptors have a unique method of signaling where a **dark current** depolarizes the photoreceptors in the absence of light stimulating glutamate neurotransmitter release. Photoreceptors hyperpolarize in response to light activation releasing less neurotransmitter. Photoreceptors also produce graded potentials meaning the amount of neurotransmitter release varies depending on the intensity or brightness of the light stimulus.

E. Besides differing in structural shape, rods and cones also contain different photopigments that respond differentially to both wavelength and intensity. (pp. 164-165)
   1. Rods contain **rhodopsin** that is very sensitive to low levels of light in the blue to green range of wavelengths.
   2. Cone photopigments require a greater amount of light to be absorbed in order to respond. Furthermore, there are three different cone photopigments with each responding to a narrow range of violet (cyanolabe), green (chlorolabe), or red (erythrolabe) wavelengths.

E. Processing by Retinal Interneurons (pp. 165-168)
   1. Each retinal interneuron has a **receptive field** defined as the area of the retina that when stimulated will produce a response, either excitatory or inhibitory, in a particular interneuron.
   2. Horizontal and amacrine cells in the retina integrate information from across small surface areas of the retina.
   3. Bipolar and ganglion cells process information about borders between areas of light and dark due to the organization of their receptive fields. The bipolar and ganglion receptive fields show antagonistic center-surround organization (center

on, surround off or vice versa), leading to their ability to respond to edges and borders of light.

4. The interneurons of the retina process visual stimuli in a manner that sharpens or exaggerates changes in light and dark areas. This contrast enhancement is achieved through a process called **lateral inhibition** in which horizontal cells inhibit neighboring bipolar cells.
5. The human eye's 1 million ganglion cells integrate the input from nearly 130 million photoreceptors and send the information to the brain via action potentials in the optic nerves.
    a. There are three types of ganglion cells: **M cells**, **P cells**, and **K cells**.
    b. Table 6.2 provides a summary of the differences between these three types of ganglion cells with regard to size, speed, size of receptive field, response to contrast, response to movement, and response to color.

F. Optic Nerve Connections (pp. 169-171)

1. The axons of the ganglion cells exit the eyes through the optic disk forming the **optic nerves**. The **optic nerves** partially decussate (in humans) in the **optic chiasm** such that portions of the visual field received by both retinas are consolidated in one location in the central nervous system. The partial crossing over of the optic nerves results in the left visual field being consolidated in the right central nervous system, while the right visual field is consolidated in the left central nervous system.
2. The **optic tracts** proceed to the **lateral geniculate nucleus (LGN)** of the thalamus, with smaller branches connecting with the hypothalamus supplying light information to regulate circadian rhythms and **superior colliculi** to guide the movement of eyes and the head toward newly detected objects.
3. The LGN has two **magnocellular layers**, which receive input from M cells, four **parvocellular layers**, which receive input from P cells, and **koniocellular layers**, which receive input from non-P-or-M cells.
4. The LGN also receives substantial input from the primary visual cortex and the reticular formation.

G. The Striate Cortex (pp. 171-173)

1. The **primary visual cortex**, also known as the **striate cortex** or V1, is located at the caudal pole of the occipital lobe and contains approximately 250 million neurons in each hemisphere.
2. V1 contains **simple cortical cells** and **complex cells** that participate in the encoding of primary features such as shape and movement.
3. Sections of cortex show a column organization, including **ocular dominance columns** responding to the input of one eye, and **orientation columns** responding to lines of a particular angle. A group of orientation columns that respond to all possible angles in a given receptive field location is called a **hypercolumn**.
4. **Cytochrome oxidase** blobs run parallel to the ocular dominance columns and participate in color vision.
5. Cortical modules contain two sets of ocular dominance columns, 16 blobs, and two hypercolumns, and form the basic unit of the primary visual cortex.

H. Visual Analysis Beyond the Striate Cortex (pp. 173-175)

1. Information regarding movement and how to appropriately interact with stimuli is processed by the **dorsal stream**, in which activity flows from the primary visual cortex to areas **MT** and **MST** in the temporal lobe.
2. Information regarding object recognition and categorization is processed by the **ventral stream**, in which activity flows from primary visual cortex to areas **V4**, **IT**, and the **fusiform face area (FFA)** of the inferior temporal lobe.

XII. Visual Perception (pp. 176-182)

There is some evidence that the cortex constructs a visual reality through the use of hierarchical processing, in which simple cells provide input to more complex cells, and so on. One model proposes that, at the highest levels of the hierarchy there should be **feature detectors** that respond to their ideal complex stimulus, but no other stimuli. This hypothetical model would require large numbers of neurons in contrast with normally efficient organization of the brain. Furthermore, the ability to respond to changes in stimulus dimension or modifications of perception due to experience also conflict with the "feature detector" model. (pp. 176-177)

A. An alternate approach is to view visual processing as a **spatial frequency analysis** or basic mathematical analysis of the contrasts and frequencies found in the visual field. (pp. 177-178)

B. The Perception of Depth (pp. 178-179)
   1. Depth perception results from monocular and binocular cues.
   2. Perspective, texture, shading, and relative size are monocular cues for depth that require only one eye.
   3. Cortical neurons known as **disparity-selective cells** respond to the slight differences in the images seen by the two eyes.
   4. The activity of these cells produces a sense of **retinal disparity**, which is a powerful binocular cue for depth.

C. Coding Color (pp. 179-182)
   1. Trichromacy and Opponent Processes
      a) The **trichromacy theory** of color perception is based on the theory that the brain can differentially examine the output of the three types of cones (blue, green, and red) that respond differentially to lights of different wavelengths.
      b) The visual system also shows a pattern of red-green and blue-yellow opponency. The opponent process theory is supported by the fact that many P cells show red-green center surround organization in their receptive fields, and non-P-or-M cells show blue-yellow center surround organizations.
      c) It appears that both trichromacy and the opponent process theories are used in the neural processing of color vision.
   2. Colorblindness
      a) Colorblindness results from missing or abnormal genes for the cone photopigments.
      b) **Dichromacy**, having only two color photopigments, is the most common form of color blindness. **Monochromacy**, having only one or no color photopigments is a much rarer condition.
      c) Red-green colorblindness is a sex-linked trait, with men ten times more likely that women to be dichromats since the genes for the red and green cone pigments appear on the X chromosome.

d) The very rare blue-yellow colorblindness is not sex-linked.
e) **Anomalous trichromacy** is characterized by the three cone photopigments having peak responses at abnormal wavelengths. There is evidence that some women may be **techromats**, having four different color pigments. Both individuals with anomalous trichromacy and techromats would likely never know that their sensations differ from the general population.

3. Color Contrast and Color Constancy
   a) The same color may appear to be very different depending on the context of surrounding colors due to a perception called **color contrast**. Color contrast is primarily an effect of the opponency of color processing by the visual system.
   b) **Color constancy** allows colors to look about the same regardless of the type of light illuminating the object. Neurons in Area V4 of the cortex may be responsible for color constancy.

XIII. The Development of the Visual System (pp. 182-183)

A. Babies under a year of age see less fine detail at a distance than adults due to their immature nervous systems. Infant toys tend to reflect the ability to perceive high-contrast, colorful objects. (p. 182)

B. Older adults experience loss of visual quality due to increased farsightedness, as called **presbyopia**, slow adaptation to changes in light, yellowing of the crystalline lens, smaller pupils, and less selectivity in cortical responses to visual input. (pp. 182-183)

XIV. Disorders of the Visual System (pp. 183-184)

A. **Cataracts** cloud the crystalline lens. (p. 183)

B. Eyeball length irregularities can lead to **myopia**, in the case of an elongated eyeball, or **hyperopia**, in the instance of a shortened eyeball, resulting in the lens failing to accurately project the focused image on the retinal surface as shown in Figure 6.33. Irregularities of the cornea can distortion of the image projected on the retina causing, **astigmatism**. (p. 184)

C. Stroke and physical damage may lead to disorders related to central nervous system damage of the visual cortex such as **scotomas**, a region of visual field blindness, or **blindsight,** the ability to perceive that a stimulus occurred in a region of the visual field without the conscious perception of the region. (pp. 184-185)

D. **Visual agnosias** occur when the brain cannot interpret what it sees, in other words a stimulus is seen but can not be correctly identified. A type of agnosia, **prosopagnosia**, occurs when a person cannot recognize faces or elements belonging to a learned category, such as models of cars. (p. 185)

XV. New Directions: The Effect of Culture on Perception (p. 186)

A. Eye movements of American and Chinese participants were recorded while they viewed visual scenes.

B. The American participants focused faster and longer on the main object of the photographs, while the Chinese participants spent equal time looking at the object and its context.

CHAPTER 10
Sexual Behavior
lecture outline

XVI. Sexual Development (pp. 285-293)

A. Sex Chromosome Abnormalities (pp. 286-288)
   1. **Turner syndrome** occurs in 1 out of 2,500 births, when a child receives only a single X chromosome (XO) instead of the usual pair (XX or XY). This condition is a random event and produces a female external appearance with abnormal ovaries and infertility.
   2. **Klinefelter syndrome** occurs in 1 out of every 500 to 1,000 male births, when a child has a XXY genotype. This condition produces male external appearance until puberty when hormone treatment becomes necessary to promote secondary male characteristics and inhibit female characteristics such as breast development.
   3. The effects of an XYY genotype are subtle and the relationship between XYY genotypes and increased aggression or antisocial behavior has been controversial.
   4. In general, sex chromosome abnormalities have mild effects with little or no influence on intelligence, mental or physical disorders, or criminal behavior.

B. Three Stages of Prenatal Development (pp. 288-290)
   1. The Development of the Gonads
      a) **Testis-determining factor**, produced by a gene on the Y chromosome called the **sex-determining region of the Y chromosome (SRY)**, turns the primordial **gonads** into **testes**.
      b) In the absence of testis-determining factor, **ovaries** will develop.
   2. Differentiation of the Internal Organs
      a) The male's testes will secrete **testosterone** and **anti-Müllerian hormone**, which promotes the development of male internal organs.
      b) In the absence of **androgens**, the female internal organs will develop.
      c) **Androgen Insensitivity Syndrome (AIS)**, a condition in a XY genotype fetus lacks androgen receptors, interferes with the development of male internal organs leading to the partial development of female genitalia (shallow vagina with no uterus or ovaries) and an association with the female **gender identity** and sexual behavior.
   3. Development of the External Genitalia
      a) Androgens released by the testes masculinize the **external genitalia**.
      b) Female external genitalia develop without any activity by the ovaries.
      c) **Congenital adrenal hyperplasia (CAH)** occurs when excess androgens masculinize the external genitalia of females.

C. Development at Puberty (pp. 291-292)
   1. The release of **gonadotropin-releasing hormones (GnRH)** by the hypothalamus signals the onset of puberty.
   2. Triggered by GnRH, the anterior pituitary gland releases **follicle-stimulating hormone (FSH)** and **luteinizing hormone (LH)** to promote the release of testosterone by the testes or estradiol by the ovaries, leading to the development of **secondary sex characteristics** in males and females respectively.
   3. **5-alpha-reductase deficiency** is characterized by ambiguous genitalia among males at birth followed by further masculinization at puberty.

XVII. Hormones and Sexual Behavior (pp. 294-300)

A. Regulation of Sex Hormones by the Hypothalamus and Pituitary Gland (p. 294)

1. **Gonadotropic-releasing hormone (GnRH)** is released by the hypothalamus.
2. In response to GnRH, the anterior pituitary releases luteinizing hormone (LH) and follicle-stimulating hormone (FSH).
3. In males, LH signals the testes to produce testosterone. Both testosterone and FSH are required for the maturation of sperm.
4. In females, LH and FSH control the menstrual cycle.

B. The Menstrual Cycle and Fertility (pp. 294-295)
The hormonal regulation of the menstrual cycle is summarized in Figure 10.11.

1. FSH from the anterior pituitary signals the development of **follicles**, small groups of cells that contain an **ovum**, by the ovary.
2. One follicle develops faster than the others, and releases estrogens to inhibit the development of other follicles.
3. About two weeks after the first day of the last menstruation, estrogens from the developing follicle signal the anterior pituitary to increase the release of LH providing the signal for release of the ovum or **ovulation**.
4. After ovulation, the ruptured follicle, now called the **corpus luteum**, releases estradiol and **progesterone**, which promotes pregnancy by inhibiting additional follicles and developing the lining of the uterus.
5. If fertilization does not occur, the corpus luteum stops releasing progesterone. Without progesterone the uterus sheds its lining in the process of menstruation.
6. Exclusive lactation, oral contraceptives, and long-term methods such as Norplant and Depo-Provera interfere with the cycle of female hormones, thus impairing fertility.
7. Following pregnancy, there is a readjustment period as hormones shift from the prolonged levels of circulating estradiol and progesterone back to normal levels. This shift in hormones may be related to **postpartum depression**, a mild depression phase experienced by many new mothers.

C. Sex Hormones and Female Behavior (pp. 297-299)
Why Does This Matter: The Controversy Over Gender Differences in Cognitive Abilities (p. 298)

1. Some species of animals undergo **estrus**, a period of female receptiveness to sexual behavior that coincides with ovulation. In nonestrus periods, females typically reject the male advances with aggression. Humans do not undergo estrus.
2. Sexual Interest in Human Females
   a) The sexual activity of human females is under little if any control of the hormones involved with ovulation.
   b) A woman's testosterone levels probably have the greatest impact on her sexual activity.
3. Estrogens and Cognition
   a) On tests of mental figure rotations, women received their best scores when testosterone levels were high and their worst scores when estrogen levels were high.

b) Verbal fluency and manual dexterity in women appear to be correlated with higher levels of estrogens.

D. Sex Hormones and Male Behavior (pp. 299-300)

1. Androgens and Competition: Male androgen levels appear to rise in response to being in competitive situations or even just viewing a competitive event.
2. Androgens and Sexual Frequency: As long a human male's testosterone level falls within normal limits, it does not provide a strong predictor of his sexual frequency.
3. Androgens and Cognitive Behavior
   a) The slight male advantage in spatial tasks may be related to androgen levels.
   b) Androgen may be related to male verbal abilities as men receiving testosterone supplements showed a 20 percent improvement in verbal fluency tasks.
4. Although still in experimental stages, synthetic testosterones or other chemicals that reduce LH and FSH may provide effective male contraception by interfering with sperm maturation.
5. **Anabolic steroids**, synthetic versions of testosterone, have legitimate medical uses in cases of malnutrition or muscle loss but are often abused by athletes seeking greater muscle mass.

XVIII. Sex Differences in the Nervous System (pp. 300-303)

A. Sexually Dimorphic Structures (pp. 300-302)

1. The **sexually dimorphic nucleus of the preoptic area (SDN-POA)** of the hypothalamus is larger in male rats than in female rats and appears to develop in response to androgens during a critical window.
2. Two of the four **interstitial nuclei of the anterior hypothalamus,** INAH-2 and INAH-3, are larger in male humans and in female humans and may be related to complex sexual behaviors.
3. The hypothalamus, anterior commissure, corpus callosum, and thalamus are also sexually dimorphic.
4. In the rat spinal cord, the **spinal nucleus of the bulbocavernosus (SNB)** also appears to be sexually dimorphic containing more motor neurons in the males in order to innervate the penis.

B. Masculinization of the Brain (pp. 302-303)

5. Estradiol resulting from **aromatization** of testosterone appears to be responsible for masculinizing the developing brain in many animals, including the hyena, but probably plays a lesser role in the masculinization of the human brain.
6. In humans and many other mammals, the placenta normally blocks the mother's estrogens.
7. Prenatal exposure to synthetic estrogens may masculinize the female brain.

XIX. Sexual orientation is a stable pattern of attraction to members of a particular sex. (pp. 303-305)

A. Correlative research suggests that prenatal exposure to hormones may influence sexual orientation. (pp. 303-304)

B. INAH-3 may be structurally different in homosexual and heterosexual males with the size of INAH-3 in homosexual males more similar to heterosexual females than heterosexual males. In addition, a difference in the size of the anterior commissure has been reported between homosexual and heterosexual males but a functional significance is unlikely. (pp. 304-305)

C. Twin studies suggest a genetic contribution to sexual orientation in males but it is important to note that the research also suggests significant influence by non-genetic factors as well. (p. 305)

XX. Attraction (pp. 306-308)

A. Symmetrical features may indicate genetic health, and therefore appear more attractive. (p. 306)

B. The Beauty of Fertility and a Good Immune System (pp. 306-308)
Why Does This Matter: Assortative Mating (p. 307)

1. Males prefer youthful female facial features and a 0.7 waist-to-hip ratio, which may indicate fertility.
2. Females prefer male facial features that indicate higher levels of androgens and male odors that signify a compatible immune system.

XXI. Romantic Love, Sexual Desire, and Parenting (pp. 308-310)
New Directions: Men, Women, and the Hook-Up (p. 310)

A. When participants viewed lovers as opposed to acquaintances, increased activity was observed by fMRI in areas associated with reward, and decreased activity was observed in areas associated with social judgment. (p. 308)

B. **Oxytocin** and **vasopressin** may influence patterns of mating and parenting. (pp. 308-309)

C. Only three percent of mammals practice **monogamy**, or exclusive mating with only one partner, with approximately twelve percent of primate species typically practicing monogamy or serial monogamy. (p. 308)

XXII. Sexual Dysfunction and Treatment: Although many cases of **erectile dysfunction** occur due to cognitive factors, recent developments in medications such as Viagra that increase nitric oxide (NO) activity have shown that physiology may also be involved sexual dysfunction. (p. 310)

Assessment 3 (Due Session 4, 6:00 p.m.)

1. a. (10 points) Describe the basic structure of the eye.
   b. (10 points) Neatly sketch the eye (freehand, not traced), using different colors for different major structures. Clearly label the following: retina, optic nerve, cornea, iris, lens, anterior chamber, vitreous chamber, pupil, fovea. (Drawings will not be evaluated based on artistic ability, but attention to detail is expected.)
2. (20 points) What cells and connections make up the layers of the retina?
3. (20 points) Describe in detail the pathways and structures along which and through which visual information travels from the point at which the optic nerve leaves the eye until the visual information reaches the striate cortex.
4. (20 points) Describe the three stages of structural development of the sexes prenatally.
5. (20 points) Describe the role hormones play in female sexual behavior and male sexual behavior.

Session FOUR
Chapters 11, 13

Chapter 11: Sleep and Waking
Chapter 13: Lateralization, Language, and Intelligence

ACTIVITIES
Quiz 3: Chapters 11 and 13
Lecture: Chapters 11 and 13
Break
Lecture: Chapters 11 and 13
Video

Secret Life of the Brain: The Aging Brain: Through Many Lives
For years, science has suggested that we lose vast numbers of brain cells as we grow older; now it turns out that this is not true -- in fact, healthy brains continue to produce new neurons well into the 70s. Drawing on the most recent neuroscience discoveries, this episode presents a new view of how the brain ages, focusing in part on the remarkable strides being made in understanding stroke, Alzheimer's Disease and Parkinson's Disease.

Video Reaction (in-class)

CHAPTER 11
Sleep and Waking
Lecture outline

XXIII. Circadian Rhythms: Daily or **circadian** rhythms respond to both internal signals and external "**zeitgebers**." Light is one of the most important zeitgebers for humans helping reset the 25 hour **free-running circadian rhythm** of humans that occurs in the absence of external cues. (pp. 313-320)

Why Does This Matter: Daytime Sleepiness (p. 316)

New Directions: Light Pollution (p. 319)

A. Variations in Sleep Patterns: Individual patterns of sleep vary, with some people preferring the morning like "larks," while others prefer the evening like "owls." (p. 314)

B. Shift Work, Jet Lag, and Daylight Saving Time (pp. 314-315)
   1. Discrepancies between circadian rhythms and travel or work schedules may result in negative symptoms.
   2. **Jet lag** is the condition of fatigue, irritability, and sleepiness that occurs when our circadian rhythm becomes out of sync with the external time due to crossing several time zones in a rapid manner such as flying. Since the free-running human cycle is longer than 24 hours, it is easier to adapt when traveling to later time zone (West) as opposed to an earlier time zone (East).
   3. Chronic jet lag may impair behavior and performance with one study demonstrating that airline flight attendants crossing time zones had reduced reaction times and increased numbers of mistakes compared to control participants that remained within a time zone.
   4. Daylight saving time in spring requires a phase advance, and can produce symptoms similar to jet lag. The time change in fall is equivalent to a phase delay, and causes little disruption for most people.

C. Internal Clocks (pp. 315-317)
   1. The body's internal clock has been localized to the **suprachiasmatic nucleus (SCN)** of the hypothalamus.
   2. The SCN receives external cues such as light via the **retinohypothalamic pathway**.
   3. The SCN appears to coordinate the activity of other internal clocks in different body tissue such as the liver, lung, and muscles.

D. The Cellular Basis of Circadian Rhythms (pp. 317-318)
   1. Oscillations of the genetic expression of proteins within single cells of the SCN may form the basis of the internal clock.
   2. Three proteins in particular have been identified as important in the regulation of SCN cell activity. The proteins, per and tim act to inhibit the Clock protein, which in turn can promote the production of per and tim. Figure 11.6 summarizes the cyclic nature of per, tim, and Clock protein expression.

E. Biochemistry and Circadian Rhythms (pp. 318-319)
   1. The SCN both regulates and responds to the hormone **melatonin**, secreted by the pineal gland during the dark phase.
   2. **Cortisol** levels are normally high first thing in the morning and low at night.

Figure 11.7 summarizes the circadian rhythms of awareness, body temperature, growth hormone, and cortisol.

F. Seasonal Affective Disorder (pp. 319-320)
1. **Seasonal Affective Disorder (SAD)** is a type of depression associated with a reduction in daylight hours during the winter months.
2. The exact cause of SAD is unknown but serotonin levels decrease during the winter and affected individuals may be more susceptible to this reduction.
3. SAD is normally treated by exposure to bright light.

XXIV. Stages of Waking and Sleep (pp. 320-323)

A. Wakefulness (p. 321)
1. Waking consists of alternations between **beta wave** and **alpha wave** patterns of brain activity.
2. Beta wave brain activity is characterized by **desynchronous**, rapid (15 to 20 hertz), irregular, and low-amplitude waves measured by an encephalogram (EEG) recording and represents alert and active thinking.
3. Alpha wave brain activity represents awake but relaxed cognition and is characterized by slightly more **synchronous**, slower (9 to 12 hertz), regular, and larger amplitude waves.
4. Patterns of alpha and beta wave activity follow **ultradian** cycles of 90 to 120 minutes.

B. Brain Activity During Sleep (pp. 321-323)
1. **Non-REM (NREM) sleep** consists of four progressively deeper stages of sleep, characterized by **theta wave** and **delta wave** brain activity.
2. **Rapid Eye Movement (REM)** sleep involves high levels of brain activity that resemble the waking stages.
   a) REM sleep is accompanied by muscle paralysis but movement of the eyes.
   b) The first REM period usually occurs after approximately 90 minutes of SWS.
   c) During the first half of the night there is a larger proportion of SWS, while the second half of the night contains a larger proportion of REM sleep.

XXV. Sleep Throughout the Lifespan (pp. 323-324)

The average daily proportions of awake, REM, and NREM periods across the lifespan are depicted in Figure 11.12.

A. Childhood (p. 323)
1. Newborn infants spend as much as 14 to 16 hours per day in sleep.
2. About half of the newborn's sleeping time is spent in REM sleep.
3. At puberty, there is a further slight decrease in REM and a substantial decrease in Stages 3 and 4 NREM sleep.

B. Adulthood (pp. 323-324)
1. NREM sleep declines further as people approach midlife.
2. Around the age of 50, total sleep time begins to decrease by about 27 minutes per decade into a person's 80s.

XXVI. Dreaming during REM and NREM: Dreaming behavior occurs during both REM sleep and NREM. (pp. 324-326)

A. REM dreams are lengthy, complicated, vivid, and storylike, providing us with the sense of firsthand experience with the events taking place. (pp. 324-326)

1. Allen Hobson and Robert McCarley (1977) proposed an activation-synthesis theory of dreaming, in which they argue that the content of dreams simply reflects ongoing neural activity.
2. Francis Crick and Graeme Mitchison (1983) suggested that dreaming offers a way for the brain to forget irrelevant and unnecessary information.
3. Jonathan Winson (1985) suggested that animals may have evolved the ability to integrate sensory experience with stored memories during REM sleep rather than while awake.
4. When the content of a REM dream is especially upsetting, we refer to the experience as a **nightmare**. **Lucid dreaming**, an awareness that one is dreaming, can be used to help control reoccurring nightmares through direction or controlling of the dream.

B. NREM dreams are short episodes characterized by logical single images and a relative lack of emotion. **Night terrors** occur during SWS when a person becomes partially aroused, disoriented, and inconsolable following an episode of dreaming with particularly negative emotional content. (pp. 324-325)

XXVII. The Functions of Sleep (pp. 327-330)

The exact function of sleep remains unknown; however, lack of sleep can have negative consequences leading to death in severe cases such as fatal familial insomnia, a genetic disorder.

Why Does This Matter: Sleep and Obesity (p. 329)

A. Sleep Keeps Us Safe: An animal's chance of predation is correlated with its sleep patterns such that species that are frequent prey sleep much less than animals are not unlikely to be prey. Figure 11.13 shows the relationship between predation, protection from shelter, and daily total sleep. (p. 327)

B. Sleep Restores Our Bodies(pp. 327-328)

1. Sleep may provide a compromise between the amount of energy that can reasonably be collected and the amount of energy expended within a 24-hour period.
2. Increased physical demands during the day appear correlated with a need for increased amounts of sleep the following night.

C. Sleep Helps Us Remember (p. 328): Sleep deprivation is correlated with poor performance on a variety of memory tasks, leading to theories about a role for sleep in the consolidation of memory.

D. Benefits of NREM Sleep: Selective deprivation of NREM usually causes a person to complain of muscle and joint aches and pains. (pp. 328-329)

E. Benefits of REM Sleep (p. 329)

1. REM sleep may contribute to learning consolidation or brain development.
2. Selective deprivation of REM produces **REM rebound** and elevated mood.

XXVIII. Brain Mechanisms of Wakefulness and Sleep (pp. 330-332)

A. The Control of Wakefulness (p. 330)

1. Pathways originating in the basal forebrain and in the reticular formation in the medulla participate in wakefulness.
2. The locus coeruleus and raphe nuclei are active during wakefulness, quieter during NREM, and silent during REM.

B. The Initiation and Control of NREM Sleep (pp. 330-331)

1. The preoptic area of the hypothalamus manages sleep debt, or the homeostatic control of wakefulness.
2. Without input from wakefulness circuits, the thalamus begins to synchronize activity of cortical neurons, leading to the slower waves seen in the NREM EEG.
3. Activity in the locus coeruleus and raphe nuclei begins to decline, preparing the brain for its next REM cycle.

A. The Initiation and Control of REM Sleep (pp. 331-332)
1. REM-on areas (active during REM but not wakefulness) are located in the pons, and in particular in the pontine reticular formation.
2. REM-off areas (the locus coeruleus and raphe nuclei) inhibit the pontine reticular formation, so when they are silent, REM occurs.
3. Characteristic brain activity during REM sleep called **PGO waves** is associated with the eye movements of REM sleep. The PGO waves begin the pons traveling to the lateral geniculate nucleus of the thalamus and then to the occipital cortex.
2. During REM, secondary visual cortex, the hippocampus, the amygdala, and the anterior cingulate cortex are very active, but most of the frontal lobe is very inactive, possibly leading to the bizarre, illogical, and socially inappropriate content of some dreams.

XXIX. The Biochemistry of Wakefulness and Sleep (pp. 332-333)

A. Acetylcholine released in the pons and basal forebrain is associated with wakefulness and REM. (p. 332)
B. Histamine activity is high during wakefulness but low during sleep, which is why traditional antihistamines used to treat allergies made people drowsy. (p. 333)
C. Serotonin and norepinephrine levels are highest during wakefulness, less active during NREM, and very low during REM, which explains by depressed patients with low serotonin activity often show too much REM sleep. (p. 333)
D. Adenosine has an inhibitory effect on many brain systems, and accumulates during wakefulness. During sleep, adenosine levels drop again, allowing wakefulness to occur in the morning. (p. 333)

XXX. Sleep Disorders (pp. 334-337)

A. **Dyssomnias**: Difficulty in the initiation, maintenance, timing and quality of sleep (pp. 334-336)
1. **Insomnia**: Difficulty initiating or maintaining enough sleep to feel rested.
   a) **Onset insomnia** is characterized by difficulty getting to sleep.
   b) **Maintenance insomnia** is characterized by not staying asleep during the night.
2. **Sleep apnea**: Interruptions in breathing during sleep likely related to an obstruction of the airway but in rarer circumstances may involve abnormalities of brainstem neurons.
3. **Narcolepsy**: The occurrence of "sleep attacks," in which episodes and features of REM sleep occur during wakefulness. **Cataplexy** is a feature of narcolepsy in which REM paralysis occurs during wakefulness.

B. **Parasomnias**: Disturbance of sleep by unusual behaviors. (pp. 336-337)
1. **Sudden Infant Death Syndrome (SIDS)**: The death of a basically healthy baby, usually between 2 and 4 months of age, while asleep. The precise cause of SIDS

is unknown; however, sleeping position may play a role in the susceptibility to SIDS.

2. Sleep talking appears to occur in both light stages of SWS and during REM sleep.
3. **Somnambulism** (sleepwalking) is much more common in children than adults and is probably a deep SWS phenomenon.
4. **REM behavior disorder** is diagnosed when normal REM paralysis does not occur allowing the affected individual to act out dream activities often resulting in injuries.
5. **Restless leg syndrome (RLS)** occurs when one of a person's limbs, usually a leg, moves at regular intervals during sleep.

CHAPTER 13
Lateralization, Language, and Intelligence

LECTURE OUTLINE

XXXI. Hemisphere Lateralization and Savant Behavior (pp. 374-375)

A. **Savant behaviors** are exceptional skills and talents found in people whose intellectual functioning otherwise falls within the range of mental retardation. (p. 374)

B. Although the origin of savant behaviors remains a mystery, current speculation centers on possible damage to the left hemisphere, leading to compensating development of the right hemisphere. (pp. 374-375)

II. Lateralization of Function (pp. 376-384)

Lateralization refers to the localization of a function in either the left or right hemisphere.

A. Hemispherectomy (p. 376)
Hemispherectomy, the removal of the entire cerebral cortex of one hemisphere, is a radical treatment for Rasmussen's disease, a seizure disorder affecting young children.

B. The Split Brain (pp. 377-379)

1. In **split-brain operations**, some or all of the pathways connecting the right and left cerebral hemispheres (**corpus callosum**, **anterior commissure**, **hippocampal commissure**, and **massa intermedia**) are severed.
2. Patients experienced no changes in personality, intelligence, or speech.
3. Language and logical thought was found to be localized in the left hemisphere, whereas intuitive, spatial processing was localized in the right hemisphere.

C. The Development of Lateralization (pp. 379-380)

1. Lateralization apparently occurred early in the course of evolution.
2. Lateralization may allow organisms to "multitask," or to split their attention between different aspects of the environment.
3. Prenatal testosterone levels may determine the extent of lateralization of function between the two hemispheres (Geschwind & Galaburda).
4. Other hypotheses for lateralization include different levels of gene expression in the two hemispheres and environmental factors.

D. Implications of Hemispheric Asymmetry for Behavior: beyond anecdotal reports, the experimental support of hemispheric dominance influencing behavior is weak at best. (pp. 380-384)
Why Does This Matter: Why Do We Have a Preferred Hand? (p. 381)

1. Handedness and Hemisphere Lateralization: Handedness is significantly correlated with the lateralization of language.
2. **Dichotic Listening** investigates the lateralization of auditory processing by supplying each ear simultaneously with a different sound.
3. The Localization of Prosody and Musical Abilities: **Prosody**, the emotional tone in speech, is typically processed by the right hemisphere. Different aspects of processing music are lateralized to the right and left hemispheres.
4. Gender Differences in Lateralization: Gender differences in handedness exist, but no significant gender differences in lateralization of language or structural symmetry have been found in a very large meta-analysis of existing data.

XXXII. Language (pp. 384-389)

A. The Origins of Language (pp. 384-386)
New Directions: Lateralization, Language, and Schizophrenia (p. 386)

1. There is no human culture on earth that exists without language.
2. Tool use and cooperation among early hominids are possible indicators for language ability.
3. An important genetic mutation in the *FOXP2* gene may have occurred only about 100,000 years ago.
4. Click languages may be the earliest form of human language.

B. Are Non-human Animals Capable of Real Language? (pp. 387-388)

1. There is an important distinction to be made between communication, no matter how complex, and language.
2. Although some animal communication systems demonstrate several core features of language, it is possible that only human language meets all ten criteria for language as listed in Table 13.1.
3. Great apes show asymmetry in Broca's area, similar to humans but lack the innate capability to acquire language skills spontaneously without laborious training.
4. Apes, dolphins, whales, parrots, and dogs may have some language capabilities.

A. Multilingualism (pp. 388-389)

1. More than half of the world's population is **bilingual**, or proficient in two languages.
2. Multiple languages use some of the same areas of the brain but that the degree of overlap is not 100 percent.
3. When early-acquisition bilinguals switch between use of each language, there is a slower reaction time and activation of the dorsolateral prefrontal cortex. Rather than representing additional language areas, one interpretation is that the language switch may simply be a function of general executive attentional systems of the brain, managed by the dorsolateral frontal cortex.

B. **American Sign Language (ASL)** is processed very much like verbal languages, in spite of its spatio-visual nature. (p. 389)

XXXIII. Communication Disorders and the Brain Mechanisms for Language (pp. 389-396)

A. Paul Broca and Patient "Tan" (pp. 389-390)

1. Parisian physician, Paul Broca (1824-1880) is credited with identifying the left inferior frontal lobe as the localization of language in the brain based on the autopsy examination of the brain of a patient "Tan" inflicted with severe **aphasia**.
2. The ventral cortex anterior to the primary motor cortex in the frontal lobe is now referred to as **Broca's area** and is known to be involved in speech production.

B. Aphasias (pp. 390-393)

1. Broca's Aphasia
    a) The primary symptom of **Broca's aphasia** is difficulty in language production.
    b) Broca's aphasia appears to the specific to language dysfunction as opposed to a motor dysfunction as it affects both oral and written language output but may not affect the ability to sing songs.

c) **Anomia** is a condition in which patients have difficulty finding the appropriate words to express their thoughts.
d) Damage in Broca's area and underlying white matter is responsible for this type of nonfluent aphasia.

2. Wernicke's Aphasia
   a) **Wernicke's area** is located on the superior surface of the left temporal lobe.
   b) **Wernicke's aphasia** produces speech that is rapid and fluent but virtually meaningless due to failures in language comprehension.
3. Conduction Aphasia
   a) Damage to the arcuate fasciculus, the neural pathway between Wernicke's area and Broca's area, and adjacent cortex is responsible for **conduction aphasia**.
   b) Repeating sentences and "confrontation tasks" in which patients must verbally name objects are very difficult for these patients.
4. Global Aphasia
   a) Both Broca's and Wernicke's areas, as well as much of the cortex and white matter between them, are affected in **global aphasia**.
   b) Abilities to speak, comprehend, read, and write are impaired.
5. **Transcortical Aphasias** affect cortical areas association with language production or comprehension.
   a) **Transcortical motor aphasia** is associated with damage to the **dorsolateral frontal cortex** or the **supplementary motor area**. Patients have difficulty producing speech, but no difficulties repeating sentences.
   b) Patients with **transcortical sensory aphasia** experience damage to connections between language centers and areas storing word meanings. Patients with retain fluent, grammatical speech, but their comprehension is impaired. Repetition is excellent.
6. Language Models
   a) According to the Wernicke-Geschwind model, Broca's area was responsible for speech production, whereas Wernicke's area was responsible for speech comprehension. The arcuate fasciculus was believed to be a one-way pathway connecting Wernicke's area to Broca's area.
   b) A more contemporary language model proposed three interacting language components: a language implementation system, a mediational system, and an conceptual system.

C. Disorders of Reading and Writing (pp. 393-395)

1. Alexia and Agraphia (p. 394)
   a) Patients with **alexia** are unable to read or to point to words and letters on command. Most cases result from damage to the left occipital cortex and the corpus callosum.
   b) **Agraphia**, or the inability to write, may arise from damage to the motor areas responsible for making skilled movements.
   c) In **phonological agraphia**, patients can't sound out words.
   d) In **orthographic agraphia**, patients can spell phonetically but can't form visual images of words to be spelled.
2. Dyslexia (pp. 394-395)

a) An anatomical basis for **dyslexia** may involve differences in hemispheric symmetry.
b) The most prevalent subtype of dyslexia occurs as a result of poor **phonological awareness**.
c) Overactivation of Broca's area, coupled with a lack of activation of Wernicke's area and the angular gyrus, appears to occur during reading by participants with dyslexia.

D. Stuttering (p. 396)
1. Approximately 1 percent of the population experiences persistent difficulty with **articulation**.
2. **Stuttering** is characterized by repetitions or prolonging of sounds and appears to be primarily a genetic condition affecting three times as many males than females.
3. Abnormal lateralization of language in the two hemispheres or abnormal activity in the basal ganglia and midbrain motor structures may contribute to stuttering.

XXXIV. Intelligence (pp. 397-399)

Why Does This Matter: Enhancing Intellect (p. 398)

A. Assessing Intelligence: Interest in assessing **intelligence** arose from compulsory education laws passed during the nineteenth and early twentieth centuries. (p. 397)

B. General or Specific Abilities: Psychologists are unable to agree on whether intelligence comprises a single underlying ability as suggested by Spearman's **general intelligence (g) factor** or some combination of separate abilities. (pp. 397-398)

C. Intelligence and Genetics (pp. 398-399)
1. Estimates of the heritability of intelligence vary widely, from between 60 to 80 percent.
2. A possible correlate of intelligence is the amount of gray matter, particularly in the frontal lobe and language areas.
3. It is likely that a large number of genes, each having a very small effect, contribute to IQ. Genetics and environment interact in complex ways to influence intelligence.

D. Biological Correlates of Intelligence: Trying to identify biological correlates of intelligence is a difficult prospect involving case study analysis such as examining differences between Albert Einstein's brain and control subjects. (p. 399)

Assessment 4 (Due Session 5, 6:00 p.m.)

1. (20 points) Describe the stages of sleep: Stage 1, Stage 2, Stage 3, Stage 4, REM.
2. (20 points) Describe the sequence of stages in a typical first cycle of a night's sleep and the changes in the pattern of the stages within cycles over the course of a night's sleep.
3. (20 points) What do studies of REM and SWS deprivation contribute to our understanding of the functions of these sleep components?
4. (20 points) Describe the localization of language in the brain.
5. (20 points) Describe the following communication disorders: Broca's aphasia, Wernicke's aphasia, dyslexia.

Session FIVE
Chapter 16

Chapter 16: Psychological Disorders

ACTIVITIES
Lecture: Chapter 16
Course Evaluation
Break
Lecture: Chapter 16
Comprehensive Exam

CHAPTER 16
Psychological Disorders
lecture outline

XXXV. **Schizophrenia** is a disorder characterized by the presence of **hallucinations**, **delusions**, disorganized thinking, and mood disturbances that affects about 1 percent of the world's population. (pp. 457-465)

A. The Genetics of Schizophrenia (pp. 458-459)
   1. The **concordance rate** is about 50 percent in identical twins and about 17 percent in fraternal twins.
   2. Environment appears to play a minimal role in the development of schizophrenia as adopted children were much more likely to become schizophrenic if their biological parents had the disorder than if their adopted parents had the disorder.
   3. Patients with schizophrenia and many of their family members show abnormal **saccades**, rapid eye movements when reading or visually tracking an object.
   4. Many genes may be abnormal in patients with schizophrenia, some of which overlap with susceptibility genes for bipolar disorder.

B. Environmental Contributions to Schizophrenia: Stress, prenatal exposure to viral infection, and complications of pregnancy may be correlated with schizophrenia. (p. 460)

C. Brain Structure and Function in Schizophrenia (pp. 460-462)
   1. Many patients with schizophrenia show enlarged ventricles (shown in Figure 16.5) and cellular disorganization in the hippocampus (shown in Figure 16.6).
   2. Hypofrontality, a lower level of frontal lobe activity, is correlated with schizophrenia and may be related to the **negative symptoms**, such as mood disturbances and social withdrawal.
   3. Schizophrenia is correlated with a higher degree of brain symmetry, ambiguous handedness, and larger than normal loss of gray matter during adolescence.

D. The Biochemistry of Schizophrenia (pp. 462-463)
   1. An increased sensitivity to dopamine may be responsible for some of the symptoms of schizophrenia but not all schizophrenia patients respond to neuroleptic drugs, such as **phenothiazines**, that block dopamine receptor sites.
   2. Based on pharmacological evidence, a decrease in glutamate sensitivity may also be associated with schizophrenia.

E. The Treatment of Schizophrenia (pp. 464-465)
   1. The discovery of typical antipsychotics, which are dopamine antagonists, revolutionized the treatment of schizophrenia.
   2. Typical antipsychotics are not specific in blocking dopamine receptors in one particular area of the brain and thus may produce serious side effects, including **tardive dyskinesia**, a chronic disorder characterized by involuntary, jerky movements.
   3. Atypical antipsychotic medications are more successful than traditional antipsychotics in treating negative symptoms, but are not necessarily safer than the older types of drugs.
   4. Persistent auditory hallucinations have been treated successfully using repeated transcranial magnetic stimulation (rTMS).

5. Psychosocial rehabilitation is also helpful in treating schizophrenia but unfortunately only approximately 10 percent of schizophrenic patients receive any additional treatment other than medications.

XXXVI. Mood Disorders (pp. 466-472)

A. **Major Depressive Disorder** is characterized by a fairly constant state, at a minimum 2 weeks, of depressed mood and loss of pleasure in normally enjoyable activities. (pp. 466-471)

1. The Genetics of Depression
   a) Based on the study of twins, heritability for depression is about 33 percent.
   b) Having a short copy of the serotonin transporter gene is not sufficient to produce depression, but among people who experience stressful life events, those with two copies of the short version are more likely than those with one or zero copies of the short version to become depressed.
2. Environmental Factors and Depression: Stress and prenatal factors may contribute to the development of major depressive disorder.
3. Brain Structure and Function in Depression
   a) People with major depressive disorder show structural and activity differences in the hippocampus, orbitofrontal cortex, and anterior cingulate cortex when compared to healthy controls. However, whether these are the cause or effect of depression or both is unknown.
   b) Depression is correlated with reduced activity in the left frontal lobe and increased activity in the right frontal lobe.
   c) Correlations between sleep patterns and depressed mood may reflect a larger disturbance in circadian rhythms. Both hypersomnia and hyposomnia are symptoms of depression and depressed patients often exhibit abnormal REM and NREM sleep patterns.
4. The Biochemistry of Depression
   a) Reductions in the availability of monoamines, particularly serotonin, have been implicated in depression.
   a) Figure 16.15 depicts a theoretical break in the feedback loop of the hypothalamic-pituitary-adrenal axis in patients suffering from mood disorders.
5. The Treatment of Depression
   a) Antidepressant medications typically are serotonin agonists, and in particular, selective serotonin reuptake inhibitors (SSRIs).
   b) Alternatives to medication include cognitive-behavioral therapy and increased aerobic exercise.
   c) Combining antidepressant and cognitive-behavioral therapy typically produces the best long-term outcome for treating major depressive disorder.
   d) Patients with major depressive disorder do not respond to antidepressants may respond to electroconvulsive shock therapy (ECT). There have been many modern advances in ECT including anesthetizing the patient and delivering lower more selective electrical stimulation since the graphic depict of ECT in *One Flew Over the Cuckoo's Nest*.

B. **Bipolar Disorder** is characterized by alternating cycles of depression and **mania**. (pp. 471-472)

1. Causes of Bipolar Disorder
   a) Genes appear to play a more significant role in bipolar disorder than in major depressive disorder.
   b) Concordance rates of bipolar disorder among identical twins are often reported to be as high as 85 percent.
   c) Genes involved with bipolar disorder might overlap with those involved with schizophrenia and major depressive disorder.
   d) Correlations suggest that bipolar disorder is more common in nations that consume less seafood, but efforts to treat bipolar disorder by adding omega-3 fatty acids to patients' diets have produced mixed results.
2. Brain Structure and Function in Bipolar Disorder: Reduced hippocampal volume, elevated basal ganglia activity, enlargements of the amygdala are correlated with bipolar disorder.
3. The Biochemistry and Treatment of Bipolar Disorder
   a) Monoamine abnormalities may account for bipolar disorder.
   b) Lithium, believed to influence the activity of second messengers and enzymes and to enhance norepinephrine reuptake, is typically used to treat bipolar disorder.

XXXVII. Anxiety disorders share the core element of unrealistic and counterproductive anxiety. (pp. 473-476)
Why Does This Matter: Understanding Self-Injury (p. 476)

A. Common Features of Anxiety Disorder
   1. A vulnerability to anxiety disorder may be inherited, but related people will not necessarily experience the same type of anxiety disorder.
   2. Neural systems connecting the brainstem and the limbic system and the frontal lobes and the limbic system seem to be involved in the various anxiety disorders.
   3. Abnormalities in the HPA axis may produce anxiety disorders and represent a link between major depressive disorder and anxiety disorder.
   4. Anxiety may result from abnormalities in multiple neurotransmitter systems such as norepinephrine, serotonin, and GABA.
   5. Treatment for anxiety disorders typically combines medication, usually GABA-agonist benzodiazepines, with some type of cognitive-behavioral therapy.

B. **Obsessive-Compulsive Disorder (OCD)** is characterized by repetitive, intrusive thoughts (**obsessions**) and/or the need to carry out repetitive behaviors (**compulsions**) in order to alleviate the obsessive thoughts.
   1. Concordance rates have been reported to be 63-87 percent for identical twins.
   2. Brain structures implicated in OCD include the **orbitofrontal cortex**, the basal ganglia, and the thalamus.
   3. OCD appears to involve abnormalities in serotonin function, and is frequently treated with antidepressants.
   4. As shown in Figure 16.19, cognitive-behavioral therapy may produce the same types of changes in brain activity as OCD medications.
   5. Some cases of OCD are treated using deep brain stimulation.

B. **Panic disorder** is diagnosed when repeated **panic attacks** are followed by at least 1 month of worrying about having another attack.

1. The locus coeruleus, the hippocampus, orbitofrontal cortex, and the cingulate cortex may mediate the panic response.
2. Research has shown that panic attacks may be triggered by administering sodium lactate.
3. Treatment for panic disorder generally consists of either antidepressant medication and/or cognitive-behavioral therapy.

C. **Post-traumatic stress disorder (PTSD)** results when exposure to trauma produces recurrent dreams, flashbacks of the incident, avoidance of stimuli associated with the trauma, hyperarousal, and impairment in daily functioning.
*See the Lecture Enrichment section for additional information of common sources of PTSD.

1. Small hippocampus size is correlated with PTSD.
   a) A preexisting smaller hippocampus may contribute to the vulnerability of developing PTSD.
   b) Alternatively, high levels of cortisol released as a stress response to the traumatic event may act to reduce the size of the hippocampus.
2. Animal models suggest that stress acts to decrease in benzodiazepine receptors in the frontal cortex thus increasing brain activity that may be involved with PTSD.
3. Typical treatment of PTSD includes antianxiety or antidepressant medication combined with cognitive-behavioral therapy.

XXXVIII. **Autism** is characterized by delays and impairments in social behavior, communication, and interests. (pp. 476-478)

A. Causes of Autism (p. 477)
1. Rates of autism incidence have risen dramatically over the past 2 decades but it is unclear if these increases reflect an actual increase in the prevalence of autism or just an increased awareness of autism.
2. The concordance rate for autism in identical twins is at least 90 percent.
3. Autism probably involves large pools of genes involved with brain development.
4. Environmental factors that might trigger autism in a genetically vulnerable individual include exposure to mercury.

B. Brain Structure and Function in Autism (pp. 477-478)
1. Brain development in autism first accelerates abnormally and then decelerates abnormally in childhood.
2. The increase in brain mass may be related to elevated levels of neurotrophins or disruptions in the pruning and apoptosis stages of postnatal neural development.
3. Minicolumns, the smallest processing units in the brain, appear smaller in individuals with autism and in a small sample of eminent scientists who did not have autism.
4. Abnormalities in the amygdala and the cerebellum are associated with autism.
5. Dysfunctions in mirror neurons might account for some of the deficits in social function seen in autism.

C. Biochemistry and Treatment of Autism (p. 478)
1. Serotonin, GABA, and glutamate activity appear to be abnormal in autism, but medications have not been effective in addressing these differences.
2. Controlled studies do not provide support for the deletion of gluten and casein from the diets of individuals with autism.

3. Autism is normally treated with intensive, early childhood learning experiences provided during most of the child's waking hours.

XXXIX. Attention Deficit/Hyperactivity Disorder is diagnosed when children demonstrate inappropriate levels of attention, impulsivity, and/or motor activity. (pp. 478-479)
Why Does This Matter: Are There Steroids for the Mind? (p. 480)

A. Genetics of ADHD: Estimates of the heritability of ADHD may be 80% or even higher.
B. Brain Structure and Function in ADHD (p. 479)
   1. The frontal lobes and caudate nucleus may show abnormally low activity levels in ADHD producing deficits in planning actions and inhibiting inappropriate behaviors.
   2. The effectiveness of the dopamine agonist drugs, **methylphenidate (Ritalin)**, **dextroamphetamine (Dexedrine or Dextrostat)**, or amphetamine salts (Adderall) in treating ADHD has focused research on dopaminergic systems.
   3. Lead contamination, low birth weight, and prenatal exposure to tobacco, alcohol, and other drugs can lead to symptoms of ADHD, either alone or in conjunction with susceptibility genes.
C. Biochemistry and Treatment of ADHD (p. 479)
   1. ADHD is treated primarily with medication, either alone or in combination with behavioral therapy.
   2. Improvements in behavioral symptoms do not persist when medication is discontinued and there is no evidence of long-term improvements in academic performance due to medication.
   3. The frequent use of medication for ADHD in the United States, five times greater than any other nation, has been the subject of considerable criticism.

XL. **Antisocial personality disorder (APD)** is characterized by a lack of guilt and empathy and a failure to conform to social and legal codes of behavior. APD is often confused with psychopathy, which does not rely so heavily on the presence of criminal behavior. (pp. 480-483)

A. Genetics of Antisocial Personality Disorder (pp. 480-481)
   1. Antisocial personality disorder shows complex interactions between genes and environment. Child maltreatment, in particular, interacts with a person’s MAOA genes.
   2. Twin studies have demonstrated strong heritability for the emotional aspects of psychopathy, such as fearless dominance, impulsive antisociality, and callousness (.46 to .67).
B. Brain Structure and Function in Antisocial Personality Disorder
   1. Antisocial behavior is correlated with reduced emotional responsiveness.
   2. Antisocial behavior may be produced by damage to the orbitofrontal cortex or reduced activity levels in the frontal lobes.
C. Treatment of Antisocial Personality Disorder: Treatment for antisocial personality disorder has been modestly effective in children and youth but somewhat discouraging among adults.
   New Directions: Epigenetics and Psychopathology (p. 482)